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MEASURING STABILITY OF 3D CHROMATIN CONFORMATIONS AND IDENTIFYING NEURON SPECIFIC CHROMATIN LOOPS ASSOCIATED WITH

SCHIZOPHRENIA RISK

A Dissertation Presented

By

TYLER BORRMAN

Submitted to the Faculty of the

University of Massachusetts Graduate School of Biomedical Sciences, Worcester

in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

NOVEMBER 12th, 2020

BIOINFORMATICS & COMPUTATIONAL BIOLOGY

MEASURING STABILITY OF 3D CHROMATIN CONFORMATIONS AND IDENTIFYING NEURON SPECIFIC CHROMATIN LOOPS ASSOCIATED WITH SCHIZOPHRENIA RISK

ii

A Dissertation Presented

By

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This thesis is dedicated to my Mom and Dad for always encouraging me to pursue my dreams and providing me with the tools required to achieve them.

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v

ABSTRACT

The 23 pairs of chromosomes comprising the human genome are intricately folded within the nucleus of each cell in a manner that promotes efficient gene regulation and cell function. Consequently, active gene rich regions are compartmentally segregated from inactive gene poor regions of the genome. To better understand the mechanisms driving compartmentalization we investigated what would occur if this system was disrupted. By digesting the genome to varying sizes and analyzing the fragmented 3D structure over time, our work revealed essential laws governing nuclear compartmentalization.

At a finer resolution within compartments, chromatin forms loop structures capable of regulating gene expression. Genome wide association studies have identified numerous single nucleotide polymorphisms (SNPs) associated with the neuropsychiatric disease schizophrenia. When these SNPs are not located within a gene it is difficult to gain insight into disease pathology; however, in some cases chromatin loops may link these noncoding schizophrenia risk variants to their pathological gene targets. By generating 3D genome maps, we identified and analyzed loops of glial cells, neural progenitor cells, and neurons thereby expanding the set of genes conferring schizophrenia risk.

The binding of T-cell receptors (TCRs) to foreign peptides on the surface of diseased cells triggers an immune response against the foreign invader. Utilizing available structural information of the TCR antigen interface, we developed computational methods for successful prediction of TCR-antigen binding. As this binding is a prerequisite for immune response, such improvements in binding prediction could lead to important advancements in the fields of autoimmunity and TCR design for cancer therapeutics.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS	iv
ABSTRACT	vi
LIST OF TABLES	xiii
LIST OF FIGURES	xiv
LIST OF COPYRIGHTED MATERIALS PRODUCED BY THE AUTHOR.	xvi
CHAPTER I: INTRODUCTION	1
Computational Biology	1
Genome structure	2
Compartments	7
Promoter-Enhancer Interactions	17
Schizophrenia, Neurons, and 3D organization	27
TCR-pMHC Interactions	33
CHAPTER II: COMPARTMENT-DEPENDENT CHROMATIN INTERACT	
DYNAMICS REVEALED BY LIQUID CHROMATIN HI-C	43
Preface	43
Abstract	44
Introduction	44
Results	47
Measuring stability of chromatin interactions and nuclear	
compartmentalization	
Chromosome conformation in isolated nuclei	50
Extensive chromatin fragmentation leads to the formation of liquid chr	
Compartmental segregation requires chromatin fragments larger than	6 kb 56
Quantification of chromosome conformation dissolution	61
After correcting for differential fragmentation LOS remains highly corre with compartment status	
Dissociation kinetics of chromatin interactions and compartments	70
Quantification of the half-life of chromosome conformation across the genome	78

Dissociation kinetics of chromatin interactions at different sub-nuclear structures
Chromatin loops dissociate upon chromatin fragmentation
Discussion
Tables
Materials and methods
Digestion, cross-linking and copolymer architecture and hetero/euchromatin phase separation
K562 nuclei purification
3C (Chromosome Conformation Capture)102
Chromosome Conformation Capture Carbon Copy (5C)
Pre-digestion of nuclei (liquify chromatin)110
Hi-C 2.0
DpnII-Seq121
DpnII Pre-digestion size assessment 127
Lamin A Immunofluorescence and DAPI133
Chromatin fractionation assay134
Micromanipulation force measurement and treatments of an isolated nuclei
3C-PCR
5C data processing
Hi-C data processing139
A/B compartments
LOS and half-life calculation141
DpnII-seq data analysis144
Fatl-seq data analysis145
DpnII pre-digestion average fragment size analysis145
Subcompartments147
Sub-nuclear structures148
Gene Expression
Compartmentalization saddle plots150

ix

Homotypic interaction saddle plots	151	Х
Scaling plot	151	
Mean z-score heatmap	152	
CHAPTER III: NEURON-SPECIFIC SIGNATURES IN THE CHROMOSO CONNECTOME ASSOCIATED WITH SCHIZOPHRENIA RISK		
Preface	153	
Abstract	154	
Introduction	155	
Results	156	
Neural progenitor differentiation is associated with dynamic 3DG remo	-	
Chromosomal contacts associated with schizophrenia risk sequences	172	
Cell type–specific schizophrenia-related chromosomal connectomes a associated with gene co-regulation and protein-protein association ne	tworks	
Discussion	206	
Tables	209	
Materials and methods	272	
In situ Hi-C from hiPSC-derived cells	272	
Hi-C loop calls using Juicer	274	
Hi-C interactions at risk loci	275	
Generation of stable selected dCas9-VP64/VPR and Cas9 NPCs	277	
In vitro transcription and transfection of gRNAs	278	
RNA transcriptomic correlation heatmaps	279	
Generation of hiPSC-derived cell types	280	
Immunofluorescent staining	283	
Hi-C A/B compartment calling	284	
Hi-C topologically associating domain calling	284	
Cumulative distribution of loop size	285	
Gene expression v. loop analyses	285	
Gene ontology	286	
ATAC-seq and accessibility processing	286	

Estimating significance of rate of loop/eQTL overlap	288	xi
Comparing interaction intensity within interactions with PCDH locus.	289	
Single SNP-level eQTL analysis	289	
Real-time qPCR	290	
Determining cell-type-specific interactions	290	
RNA-seq and transcriptome processing	290	
Protein-protein interaction network analysis	292	
Human postmortem prefrontal cortex tissue	293	
Biochemical fractionation		
Sample preparation and LC-SRM/MS		
Mass spectrometry data processing, informatics and statistics	295	
CHAPTER IV: ATLAS: A DATABASE LINKING BINDING AFFINITIES	WITH	
STRUCTURES FOR WILD-TYPE AND MUTANT TCR-PMHC COMPLE	XES 296	
Preface	296	
Abstract	296	
Introduction	297	
Results	302	
Using the data in ATLAS to develop energy functions	303	
The web-based user interface of ATLAS	310	
Discussion	314	
Tables	317	
Materials and methods	319	
Data collection	319	
Metadata fields	320	
Protein modeling	321	
Regression analysis	321	
Architecture	322	
CHAPTER V: HIGH-THROUGHPUT MODELING AND SCORING OF T PMHC COMPLEXES TO PREDICT CROSS-REACTIVE PEPTIDES	-	
Preface	323	
Abstract	323	

Introduction	xii
Results	
High-throughput modeling reproduces experimentally observed enrichment of binder peptides	
Correlation between top computationally selected peptides and top experimentally selected peptides	
Discussion	
Materials and methods346	
Sequence extraction	
Peptide structure modeling346	
Prediction of peptide–MHC/TCR binding free energy	
CHAPTER VI: DISCUSSION	
Defining compartment principles by liquid chromatin Hi-C	
Chromatin loops and liquid chromatin Hi-C	
3D genome architecture and schizophrenia	
Prediction of TCR-pMHC binding energies and cross-reactive peptides366	
Conclusion	
BIBLIOGRAPHY	

LIST OF TABLES

Table 2.1 Public datasets used to associate liquid chromatin Hi-C measured stability with various chromatin features	96
Table 3.1 Hi-C and ATAC-Seq sequencing summary and quality controls	
Table 3.2 Gene ontology (GO) of Brain-specific loops	
Table 3.3 Neuron-specific loops GO	
Table 3.4 NPC-specific loops GO	
Table 3.5 Glia-specific loops GO	
Table 3.6 GM12878 lymphoblastoid-specific loops GO	
Table 3.7 TADs expanded in neurons compared to glia	
Table 3.8 TADs expanded in neurons compared to NPC	228
Table 3.9 Loops anchored in schizophrenia (SZ) risk sequence.	
Table 3.10 Chromosomal contacts anchored in SZ GWAS co-localized eQTI	LS
Table 3.11 Gene-level single-SNP eQTLs for clustered PCDH	
Table 3.12 Locus-specific chromosomal contacts in fetal brain compared to	
hiPSC-derived cells	240
Table 3.13 Oligonucleotide sequences for gRNA in vitro transcription	243
Table 3.14 qPCR primer sequences for RNA quantification in CRISPR	
experiments	246
Table 3.15 Genes located in cell-type specific SZ risk associated chromoson	nal
contacts	
Table 3.16 GO for genes in SZ risk-associated chromosomal contacts	252
Table 3.17 List of genes shown in RNA correlation heatmaps	258
Table 3.18 Summary of results from RNA-seq sampling/permutation anslyse	s
	263
Table 3.19 Neuronal signaling and chromatin regulatory genes clustered in F	RNA
heatmap	264
Table 3.20 GOs of gene clusters with high correlation scores from Table 3.19	9
	266
Table 3.21 Protein networks (string-db) in SZ risk connections	
Table 3.22 Demographics of brain cohort used for proteomic analysis	271
Table 4.1 ATLAS entries with both wildtype and mutant crystal structures	
available	
Table 4.2 Rotamer analysis for designed mutations	318

LIST OF FIGURES

Figure 1.1 Illustration of compartmentalization
Figure 1.3 Crystal structure of TCR-pMHC complex
Figure 2.3 Chromosome conformation in isolated nuclei
57 Figure 2.5 Hi-C analysis reveals chromosome disassembly upon chromatin liquefication
Figure 2.6 Experimental protocol and computational workflow for DpnII-seq64 Figure 2.7 Average fragment size per bin and correlation with chromatin stability
Figure 2.8 Liquid chromatin Hi-C results are reproducible using the restriction enzyme Fatl
Figure 2.9 Variations in Half-life and LOS are not explained by DpnII digestion kinetics
Figure 2.10 Liquid chromatin-Hi-C protocol and quantification of loss of structure after chromatin pre-digestion
Figure 2.11 Kinetics of chromatin fragmentation and chromatin dissolution 76 Figure 2.12 Dissociation kinetics of chromatin interactions at different sub-
nuclear structures
interaction stability
Figure 2.15 Illustration of chromatin interaction dynamics in the nucleus and model for cohesin loss after chromatin digestion
Figure 3.1 Neural differentiation is associated with large-scale remodeling 158
Figure 3.2 Cell- and tissue-specific gene expression profiles
Figure 3.4 Cell type-specific features of the 3D genome
model systems
Figure 3.6 Loop/TAD size comparisons across multiple datasets
Figure 3.8 Cell type–specific chromosomal contact maps at schizophrenia risk loci

Figure 3.9 Epigenomic and genomic editing at schizophrenia risk-associated chromosomal conformations	
Figure 3.10 Expanded GWAS risk connectome is associated with gene	100
coregulation	186
Figure 3.11 Determining cell type-specific PGC interactions Figure 3.12 Expanded GWAS risk connectome is associated with significant	
gene coregulation and protein-protein association networks	190
Figure 3.13 Expanded GWAS risk connectome is linked to protein-protein	
association networks	195
Figure 3.14 NPC schizophrenia risk-associated protein-protein association	
network	
Figure 3.15 Neuron schizophrenia risk-associated protein-protein association	
network Figure 3.16 Glia schizophrenia risk-associated protein-protein association	197
network	199
Figure 3.17 NPC schizophrenia risk-associated STRING subset genes show	
greater transcriptional organization than full risk connectome gene list (PGC2)	
Figure 3.18 Neuron schizophrenia risk-associated STRING subset genes sho	
greater transcriptional organization than full risk connectome gene list (PGC2)	1
Figure 3.19 Glia schizophrenia risk-associated STRING subset genes do not	
show greater transcriptional organization than full risk connectome gene list	
(PGC2)	
Figure 4.1 ATLAS data statistics	
Figure 4.2 Results of predicting binding free energies in ATLAS Figure 4.3 Results of predicting binding free energies in ATLAS after modeling	
flexibility of CDR loops	•
Figure 4.4 ATLAS web interface and data accession	
Figure 5.1 Prediction of TCR-pMHC binding free energies	
Figure 5.2 Distributions of $\Delta GBIND$ for peptides recovered from different	
	331
Figure 5.3 Amino acid frequencies for top peptides selected by yeast display	
by computation for mouse and human TCRs	
Figure 5.4 Prediction comparison of scoring approaches.	337

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Because the history of evolution is that life escapes all barriers. Life breaks free. Life expands to new territories. Painfully, perhaps even dangerously. But life finds a way.

-Michael Crichton (1990), Jurassic Park

CHAPTER I: INTRODUCTION

Computational Biology

The origins of computational biology can be traced back to the early 1960s. Amino acid sequence arrangements of proteins were being determined for the first time. However, method limitations required larger protein sequences to be assembled by their smaller peptide sequence fragments. For large proteins composed of hundreds or thousands of amino acids, sequence assembly proved to be a combinatorial nightmare. To address this challenge Margaret Dayhoff and colleagues developed COMPROTEIN: a computer program to aid primary protein structure determination (Dayhoff and Ledley, 1962; Gauthier *et al.*, 2019). COMPROTEIN was one of the first programs described as bioinformatics software and was coded entirely on punch-cards.

As the sequences of more proteins were determined and technology advanced, computational biology expanded from its use in determining protein primary structure to predicting tertiary structure, the three-dimensional (3D) shape of a protein. Since its establishment in 1994, the Critical Assessment of protein Structure Prediction (CASP) initiative has held a prediction experiment every two years where dozens of groups across the globe implement computational methods with the goal of predicting target protein 3D structure from sequence (Moult *et al.*, 1995). Beyond protein studies, computational biology has become a fundamental component to the analysis of nucleic acids. The increase in computational biology pioneers and methods was driven largely by the publication of the human genome at the start of the 21st century and the advancements to next generation sequencing technologies which followed (Craig Venter *et al.*, 2001; Lander *et al.*, 2001). As sequencing costs decreased and data increased a need emerged for the computational biologist, capable of drawing connections in such vast and complex data.

Today, computational biology describes a field fundamental to the study of proteomics, transcriptomics, genomics, and epigenomics. From the processing of billions of paired-end DNA sequences to machine learning strategies for prediction of protein binding affinity, this thesis describes in detail the development of computational methods, models, and algorithms, to explain biological phenomena.

Genome structure

In the field of biology and beyond, the ability to visually represent what is hidden from the naked eye has been key to understanding how our world works.

The discovery that DNA forms a 3D double helix structure led to many critical scientific breakthroughs over the last sixty-seven years (James Watson and Crick, 1953). The knowledge that a single strand of the double helix can act as a template to code for a partner strand allowed us to answer the question of how DNA is replicated from cell to cell, and how inherited traits can be passed down from generation to generation (J Watson and Crick, 1953; Meselson and Stahl, 1958). The central dogma of molecular biology, that information is transferred from DNA and RNA to proteins but information cannot be transferred from a protein to DNA, was also predicated upon solving the 3D structure of DNA (F. H. Crick, 1958). Although the atomic structure of DNA has been known for some time, the larger picture illustrating how exactly the double helix is folded inside the cell nucleus still remains a puzzle.

Only within the last decade, with the aid of deep sequencing technologies, have we been able to begin unraveling the full 3D architecture of DNA in the context of how markedly long molecules of double helix DNA spatially fit inside the cell nucleus. The sheer size of a genome can make this question difficult to answer. The human genome is roughly three billion base pairs of DNA in length. Fitting all of that DNA inside a nucleus roughly 10 μ m in diameter is analogous to fitting 30 miles of string inside a basketball. The nucleus is packed with DNA; however, we now know that this packing is not an arbitrary random entanglement of the DNA polymer. From human to mouse to fly and even in single cell organisms, the 3D architecture of DNA inside the nucleus is highly organized in a manner to promote cell function (Lieberman-Aiden *et al.*, 2009; Duan *et al.*, 2010; Sexton *et al.*, 2012; Zhang *et al.*, 2012).

Although some general features of this 3D architecture are conserved across varying cell types and even across different species (Dixon *et al.*, 2012), changes in the 3D architecture of DNA have been associated with gene regulation, cell type specificity, and cell fate (Dixon *et al.*, 2015; Tang *et al.*, 2015; 4 Schmitt *et al.*, 2016). It follows that the mechanisms controlling the 3D architecture must be properly regulated and impairment of this architecture can lead to improper cell function and subsequent disease (Lupiáñez *et al.*, 2015).

A DNA molecule does not solely fold itself into its proper 3D conformation, but is aided by a myriad of DNA-binding proteins to form chromatin. In eukaryotes, the basic structural unit of chromatin is generated by the wrapping of 146 base pairs of DNA around eight histone proteins forming the nucleosome (Kornberg, 1974; Morse and Simpson, 1988). This wrapping of DNA into nucleosome structures not only helps to compact DNA molecules to fit inside the nucleus, but furthermore can act as method of gene regulation. Nucleosomes can inhibit transcription initiation and inversely, loss of nucleosomes can lead to transcriptional activation (Lorch, LaPointe and Kornberg, 1987; Han and Grunstein, 1988). Zooming out from the nucleosome level and examining full chromosomes, condensin complex proteins condense DNA to a further degree when preparing the cell for mitotic division (Hirano and Mitchison, 1994). The 3D architecture of chromatin inside the nucleus is never static, but instead a dynamic system that must replicate chromosomes followed by radical condensation and spatial chromosome segregation every cell cycle.

Visualization of mitotic cells through microscopy show chromosomes as condensed sausage like structures (Rieder and Khodjakov, 2003); however, the spatial occupancy of chromosomes during interphase is more difficult to resolve. Early microbeam radiation experiments, fluorescence microscopy results, and more recent deep sequencing data have all supported the now unequivocal conclusion that individual chromosomes again occupy separate territories during interphase of the cell cycle (Cremer *et al.*, 1982; Bolzer *et al.*, 2005; Lieberman-Aiden *et al.*, 2009).

While the existence of chromosome territories is clear, how certain features of the chromosome relate to the chromosome's spatial localization is still under debate. For instance, there is evidence for both random and non-random proximity of homologous chromosomes in somatic cells dependent upon cell type and species (Henikoff, 1997; Bolzer *et al.*, 2005). In humans, chromosome size has been associated with nuclear positioning with smaller chromosomes primarily located in the interior and larger chromosomes residing around the nuclear periphery (Sun, Shen and Yokota, 2000). However, this positioning may not be a result of chromosome size, but instead due to the clustering of gene rich active regions of chromosomes versus inactive gene poor regions. In agreement with this notion, the similar sized human chromosomes, 18 and 19, were shown to occupy different nuclear neighborhoods in lymphoblastoid cells. Specifically, gene rich Chromosome 19 was positioned near the nuclear center while the gene poor Chromosome 18 was closer to the periphery (Boyle, 2001).

Keeping active and inactive regions of the genome spatially segregated makes logical sense. Just as a city may cluster its financial district into a space separated from parks and housing to promote commerce, the cell may separate 6 gene rich from gene poor regions to promote transcriptional activity (Figure 1.1).

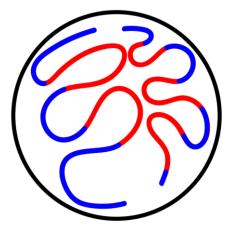


Figure 1.1 | Illustration of compartmentalization.

Nucleus (black) housing chromosomes with spatially separated active regions (red) and inactive regions (blue) of the genome.

Experimental evidence from the last couple of decades has made this hypothesis of nuclear compartmentalization undoubtable (Fraser and Bickmore, 2007; Lieberman-Aiden *et al.*, 2009). However, information regarding the stability of these compartments and the mechanisms governing compartmentalization is still lacking. In the next section, I review the tools and experiments used to identify compartments and the current hypotheses proposed regarding compartment formation and its properties.

Compartments

Fluorescence in situ hybridization (FISH) and microscopy techniques provided some of the first evidence for the spatial segregation of active and inactive chromatin domains into compartments. In FISH, hybridization of a fluorescent probe to a genomic region of interest facilitates visual localization of the genomic region within the nucleus. FISH studies in Drosophila showed that the eye color gene brown can relocate near inactive regions after an insertion of inactive chromatin in the *brown* gene itself. Silencing of wildtype *brown* due to its proximity to inactive chromatin provided further evidence supporting a functional role for compartmentalization (Dernburg et al., 1996). Similar FISH experiments in mouse indicate that active genes migrate toward specific nuclear regions promoting transcription, which may act as 'transcription factories' (Osborne et al., 2004; Shopland *et al.*, 2006). However, this model has been challenged by more recent studies which provide evidence for highly dynamic and transient transcription clusters (Cisse et al., 2013; Furlong and Levine, 2018). While FISH studies laid the groundwork for visualizing interphase compartmentalization patterns in eukaryotic cells, they were typically restricted to investigating a single locus or only a handful of loci. Later chromosome conformation capture experiments extended these clues from individual loci to validating a genome wide phenomenon.

Chromosome conformation capture (3C) experiments and their derivatives 8 allow for the detection of proximal DNA loci within a population of cells (Dekker *et al.*, 2002). In brief, these experiments apply the following three steps:

(1) Cells or isolated nuclei are crosslinked with formaldehyde, linking protein to protein and DNA to protein interactions throughout the nucleus. (2)
 DNA is digested with a restriction enzyme creating free ends of the cut DNA. (3)
 Free ends of cross-linked DNA fragments are ligated together.

The resulting DNA ligation products indicate a spatial proximity between the DNA loci on either end of the ligation junction. These loci can then be detected using specific primers in PCR reactions, microarrays, or through deep sequencing (Dekker *et al.*, 2002; Simonis *et al.*, 2006; Lieberman-Aiden *et al.*, 2009).

Chromosome conformation capture-on-chip (4C) extended the 3C method to interrogate the interaction profile of a single locus with the rest of the genome. This was accomplished by hybridizing PCR products from circular ligations on a microarray containing probes for loci scattered throughout the genome (Simonis *et al.*, 2006). 4C provided evidence that the active β -globin genes in fetal liver interact with other actively transcribed regions, and conversely the inactive β globin genes in fetal brain interact with other transcriptionally silent regions (Simonis *et al.*, 2006). The advent of the 3C/4C methods along with advances in microarray technology allowed for validation of the compartmentalization features seen in FISH by probing the interactions of one locus versus the rest of the genome. A few years later, a further derivative of the 3C technology, known as Hi-C, would harness deep sequencing technology to simultaneously interrogate all interactions between all genomic loci.

By adding a biotin incorporation step before ligation and subsequent pull down of ligation junctions followed by deep sequencing, the Hi-C method led to the production of the first chromatin contact map of the human genome at one megabase (Mb) resolution (Lieberman-Aiden et al., 2009). Chromosomal territories reported for interphase cells by previous microscopy studies were well represented in the genome wide contact maps, as loci within each chromosome (intra-chromosomal) were found to interact with much greater frequency than loci on different chromosomes (inter-chromosomal). Perhaps the most interesting observations came from chromatin contacts maps generated for intrachromosomal interactions. For example, examining the contact map of Chromosome 14, a striking checkerboard pattern emerged from the Hi-C data. Overlapping the checkerboard pattern with gene density, histone modification, and open chromatin tracks, it was evident the checkerboard pattern reflected the spatial preferences of compartmentalization. Dark squares of the checkerboard represented either preferential interactions between active genomic regions or the preferential interactions between inactive regions, while light squares represented the depletion of interactions between inactive and active regions. A principle component analysis on the Hi-C contact matrix was used to demarcate

boundaries between the spatially segregated active and inactive genomic regions, defining them as A and B compartments, respectively. (Lieberman-Aiden *et al.*, 2009).

Over the next decade, Hi-C contact maps have demonstrated that compartmentalization occurs across a wide array of human cell types (Schmitt *et al.*, 2016), in other mammals (Zhang *et al.*, 2012; Bonev *et al.*, 2017), in chicken (Gibcus *et al.*, 2018), in fly (Rowley *et al.*, 2017), and even in some plants (Dong *et al.*, 2017). Clearly, A/B compartmentalization of interphase nuclei is a conserved organizational principle (Jost, Bertulat and Cardoso, 2012) for many eukaryotes. Next, I will review the genetic and epigenetic properties of A/B compartment regions and introduce the current theoretical mechanisms proposed for compartment formation.

While direct evidence for genome wide segregation of active and inactive chromatin during interphase was not available until recently, many results from previous chromatin studies fall in line with this concept of compartmentalization and aid in distinguishing A compartments from their B counterparts. As early as 1929, Emil Heitz coined the terms *heterochromatin* and *euchromatin* to describe chromatin that appeared darker and lighter, respectively, on mitotic chromosomes stained with DNA dyes (Jost, Bertulat and Cardoso, 2012). Sedimentation experiments provided further evidence for two structurally distinct types of chromatin: condensed/closed heterochromatin and decondensed/open euchromatin (Gilbert *et al.*, 2004). These two structural states of chromatin are

also in line with gene positioning along chromosomes with euchromatin enriched 11 in genes and heterochromatin comprised primarily of gene deserts. Accordingly, A compartments are predominately composed of euchromatic regions and B compartments are predominately heterochromatin. It is important to note that the A/B compartment designations used here are defined strictly by the spatial segregation illustrated in Hi-C contact maps and although A compartments typically contain active genes there are exceptions to the rule (Vieux-Rochas *et al.*, 2015; Bonev *et al.*, 2017).

While genetic sequence alone may not be enough to accurately predict compartments regions, correlations can be made between sequence composition and A/B compartment designation. B compartments contain many transposable elements and are enriched in adenine (A) and thymine (T) nucleotides, while A compartments are generally more gene dense and enriched in guanine (G) and cytosine (C) (Slotkin and Martienssen, 2007; Lieberman-Aiden *et al.*, 2009; Imakaev *et al.*, 2012). Beyond sequence characteristics, epigenetic marks have shown high correlations with A/B compartments and provide further clues into compartment formation and function (Lieberman-Aiden *et al.*, 2009; Allis and Jenuwein, 2016; Allshire and Madhani, 2018).

Chemical modifications to the lysines of nucleosome histone tails can regulate gene expression by changing the local chromatin environment (Bannister and Kouzarides, 2011). Typically, acetylation of histone tails is associated with active transcription and an open chromatin environment, while methylation is associated with either transcriptional activation or repression dependent upon the methylation site. For example, the methylated histone marks, H3K4me3 and H3K36me3, are located near active regions (Bannister *et al.*, 2005; Barski *et al.*, 2007), while the marks, H3K27me3 and H3K9me3, are found in repressed regions (Becker, Nicetto and Zaret, 2016; Wiles and Selker, 2017). In some of the first 1 and 0.5 Mb scale contact maps, compartments were shown to correlate with active and repressive histone marks (Lieberman-Aiden *et al.*, 2009; Kalhor *et al.*, 2012). As technology increased and sequencing costs decreased, higher resolution Hi-C maps emerged further refining the compartmentalization phenomenon and its relation to the epigenetic chromatin landscape (Rao *et al.*, 2014; Rowley *et al.*, 2017).

Upon examining kilobase resolution contact maps comprised of billions of Hi-C contacts in human lymphoblastoid cells, spatial preferences appeared within and across the original A/B compartments (Rao *et al.*, 2014). The positions of these subcompartments (A1, A2, B1, B2, and B3) were found to correlate with specific histone modifications and proximity to different sub-nuclear structures revealing a higher order level of 3D chromatin organization.

A1 and A2 subcompartments were found to be enriched in the original A compartment and are both correlated with active histone markers such as H3K27ac, H3K9ac, and H3K36me3 (Rao *et al.*, 2014). However, in contrast to A2, the A1 subcompartment was found to specifically co-localize with nuclear speckles, which are hubs for pre-mRNA splicing factors (Chen *et al.*, 2018). The

co-localization of A1 chromatin with nuclear speckles would in effect bring actively transcribed regions to the machinery which processes transcribed products for nuclear export (Misteli, Cáceres and Spector, 1997), or conversely speckles may form at active genes to promote efficient processing.

B1, B2, and B3 subcompartments all correlate with inactive heterochromatic regions of the genome. B2 and B3 subcompartments are both depleted of active histone marks and enriched for lamin A/C, a protein localized at the nuclear periphery (Rao *et al.*, 2014). In contrast to B3, the B2 subcompartment was found to additionally and specifically co-localize with the nucleolus, where ribosome biogenesis occurs (Boisvert *et al.*, 2007). Unique from all other subcompartments, the B1 subcompartment is specifically enriched in the repressive histone mark H3K27me3 (Rao *et al.*, 2014). Polycomb group proteins trimethylate H3K27 to silence genes, a mechanism that is essential for proper vertebrate and invertebrate development (Aranda, Mas and Croce, 2015). Genomic loci bound by polycomb are shown to cluster inside the nucleus into visible polycomb bodies (Saurin *et al.*, 1998), validating preferential B1 interactions and suggesting a functional role for their subcompartmentalization.

Low (Mb) resolution interaction maps illustrated the spatial segregation of the genome into two compartments highly correlated with active and inactive regions. Later, higher (kb) resolution maps revealed chromatin state dictates a finer level of compartmentalization by the co-localization of regions with similar epigenetic marks or co-localization of regions interacting with specific subnuclear structures. While spatial compartmentalization of the genome is evident, 14 the mechanisms driving this compartmentalization remain ambiguous.

Evidence suggests transcription may be a driver for compartmentalization. Specifically, experiments inhibiting transcription have led to a reduction in compartmentalization (Rowley *et al.*, 2017). However, it is still debatable whether such reductions are a consequence of inhibiting transcription processes such as initiation and elongation or a consequence of loss in RNA polymerase II (Pol II) binding to DNA (Rowley and Corces, 2018).

Applying theories from polymer physics has led to some notable insights into compartment formation. Modeling chromosomes as polymers, a chain of elementary units called monomers, simplifies the complex conformational structure of chromatin. Specifically, the conformation a polymer can adopt relies on three characteristics: (1) flexibility of the chain, (2) interactions between monomers on the chain and (3) interactions with the polymer's surroundings (Rubinstein and Colby, 2003). A chromosome with alternating blocks of A and B compartments where each block may vary in size, can be modeled as a block copolymer. In a block copolymer, each block is comprised of consecutive A or consecutive B monomers. Simulations using block copolymers are capable of reproducing the checkerboard pattern displayed in Hi-C interaction maps (Haddad, Jost and Vaillant, 2017; Falk *et al.*, 2019). Importantly, such studies can provide estimations of attractive forces between A/B monomer types which aid in solving the puzzle of compartment formation. In contradiction to results that support transcription driving compartment formation, a recent study utilizing block 15 copolymer modeling to investigate compartmentalization in mouse described euchromatic A-A interactions as weak and more or less dispensable for compartmentalization. This study rather suggests that strong heterochromatic B-B interactions are responsible for genome compartmentalization via a phase separation mechanism (Falk *et al.*, 2019).

Similar to the phase separation of oil and water, recent studies have suggested that compartmentalization may be due to phase separation of compartment regions with the surrounding nucleoplasm (Hnisz et al., 2017; Larson et al., 2017; Strom et al., 2017). Liquid-liquid phase separation may occur through the oligomerization of soluble multivalent proteins (Erdel and Rippe, 2018). Self-associating oligomerization of these proteins can lead to a liquid-like droplet forming a phase separate from its surroundings. When these selfassociating proteins bind to chromatin they may induce compartmentalization by incorporating one compartment into the liquid-like droplet phase while excluding the unbound chromatin of the opposite compartment. For example, the multivalent protein HP1 α binds the H3K9me3 mark enriched in heterochromatin. In vitro binding of HP1 α with DNA can form liquid-like droplets (Larson et al., 2017). Furthermore, DNA curtain experiments reveal HP1 α proteins are capable of compacting DNA (Larson et al., 2017). Accordingly, B compartmentalization may be due in part to the liquid-liquid phase separation of heterochromatic regions bound by HP1 α .

Similar to blocking transcription, perturbing other elements intrinsic to a functional cell nucleus has led to changes in compartmentalization. For instance, chromatin is known to form loops spanning more than a megabase in genomic distance (Rao *et al.*, 2014). Degrading proteins vital to loop formation leads to an elimination of loops and a subsequent enhancement of compartmentalization (Rao *et al.*, 2017). These results suggest chromatin loops can act antagonistically to the compartmentalization phenomenon bringing A and B loci in closer proximity than attractive and repulsive forces between them would ordinarily allow.

In terms of understanding how compartments are formed and maintained, there is still much work to be done. Chapter II of this thesis focuses on answering the unknowns of compartmentalization and leads to the discovery of new principles and characteristics pertaining to compartmentalization of interphase nuclei. In the next section, I further introduce another 3D structure, the chromatin loop, and expand on its role in gene regulation, specifically in mediating promoter-enhancer interactions (Figure 1.2).

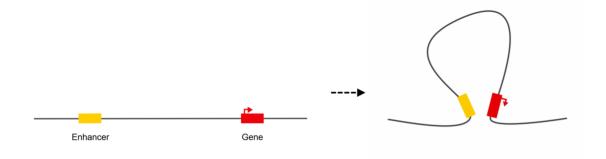


Figure 1.2 | Illustration of chromatin loop mediated promoter-enhancer interaction.

Chromatin loop formation allows a distal enhancer region (yellow) to regulate its target gene (red) by placing the enhancer region in close physical proximity to its target gene promoter.

Promoter-Enhancer Interactions

In eukaryotes, gene activation is defined by its transcription conducted by the Pol II multiprotein enzyme. As a prerequisite to gene activation, Pol II must first bind a promoter region proximal to the transcription start site (TSS) of a gene. Binding of Pol II and other transcription factors (TFs) to promoter regions leads to the initiation of transcription. Hence, occupancy of Pol II and TFs at promoters regulates gene activity (Andersson and Sandelin, 2020). In a similar fashion, enhancer regions distal from a gene in terms of linear sequence can activate a gene through recruitment of regulatory TFs. Enhancer function was identified as early as 1981 by transfection assays which showed a 72 bp repeat of viral DNA could increase activity of the β -globin gene across a distance of over 3 kb (Banerji, Rusconi and Schaffner, 1981). Later studies provided evidence enhancers can act on gene targets as far as a megabase away (Carter *et al.*, 2002; Lettice *et al.*, 2003). To reconcile how enhancers could regulate such distant targets, the 3D structure of chromatin at these regions was investigated.

3C studies at the β -globin locus revealed chromatin is capable of forming a loop structure linking a distal enhancer to its target promoter and activating β globin gene expression (Tolhuis *et al.*, 2002; Deng *et al.*, 2012). Formation of a chromatin loop could reconcile how distal enhancers can regulate their target genes. However, the relationship between chromatin loop structures and transcriptional activity is a topic still under much deliberation.

Identifying loop machinery and characterizing their modes of action in interphase nuclei has provided some insight into the role of chromatin loops in gene regulation. Two key components, CCCTC binding factor (CTCF) and the cohesin complex, have proven to be critical for the formation of many chromatin loop structures. CTCF is a highly conserved transcription factor capable of forming homodimers and known to recognize a sequence specific DNA binding motif (Nichols and Corces, 2015). The cohesin complex is a ring shaped protein complex known to regulate the separation of sister chromatids during cell division (Makrantoni and Marston, 2018). Together, CTCF and cohesin may mediate chromatin loop formation via a loop extrusion mechanism (Fudenberg *et al.*, 2016). In this model, binding of cohesin to chromatin leads to translocation of cohesin along the chromatin fiber causing extrusion of a small chromatin loop away from or out of the cohesin ring. Extrusion continues until cohesin is stalled by CTCF anchors bound to chromatin at opposite loop ends. Hence, in this model cohesin would bring the CTCF anchors of a loop close together in 3D space and loop size would be dependent on the 1D genomic distance between CTCF anchor sites. In support of the loop extrusion model, recent *in vitro* microscopy experiments show human cohesin complexes can form loops at up to 2.1 kb per second likely in a pseudo-topological or non-topological manner (Davidson *et al.*, 2019; Kim *et al.*, 2019).

Although CTCF/cohesin mediated loops may appear as a promising mechanistic candidate for the linking of promoters with enhancers, the relationship between CTCF/cohesin mediated loops and promoter-enhancer interactions is not as clear cut. Upon identification of loop structures in a high resolution Hi-C map of the human cell line GM12878, a majority of loop anchors are bound by CTCF (~86%) and cohesin (~86%) (Rao *et al.*, 2014). However, far fewer of these loops (~30%) actually linked together known promoters and enhancers. It follows, the functional role of CTCF/cohesin loops likely extends beyond the direct facilitation of promoter-enhancer interactions.

ChIA-PET experiments provide 3D chromatin interaction information between DNA loci bound by a protein of interest (Fullwood *et al.*, 2009). ChIA-PET experiments targeting CTCF have shown CTCF loop anchors are enriched for active epigenomic markers, Pol II occupancy, and TSSs of genes (Tang *et al.*, 2015). Furthermore, constitutively expressed genes were found to be enriched at CTCF/cohesin loop anchors, while tissue specific genes primarily resided within the body of loops suggesting a functional role for loops in mediating cell type specificity (Tang *et al.*, 2015). The binding motif of CTCF is non-palindromic and thus directional. The vast majority of CTCF loops identified in the human cell line GM12878 contained anchors with CTCF binding motifs in a convergent orientation (Rao *et al.*, 2014). Surprisingly, the directionality of transcription for genes with a TSS located in a CTCF loop anchor is in harmony with the CTCF motif orientation, again suggesting a regulatory role for CTCF/cohesin loops (Tang *et al.*, 2015).

Beyond associations between CTCF/cohesin loops and gene positioning or activity, mutagenesis studies have shown changes in gene expression can occur upon induced loop disruptions. Regulation of the *Shh* gene in mouse by the ZRS enhancer (located ~850 kb away from the *Shh* promoter) is vital to proper limb development. Disrupting CTCF/cohesin loop formation by inversion of a region encompassing the ZRS enhancer leads to downregulation of *Shh* in limb buds and malformation of limb structures (Symmons *et al.*, 2016). Interestingly, limb formation is partially rescued upon placing the inverted ZRS region in closer genomic distance to *Shh* (Symmons *et al.*, 2016). Similarly, inversion and deletion experiments disrupting CTCF loops at the *EPHA4* locus in mice causes ectopic interactions and expression patterns resulting in pathogenic phenotypes (Lupiáñez *et al.*, 2015). Hence, CTCF/cohesin loops may function to provide insulated neighborhoods within which dynamic movement of chromatin can link promoters and enhancers with a higher probability of interaction occurring based on linear genomic distance. In further support of this model, inversion of CTCF binding-sites away from the convergent orientation seen in loop formations can result in an altered 3D architecture at loop loci, loss of contacts between promoters and enhancers, and changes in gene expression (Guo *et al.*, 2015).

To further dissect the functionality of CTCF/cohesin mediated loops, recent studies have utilized the auxin-inducible degron system to degrade CTCF or a specific subunit of the cohesin complex (Nora *et al.*, 2017; Rao *et al.*, 2017). Depletion of CTCF or a subunit of the cohesin complex led to a loss of loop structures present in 3C based conformation maps (Nora *et al.*, 2017; Rao *et al.*, 2017).

Degradation of CTCF was shown to result in the differential expression of nearly 5,000 genes in mouse which was roughly evenly split between upregulated and downregulated genes (Nora *et al.*, 2017). Downregulated genes due to CTCF degradation were enriched in CTCF near promoters which suggested a role for CTCF/cohesin loop formations in promoting the activity of these genes. However, few of these genes overlap with loop structures that would connect their promoter to an active enhancer region. Hence, CTCF degradation is likely not directly disrupting promoter-enhancer loops formations. Instead of CTCF functioning to actively link promoter with enhancer, its role in gene regulation may be more often inhibitory. Indeed, when analyzing upregulated genes after CTCF degradation, a higher majority of upregulated 21

genes were genomically closer to active enhancers than down-or non-regulated 22 genes (Nora *et al.*, 2017). Such a result would support CTCF/cohesin loops as forming insulated neighborhoods preventing ectopic interactions between promoters and enhancers which reside in different loop domains.

Contrary to a model where CTCF/cohesin loops function to prevent ectopic promoter-enhancer interactions, degradation of a cohesin subunit resulted in a limited number of gene activations. Only roughly 1% of the unexpressed genes prior to cohesin subunit depletion were activated after subunit depletion (Rao *et al.*, 2017). Upon examining expressed genes before cohesin subunit depletion, the large majority of these genes presented similar expression levels after cohesin subunit depletion and subsequent loss in loop formations (Rao *et al.*, 2017). Hence, while a small subset of genes may be affected by a loss in CTCF/cohesin mediated loops, results from degradation studies imply CTCF/cohesin looping structures may play a limited role in gene regulation.

Beyond CTCF and cohesin, promoter-enhancer interactions may be mediated by other factors. The transcription factor Yin Yang 1 (YY1) is known to bind both enhancer and promoter elements and can form dimers. Deletion of YY1 binding sites or depletion of YY1 has been shown to disrupt chromatin loop formations and alter gene expression (Weintraub *et al.*, 2017). Another factor with evidence for facilitating promoter-enhancer interactions is the transcriptional coactivator mediator. Mediator forms a complex with cohesin capable of connecting distal genomic loci. 3C studies show occupancy of mediator and cohesin at promoter and enhancer elements predicts chromatin loop formation linking enhancers to the promoters of *Nanog*, *Oct4*, *Phc1*, and *Lefty1* in mouse (Kagey *et al.*, 2010). Aside from protein complexes, there is also evidence non-coding RNAs may play a role in stabilizing promoter-enhancer interactions (Lai *et al.*, 2013; Hsieh *et al.*, 2014). Many questions still remain regarding how these factors coincide with CTCF/cohesin loops and their effect on transcription.

Identification of promoter-enhancer interactions is fundamental to the mapping of regulatory pathways and critical to the dissection of abnormal regulation in diseased states. High resolution Hi-C interaction maps are a powerful resource for the detection of promoter-enhancer looping interactions (Rao *et al.*, 2014). A chromatin loop in a Hi-C map appears as a 'dot' of significant interaction intensity above its background neighborhood. These dots represent the high probability of interactions between anchors of loop loci, commonly CTCF sites brought together in close proximity by the cohesin complex in the loop extrusion model (Fudenberg *et al.*, 2016). From a computational standpoint, genome wide identification of these loci presents a significant challenge. Dots representing chromatin loops appear at a resolution of ~10 kb in a Hi-C interaction map. At this resolution, investigating every 3D interaction in a map of the human genome would require the investigation of over a hundred billion pairs of loci.

Several computational methods have been developed for the identification 24 of looping interactions in Hi-C interaction maps. These methods typically come in two flavors: (1) significant enrichment of looping interaction above global background (all interactions at equivalent genomic distance), or significant enrichment of looping interaction above a local background (interactions occurring in an \sim 50 kb radius surrounding looping interaction). In both methods, significant enrichment is determined using binomial or Poisson statistics and resultant *p*-values are corrected for multiple testing (Ay, Bailey and Noble, 2014; Rao et al., 2014; Mifsud et al., 2017). While these methods have been successful in identification of loops and promoter-enhancer contacts, the reproducibility of significant interactions between replicates was shown to be low for all methods (Forcato et al., 2017). Furthermore, the number of looping interactions identified was shown to be dependent upon sequencing depth, which could convolute results made from samples of differing coverage (Forcato et al., 2017). It is also important to note that identified significant interactions represent only 3D looping interactions which may or may not link a promoter and enhancer. As Hi-C interaction maps represent a population average, some dynamic promoterenhancer interactions may also fail to be identified as significant via such loop calling methods. It follows, while 3D conformations assays provide evidence for promoter-enhancer interactions, they may be most suitable when investigating candidate promoter and enhancer elements identified previously by other genomic assays.

As less than 2% of the human genome is comprised of protein coding sequences, annotating non-coding enhancer elements and their target promoters could lead to important discoveries in disease pathologies, particularly when disease causing genetic variations lie outside of coding genes (Alexander et al., 2010). It follows, the annotation of enhancer elements is a primary goal for many scientific groups including the Encyclopedia of DNA elements (ENCODE) consortium, a community consisting of dozens of research groups specializing in various fields of genomics (Dunham et al., 2012). Numerous computational methods have utilized assays that identify open chromatin regions (e.g. DNaseseq) or specific histone marks associated with enhancer activity (e.g. H3K27ac or H3K4me1 ChIP-seq) to identify enhancer elements (Ernst et al., 2011; Hoffman et al., 2012; Thurman et al., 2012; Rajagopal et al., 2013; Kundaje et al., 2015). In some cases, enhancers do not always activate their nearest gene in terms of linear sequence space complicating the mapping of enhancers to their gene targets (Lettice et al., 2003). This difficulty in linking promoters with enhancers has been resolved via chromosome conformation assays similar to methods described earlier (Li et al., 2012; Mifsud et al., 2015) or by correlating genomic and epigenomic signals at enhancers and promoters across a range of varying biosamples (Ernst et al., 2011; Thurman et al., 2012). Such efforts have led to lists of candidate regulatory enhancer elements in a variety of different tissue types. However, while genetic or epigenetic marks along with 3D proximity information may be predictive of enhancer activity such assays do not provide a

25

ground truth for enhancer identification. To validate an enhancer one must show 26 its presence definitively leads to the expression of its target gene.

The canonical method for validating enhancer activity is to clone an enhancer sequence upstream of a minimal promoter of a reporter gene (e.g. *LacZ*) and analyze reporter gene expression. Using such methods *in vivo*, the VISTA enhancer browser contains results from the interrogation of hundreds of enhancer sequences (Visel *et al.*, 2007). Here, the enhancer reporter construct is microinjected into a fertilized mouse egg followed by implantation into a female mouse. *LacZ* staining of the harvested mouse embryo reveals tissue specific enhancer activity (Visel *et al.*, 2007). A drawback to such transgenic mouse assays, is that transfection of the enhancer reporter construct leads to random integration in the mouse genome. Hence, while results from reporter assays validate enhancer function they do not take into account the enhancer's native chromatin context.

The CRISPR/Cas9 genome editing system can be utilized to delete genomic sequences inside the nucleus of the cell (Cong *et al.*, 2013; Yao *et al.*, 2014; Won *et al.*, 2016). This is accomplished by the design of single-guide RNAs (sgRNAs) complementary to regions flanking the DNA sequence selected for deletion. The binding of sgRNA to a catalytically active Cas9 protein guides Cas9 to a site flanking the selected DNA sequence for subsequent Cas9 mediated cleavage. To validate enhancer function, this method can be adapted to deletion of a candidate enhancer element. Quantitative measuring of gene expression for the candidate enhancer target before and after CRISPR/Cas9 mediated deletion of the enhancer sequence provides validation of enhancer function within the enhancer's original chromatin environment (Yao *et al.*, 2014; Won *et al.*, 2016).

Even when applying current methods and technologies, identification and validation of promoter-enhancer interactions remains a significant challenge. Chapter III of this thesis utilizes Hi-C experiments to assay the 3D genome architecture in human glial cells, neural progenitor cells and neurons. The resultant Hi-C interaction maps are used to predict cell-type specific promoter-enhancer interactions occurring at or near schizophrenia risk variants. Upon identification of risk variants in close spatial proximity to potential gene targets, CRISPR/Cas9 strategies are applied to test the regulatory functions of risk variant loci.

Schizophrenia, Neurons, and 3D organization

Symptoms of schizophrenia include hallucinations, delusions, disorganized speech, and social withdrawal, along with a broad set of cognitive dysfunctions (Kahn *et al.*, 2015). Schizophrenia is not considered a rare disorder. Studies of disease prevalence estimate about seven individuals per one thousand will develop schizophrenia during their lifetime (McGrath *et al.*, 2008). The disorder has also been shown to have an effect on average life span. Based on the standardized mortality ratio, individuals diagnosed with schizophrenia have a two to threefold increased risk of dying with suicide as a main contributing 28 factor (McGrath *et al.*, 2008).

Current treatments of schizophrenia rely on the action of antipsychotic drugs such as chlorpromazine or clozapine which block receptors in the dopamine pathway (Kane and Correll, 2010; Miyamoto *et al.*, 2012). While such antipsychotic drugs help to manage patient symptoms, in many cases, patients symptoms are either resistant to treatment or successful symptom treatment is accompanied by adverse side effects (Lally and MacCabe, 2015). It follows, specialized treatments taking into account patient specific characteristics may result in healthier patient outcomes. However, the lack of knowledge in regard to the etiology of schizophrenia presents a major challenge to the advent of improved or specialized therapeutics to treat the disorder.

While the etiology of schizophrenia is largely unknown, evidence suggests that along with environmental factors the disorder carries a heritable genetic component. In a study of over 30,000 twin pairs born in Denmark with 448 twin pairs affected by schizophrenia, heritability of the disorder was estimated to be 79% in accordance with previous studies (Sullivan, Kendler and Neale, 2003; Hilker *et al.*, 2018). Both rare and common allele variants have been implicated in the pathology of schizophrenia (McClellan, Susser and King, 2007; Purcell *et al.*, 2009). To further explore this landscape of genetic variants, large scale genome-wide association studies (GWAS) have been employed to search for SNPs associated with schizophrenia. A recent GWAS encompassing genotypes

from 36,989 schizophrenia cases and 113,075 controls reported 108 risk loci harboring risk variants significantly associated with the disorder (Ripke *et al.*, 2014). Expanding upon this study, Pardiñas and colleagues performed GWAS analysis on an additional 11,260 cases and 24,542 controls, which resulted in the identification of 50 additional novel risk loci (Pardiñas *et al.*, 2018). When risk loci were overlapped with epigenetic marks for active enhancers, risk SNPs were found to be enriched in enhancers active in brain (Ripke *et al.*, 2014).

The enrichment of schizophrenia risk SNPs at active enhancers in brain suggests disruption of regulatory mechanisms may underlie disorder pathology. Such a model would assign a possibility of risk to noncoding variants which, unlike coding variants, are incapable of directly altering the amino acid composition of a protein product. Supporting this model, schizophrenia risk variants were shown to be enriched for alleles that affect gene expression and lie within promoter or enhancers (Roussos *et al.*, 2014). Expression quantitative trait loci (eQTLs) are defined as loci containing variants which alter gene expression (Nica and Dermitzakis, 2013). A schizophrenia associated eQTL within the intron of its target gene was found to be in closer spatial proximity to its target gene TSS compared with other sequences closer in genomic space (Roussos *et al.*, 2014). Furthermore, this eQTL variant locus resided in an enhancer region suggesting potential promoter-enhancer looping mechanisms may be at play.

It is plausible that eQTL variants located in enhancer regions could alter their gene target's expression by disrupting binding of transcription factors to 29

enhancer loci, disrupting the local chromatin state of the enhancer neighborhood, 30 or by disrupting promoter-enhancer loop formation, none of which are necessarily mutually exclusive events. In further support of risk variants modulating gene expression, complex trait associated SNPs are significantly more likely to be eQTLs than minor-allele-frequency matched SNPs (Nicolae *et al.*, 2010). Additionally, when overlapping GWAS identified schizophrenia risk loci with an independent eQTL dataset generated from RNA sequencing of dorsolateral prefrontal cortex from both schizophrenia and control cases, results indicated ~20% of schizophrenia risk loci have variants that could contribute to altered gene expression (Fromer *et al.*, 2016).

Genetic regulatory pathways contributing to schizophrenia pathology may have cell type specific characteristics. Neurons and glial cells make up two broad classes of cells in the nervous system. Neurons are electrically excitable capable of relaying information through electrical impulses, while glial cells provide mechanical and metabolic support for neurons among other functions (Squire *et al.*, 2008). Recent studies have shown a preferential link between schizophrenia risk architecture and the neuron cell type (Genovese *et al.*, 2016; Skene *et al.*, 2018). Correlations between cell type specificity and enrichment for schizophrenia SNP heritability across genes, associated neurons with schizophrenia risk architecture over embryonic, progenitor or glial cells (Skene *et al.*, 2018). Similarly, an analysis of ultra-rare variants, reported individuals with schizophrenia were enriched for ultra-rare variants in genes that were neuronally expressed (Genovese *et al.*, 2016). It follows that an analysis of cell type specific 31 gene regulation systems may lead to pivotal observations concerning schizophrenia etiology.

Human induced pluripotent stem cell (hiPSC) technology has advanced the study of human disease mechanisms in vitro. Over a decade ago it was discovered adult mice fibroblasts can be reprogrammed into pluripotent stem cells by the introduction of four transcription factors: Oct3/4, Sox2, c-Myc, and Klf4 (Takahashi and Yamanaka, 2006). Building upon this, Brennand and colleagues reprogrammed fibroblasts from schizophrenia patients into hiPSCs followed by subsequent differentiation into both neural progenitor cells (NPCs) and neurons (Brennand et al., 2011). RNA analysis of control and schizophrenia hiPSC neurons showed differential expression in several genes characterizing potential regulatory mechanisms of the disorder (Brennand et al., 2011). Further advancements in hiPSC technology has led to the differentiation of hiPSC derived NPCs by overexpression of the neuronal transcription factor, NGN2, producing near pure populations of postmitotic neurons capable of forming mature pre and post-synaptic formations (Ho et al., 2016). Alternatively, hiPSC derived NPCs may also be differentiated into astrocytes of the glial lineage capable of responding to inflammatory stimulants and displaying phagocytic capacity (TCW et al., 2017). Such methods provide new systems for testing the effects of differentiation and cell type specificity on regulatory gene networks and 3D genome organization.

Gene expression changes influence determination of cell type and cell fate 32 (Dunham et al., 2012; Wang et al., 2018). However, much is still unknown regarding the mechanisms of cell type specific regulation and the related role of 3D genome architecture. Many 3D genome conformations are conserved across a variety of cell types, tissues, and even syntenic regions between species (Dixon et al., 2012; Schmitt et al., 2016). While 3D genome architecture is highly conserved, cell type specific changes in 3D architecture such as compartment switching and altered looping contacts have been reported (Rao et al., 2014; Schmitt et al., 2016; Won et al., 2016). Such changes have also been described throughout the course of development. A/B compartment switching was observed upon differentiation of human embryonic stem cells (ESCs) into four other ESCcell-derived lineages (Dixon et al., 2015). Differentiation of mouse ESCs to neural stem cells resulted in a widespread gain of chromatin loop formations (Pekowska et al., 2018). High resolution Hi-C maps of mouse ESCs, derived NPCs and further derived cortical neurons revealed dynamic chromatin looping formations occur near genes encoding neural transcription factors in a cell type specific manner which correlated with gene expression (Bonev et al., 2017). Further experiments associating cell type specific regulatory programs with changing 3D architecture will help unravel the mechanisms driving cell fate and likely elucidate discoveries related to diseases driven by malfunctions in specific cell types.

Won and colleagues, characterized 3D genome architectures in human brain development in the context of associating the 3D genome with schizophrenia risk variants (Won *et al.*, 2016). This study generated Hi-C maps of bulk tissue from the germinal zone (primarily neural progenitor cells) and cortical plate (primarily adult neurons). In Chapter III, we expand upon this study by applying hiPSC technology to produce high resolution Hi-C maps of hiPSC derived NPCs, glial cells and neurons. Such contact maps allowed for analyses revealing cell type specific chromatin conformation changes which we further studied in the context of schizophrenia risk.

In the next section, I move away from the 3D genome and introduce background relevant to Chapters IV and V, which describe computational methods for prediction of T cell receptor antigens via 3D structural information.

TCR-pMHC Interactions

In humans and other vertebrates, T cells and B cells cooperate in the adaptive immune system to protect the body from invading pathogens. Immunological protection is facilitated via recognition, elimination and memory of such pathogens (Owen *et al.*, 2013). While B cells produce antibodies which can directly bind pathogen antigens and signal for their destruction, most T cells recognize antigens displayed by specific proteins termed major histocompatibility complex (MHC) molecules, on the surface of antigen presenting cells (Zinkernagel and Doherty, 1974). Upon recognition of pathogenic antigens displayed on infected cells, T cells may initiate the direct killing of such infected cells or trigger other immune cells to attack the identified pathogen. Before a T cell is equipped to successfully and specifically target infected 34 cells, each T cell must be tested for competence during development. T cell precursors originating in the bone marrow travel to the thymus via the bloodstream to undergo critical developmental processes including: T cell receptor formation, positive selection and negative selection (Koch and Radtke, 2011).

T cells use T-cell receptors (TCRs) to recognize antigens displayed by MHC molecules on infected cells. The TCR is made up of two chains linked by disulfide bonds on the cell surface. These chain pairs are typically α and β , or γ and δ , which are encoded by the four genetic loci TRA, TRB, TRG, TRD, respectively. $\alpha\beta$ T cells represent the dominant participants in adaptive immune functions and will be the focus of work presented in this thesis. In order to recognize a diverse and broad range of hazardous antigens, $\alpha\beta$ T cells in the blood encompass a large repertoire of variable $\alpha\beta$ TCRs, estimated to be on the order of 10⁷ unique $\alpha\beta$ TCRs per human (Arstila *et al.*, 1999). This large assortment of variable TCRs is accomplished by V(D)J recombination events during thymic development.

Within each TRA and TRB locus, multiple genes exist to encode single domains of the TCR chain. For example, in humans, the TRB locus contains over 60 genes encoding the variable domain of the TCR β chain (Lefranc, 2011). V(D)J recombination of these genes leads to the translation of a functional chain encoded by a random subset of these genes (Hozumi and Tonegawa, 1976).

Nucleotide deletions and insertions during the recombination process, and combinatorial $\alpha\beta$ pairing, leads to further diversification of the TCR repertoire. Hence, before successful exit from the thymus, each T cell will express a distinct TCR, such that a population of mature T cells will be adequate for recognition of variable pathogenic antigens.

Once a T cell has formed a TCR, functional testing of the TCR takes place during positive selection. Here, further maturation of the T cell is dependent upon the affinity between the TCR and MHC molecules of an antigen presenting cell displaying self (non-foreign) antigens. If binding between TCR and self-antigen-MHC is not strong enough, the developing T cell will not be positively selected and instead undergo apoptosis (Boehmer and Kisielow, 1990; Starr, Jameson and Hogquist, 2003). Therefore, positive selection functions to ensure the population of mature T cells are capable of recognizing antigen-MHC targets. However, strong recognition of self-derived antigens is hazardous, as this could lead to an immune response and attack against healthy cells and tissues. To ensure the mature T cell engages the correct targets, negative selection deletes T cells with too strong an affinity for self-antigen-MHC complexes (Boehmer and Kisielow, 1990; Starr, Jameson and Hogquist, 2003). Finally, if a T cell survives both positive and negative selections in the thymus, it is then recruited to secondary lymphoid organs such as the lymph node to patrol for harmful invaders.

The first step toward an immune response occurs via recognition of antigen-MHC by a patrolling TCR. These MHC molecules are highly polymorphic with over 25,000 recorded human MHC alleles (Robinson *et al.*, 2020). The high degree of polymorphism has been attributed to evolutionary causes, whereby infectious diseases may drive MHC polymorphism (Radwan *et al.*, 2020). Antigen presenting MHC molecules are categorized into distinct classes. Class I MHC molecules are comprised of a single α chain noncovalently bound to a β_2 microglobulin protein. These molecules are expressed on the surface of the majority of nucleated cells in the body, and they display peptide fragments from endogenous antigens degraded in the cytosol. In contrast, class II MHC molecules are comprised of an α chain and a noncovalently associated β chain, are expressed in a limited set of cell types, and they display peptide fragments from exogenous antigens endocytosed into the cell (Owen *et al.*, 2013).

During thymic development T cells also commit to a specific cell lineage by expression of either cluster of differentiation 4 (CD4) or cluster of differentiation 8 (CD8) coreceptor molecules. These coreceptors maintain specific binding preferences, such that CD4+ T cells interact with MHC class II molecules and CD8+ T cells interact with MHC molecules of class I (Germain, 2002). Upon recognition of antigen, these T cell lineages have varying effector functions. CD4+ T cells (helper T cells) help to activate an immune response by stimulating responses in other cells; for example, helper T cells can stimulate B cells to produce antibodies to opsonize pathogenic targets (Crotty, 2015). Alternatively, CD8+ T cells (cytotoxic T cells) are capable of directly killing infected cells; for example, cytotoxic T cells may release serine proteases into the target cell triggering caspase activation and cell death (Barry and Bleackley, 2002). Hence, the MHC class preference of T cell coreceptors also functions to guide T cell specific effector functions toward their appropriate target cell type. Given the larger setting of how T cells mount an immune response, the remainder of this introduction will focus on a specific step in the immune response, namely the interaction between TCR and peptide-MHC (pMHC) (Figure 1.3).

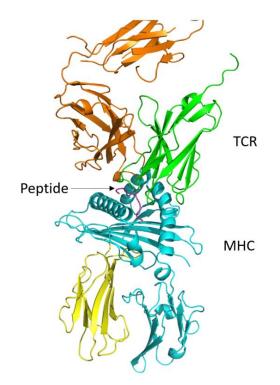


Figure 1.3 | Crystal structure of TCR-pMHC complex.

Crystal structure of the human A6 TCR (α chain:green, β chain: orange) recognizing the Tax peptide (magenta) and MHC allele HLA-A*02:01 (cyan). β_2 -microglobulin is shown in yellow (Garboczi *et al.*, 1996).

In 1987, Bjorkman and colleagues successfully determined the first MHC structure, the human HLA-A2 molecule. The crystal structure revealed membrane distal domains formed a β -sheet platform topped by α -helices with a long groove between the helices, likely representing the location for bound peptide antigen (Strominger *et al.*, 1987). Determination of the class I MHC structure led to many hypotheses concerning the docking orientation of an interacting TCR. It was not until nearly a decade later that human and mouse TCR-pMHC complex structures would be solved, and these provided answers to previous hypotheses (Garboczi *et al.*, 1996; Garcia *et al.*, 1996). In both reports, the TCR was found to dock in a conserved diagonal orientation over the pMHC molecule surface (Figure 1.3).

The variable domain of each TCR α and β chain contains three complementarity-determining region (CDR) loops. CDR1 and CDR2 loops are encoded by the variable gene segment, while the CDR3 loops are encoded by the junction of variable and joining (or variable, diversity, and joining) gene segments, leading to a greater diversity in CDR3 (Hughes *et al.*, 2003). The solved TCR-pMHC structures showed that the CDR3 loops were primarily positioned to contact the peptide antigen, whereas the less diverse CDR1 and CDR2 loops were positioned closer to the MHC helices. Placing the more diverse TCR region over the peptide makes logical sense, as this could help improve discrimination against the vast space of foreign peptide antigens. Future studies 38

have shown a consensus of diagonal docking topologies for TCR interactions between class I and class II pMHC complexes, although atypical docking topologies do exist (Rossjohn et al., 2015).

Due to negative selection, TCRs bind pMHC molecules with weak affinities and short half-lives in comparison with antibody-antigen affinities. Dissociation constants, K_Ds, from TCR-pMHC binding assays are in the micromolar range ($K_D = 0.1 \mu M - 500 \mu M$) and half-lives are on the order of seconds (Cole et al., 2007, 2013; Bridgeman et al., 2012).

Structural features of the TCR-pMHC interface have been associated with affinity changes. The α -helices which cradle the peptide in MHC class I molecules are closed at their ends in contrast to an open conformation in MHC class II structures (Brown et al., 1993). This structural feature conforms to MHC class I molecules displaying peptides with lengths typically ranging from eight to ten amino acids, while class II molecules can display peptides with lengths beyond fourteen amino acids, protruding from open ends of the MHC binding groove (Bjorkman, 2015). The different antigen surfaces of pMHC classes are associated with a change in binding affinity by interacting TCRs. Specifically, TCR-pMHC affinities for class I pMHC molecules are stronger, on average, compared with class II pMHC molecules. Changes in affinity were ascribed to significantly greater on-rates for TCRs binding with class I pMHC compared with class II pMHC, while similar off-rates were reported (Cole et al., 2007).

Aside from structural distinctions due to MHC class, weaker affinities for 40 pMHC have also been noted for autoreactive TCRs binding self peptides compared with canonical TCRs engaging foreign peptides (Bridgeman *et al.*, 2012). This observation may be in concordance with thymic selection, whereby the weak binding of such autoreactive TCRs works to prevent harmful autoimmune responses.

Structural conformations of the TCR also play a role in determining binding affinity. Increased contacts made between the TCR CDR3 loops and peptide result in higher affinity TCR-pMHC interactions (Cole *et al.*, 2013). Furthermore, structure based design methods have led to the development of TCR mutants capable of binding pMHC up to 400 times more strongly than wildtype TCR (Haidar *et al.*, 2009; Zoete *et al.*, 2013; Malecek *et al.*, 2014; Pierce *et al.*, 2014). Collectively, such studies support the hypothesis that 3D interactions at the TCR-pMHC interface can be used to inform studies aimed at altering TCR-pMHC binding affinity.

Binding affinity for TCR-pMHC complexes can be determined using surface plasmon resonance (SPR) or isothermal titration calorimetry (ITC) experiments. In SPR experiments, binding affinity is quantified by optically measuring how fast light travels through a sensor surface (i.e. measuring the refractive index of the sensor surface). Here, the sensor surface makes up the floor of a flow cell, through which an aqueous solution can pass under continuous flow. To detect binding, a ligand (e.g. pMHC) is immobilized onto the sensor surface, while an analyte (e.g. TCR) is flowed over the ligand bound surface. As 41 the analyte binds to the ligand, an accumulation of binding complexes on the surface leads to a change in the refractive index of the surface. Analyzing these changes in refractive index over time and at different concentrations of analyte leads to determination of analyte-ligand binding affinity, K_D (Merwe, 2000). Similarly, ITC experiments can be used to derive K_D values for TCR-pMHC interactions by directly measuring changes in enthalpy. However, ITC is rarely used for analyzing TCR-pMHC binding due to the much larger amounts of protein required for the study in comparison to SPR (Miller *et al.*, 2007). Indeed, the vast majority of affinity values used for training and testing in Chapter IV were obtained from SPR experiments.

Several methods exist for the computational prediction of TCR-pMHC interactions. These methods can be categorized as either sequence based or structure based depending on the information used for prediction. Sequence based methods rely on machine learning algorithms that input sequence features of TCRs, peptides, and MHC molecules during model training. Such methods have been utilized to predict immunogenicity of a peptide or to predict reactivity of a TCR sequence (Tung *et al.*, 2011; Gielis *et al.*, 2019). Structural based methods rely on energy force fields and scoring functions, sometimes in combination with machine learning, to predict TCR-pMHC interactions. Such methods have similarly been utilized to predict immunogenicity of a peptide, but

also to predict TCR-pMHC binding affinity (Pierce and Weng, 2013; Lanzarotti, 42 Marcatili and Nielsen, 2018; Schneidman-Duhovny *et al.*, 2018).

Although TCR-pMHC binding is a prerequisite for an immune response, binding affinity does not directly confer immunogenicity (Stone and Kranz, 2013). Extracellular TCR-pMHC binding causes a cascade of intracellular signaling events which eventually leads to activated transcription factors initiating immune response programs (Owen *et al.*, 2013). Experimental assays measuring T cell proliferation, cytotoxicity, or cytokine secretion are vital toward unraveling the larger picture of antigen immunogenicity.

In Chapters IV and V, I focus on the TCR-pMHC interaction in the context of antigen prediction via structural properties. Chapter IV introduces the development of a publicly available database, Altered TCR Ligand Affinities and Structures (ATLAS), linking 3D TCR-pMHC complexes with their experimentally measured binding affinities. Regression analyses performed with data from ATLAS were used to predict hundreds of TCR-pMHC binding energies. In Chapter V, we repurpose deep sequencing data to expand previous antigen predictions methods by investigating over two million modeled TCR-pMHC interactions.

CHAPTER II: COMPARTMENT-DEPENDENT CHROMATIN INTERACTION DYNAMICS REVEALED BY LIQUID CHROMATIN HI-C

Preface

This chapter is adapted from a manuscript currently under review at Nature Genetics authored by Houda Belaghzal, myself, Andrew D. Stephens, Denis L. Lafontaine, Sergey Venev, Zhiping Weng, John F. Marko, and Job Dekker titled: Compartment-dependent chromatin interaction dynamics revealed by liquid chromatin Hi-C.

The project was conceived by Job Dekker. 3C, 5C, Hi-C and liquid chromatin Hi-C and chromatin fractionation experiments were performed by Houda Belaghzal. Restriction digestion efficiency (DpnII-seq) experiments were performed by Denis L. Lafontaine. Micromechanical studies and their analysis was performed by Andrew D. Stephens. Data analysis was performed by myself and Houda Belaghzal. Specifically, I computationally processed liquid chromatin Hi-C sequencing datasets, developed the liquid-chromatin-Hi-C computational analysis toolkit to calculate LOS and t_{1/2} stability metrics, developed the DpnII-seq computational pipeline, processed and analyzed fragment size assessment sequencing data, and performed the sub-nuclear structure analysis. Tools for liquid chromatin Hi-C analysis were contributed by Sergey Venev. Polymer scaling ideas relevant to data interpretation were provided by John F. Marko. The

paper was written and figures produced by Houda Belaghzal, myself, and Job 44 Dekker with contributions from all coauthors.

Abstract

Nuclear compartmentalization of active and inactive chromatin is thought to occur through microphase separation mediated by interactions between loci of similar type. The nature and dynamics of these interactions are not known. We developed liquid chromatin Hi-C to map the stability of associations between loci. Before fixation and Hi-C, chromosomes are fragmented, removing the strong polymeric constraint to enable detection of intrinsic locus-locus interaction stabilities. Compartmentalization is stable when fragments are over 10-25 kb. Fragmenting chromatin into pieces smaller than 6 kb leads to gradual loss of genome organization. Lamin-associated domains are most stable, while interactions for speckle and polycomb-associated loci are more dynamic. Cohesin-mediated loops dissolve after fragmentation. Liquid chromatin Hi-C provides a genome-wide view of chromosome interaction dynamics.

Introduction

Genomic and imaging approaches are producing high-resolution descriptions of the conformation of chromosomes in cell populations, in single cells, across the cell cycle, and during development (Lieberman-Aiden et al., 2009; Bickmore and Van Steensel, 2013; Nagano et al., 2017; Naumova et al., 2013; Nagano et al., 2013; Rao et al., 2014; Bonev and Cavalli, 2016; Wang et al., 2016; Dekker and Mirny, 2016; Ramani et al., 2017; Dekker et al., 2017; Hug et al., 2017; Nir et al., 2018; Chen et al., 2018; Gibcus et al., 2018; Kaaij et al., 2018). At the mega-base (Mb) scale chromosomes are compartmentalized and different types of chromosomal domains can be discerned. Hi-C interaction maps display a "plaid" pattern, which reflects the segregation of the genome in two major spatial compartments referred to as A and B compartments that correspond to open, active chromatin and closed, silent chromatin, respectively (Simonis et al., 2006; Lieberman-Aiden et al., 2009). High-resolution (kb) Hi-C maps allowed splitting compartments in 5 subtypes (A1, A2, B1, B2, and B3) that differ in interaction patterns and chromatin state (Rao et al., 2014). At the scale of tens to hundreds of kb, topologically associating domains (TADs) were identified as domains separated by boundaries that are in many cases bound by CTCF. Higher resolution Hi-C (Rao et al., 2014), ChIA-PET (Tang et al., 2015), and 4C data (de Wit et al., 2015; Guo et al., 2015; Vietri Rudan et al., 2015) showed that convergent CTCF sites at boundaries can engage in looping interactions.

Major questions revolve around the molecular and biophysical processes by which different aspects of chromosome conformation form. TADs and loops between CTCF sites form via loop extrusion cohesin (Riggs, 1990; Nasmyth, 2001; Alipour and Marko, 2012; Sanborn *et al.*, 2015; Fudenberg *et al.*, 2016, 2018; Rao *et al.*, 2017). Less is known about the processes that determine compartmentalization. Compartmentalization has been proposed to be the result of polymer phase separation driven by attractions between chromatin domains of 45

the same or similar status (Lieberman-Aiden *et al.*, 2009; Jost *et al.*, 2014; Di Pierro *et al.*, 2016; Michieletto, Orlandini and Marenduzzo, 2016; Erdel and Rippe, 2018; MacPherson, Beltran and Spakowitz, 2018; Nuebler *et al.*, 2018; Shi *et al.*, 2018; Falk *et al.*, 2019). Polymer models simulating such attractions can reproduce the plaid pattern characteristic of Hi-C interaction maps (Jost *et al.*, 2014; Di Pierro *et al.*, 2016; Nuebler *et al.*, 2018; Falk *et al.*, 2019).

Hi-C interaction maps are steady-state datasets and do not reveal the biophysical nature of the interactions that drive compartment formation or the dynamic mobility of loci within them. Live cell imaging studies have shown that loci are constrained in their motion and that there is variation in the dynamics and mobility of loci, e.g. euchromatic vs. heterochromatic loci and loci tethered to the nuclear periphery vs. loci located in the nuclear interior (Marshall et al., 1997; Hediger et al., 2002; Thakar, Gordon and Csink, 2006; Bronshtein et al., 2009, 2015; Therizolsa et al., 2010; Shinkai et al., 2016; Nagashima et al., 2019). Imaging-based studies have been instrumental in uncovering aspects of chromatin interactions and dynamics, but are limited in scale, i.e. only one or a few specific loci can be studied at one time. In addition, when whole genome dynamics are analyzed microscopically (e.g. (Zidovska, Weitz and Mitchison, 2013)), positions of specific sequences have not yet been determined. Therefore, new approaches are required to identify and quantify the molecular processes and biophysical forces involved in chromatin interactions and nuclear compartmentalization. Here we describe liquid chromatin Hi-C, a Hi-C variant

that quantifies the stability of chromosome conformation and chromatin interactions genome-wide.

Results

Measuring stability of chromatin interactions and nuclear

compartmentalization

The formation of spatially segregated heterochromatic and euchromatic domains can be viewed as microphase separation of a polymer composed of different types of monomers (loci). A "block copolymer" is a polymer that contains a series of alternating blocks (e.g., A-type and B-type, or blocks of euchromatin and heterochromatin), each composed of multiple monomers (A monomers and B monomers; Figure 2.1, A). When As attract As and Bs attract Bs, such polymer can fold into spatially segregated domains of As and Bs (Figure 2.1, A, (de Gennes, 1979; Leibler, 1980; Matsen and Schick, 1994)). Applied to chromatin in vivo, microphase separation may underlie the formation of segregated compartments. The biophysical forces and interaction dynamics that determine chromosome compartmentalization are not known.

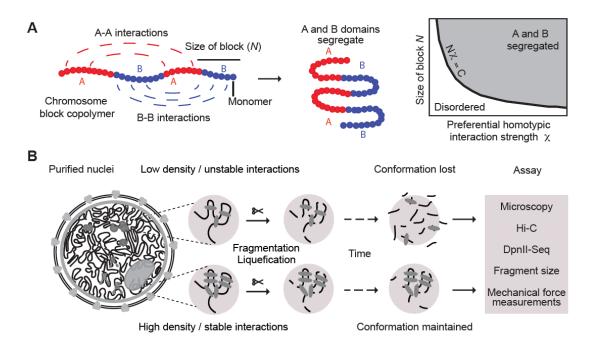


Figure 2.1 | Approach for measuring chromatin interaction stability.

(A) Block copolymer composed of a series of alternating A and B blocks, each composed of a number of monomers (left). The polymer can fold into spatially segregated domains of As and Bs (middle). Flory-Huggins polymer theory predicts that spatial segregation will occur when the product of the length of the blocks N (the number of monomers that make up blocks) and their effective preferential homotypic interaction strength χ (difference in the strength of homotypic interactions as compared to heterotypic (A-B) interactions) is larger than a critical value C. (B) Workflow to determine the stability of chromatin interactions genome-wide. DNA: black, varying chromatin features or proteins maintaining DNA conformation: grey ovals.).

Whether microphase separation of a block copolymer occurs depends on the interaction strengths between monomers as well as the lengths of the blocks of monomers of each type (Figure 2.1, A). Flory-Huggins polymer theory predicts that spatial segregation will occur when the product of the length of the blocks (N, the number of monomers that make up blocks) and their effective preferential homotypic interaction strength (χ , a parameter that represents the difference in 49 the strength of homotypic interactions as compared to heterotypic (A-B) interactions) is larger than a critical value C, (de Gennes, 1979; Leibler, 1980; Matsen and Schick, 1994). Large blocks of a polymer can spatially segregate even when attractive interactions among monomers are weak, while short blocks will only phase separate when interactions are sufficiently strong. The dependence of microdomain formation on the product of block size and interaction strength suggests an experimental approach to quantify the strengths and dynamics of interactions between individual loci that drive chromosome compartmentalization (Figure 2.1 A, B). One can start with a compartmentalized state of the genome and fragment the chromosomes by in situ restriction digestion, and then identify conditions where chromatin fragments become so short that the chromatin interaction strength between the segments is not sufficient to maintain a phase- or microphase-separated (due to restriction of separation by the polymeric constraint) state. As a result, chromosomal domains and compartments will disassemble over time and the chromosomal fragments of different type (e.g., As and Bs) will become mixed, i.e. chromatin becomes liquidlike. The kinetics of this dissolution and mixing process can then be assessed genome-wide by Hi-C at different times after chromatin fragmentation. Domains formed by strong, stable, and abundant interactions will dissociate more slowly than domains formed by weak, unstable, or infrequent interactions (Figure 2.1, A, B). Here we describe such a strategy that we call liquid chromatin Hi-C.

Chromosome conformation in isolated nuclei

To facilitate enzymatic fragmentation of chromosomes, we isolated nuclei from K562 cells. We performed four analyses to demonstrate that chromosome conformation in isolated K562 nuclei was the same as that in intact cells. First, DAPI staining and imaging showed that nuclei were intact with Lamin A as a ring at the nuclear periphery (Figure 2.2, A). Second, using 3C (Dekker *et al.*, 2002) we readily detected known looping interactions in the beta-globin locus (Dostie et al., 2006; Chien et al., 2011) (Figure 2.3). Third, 5C analysis (Dostie et al., 2006) of a 1 Mb region surrounding the beta-globin locus showed that known CTCFmediated interactions were preserved (Figure 2.3, (Tolhuis et al., 2002; Dostie et al., 2006; Splinter et al., 2006; Kang et al., 2017). Fourth, genome-wide Hi-C analysis (Lieberman-Aiden et al., 2009; Belaghzal, Dekker and Gibcus, 2017) confirmed that chromosome territories, compartments (determined by principle component analysis, with compartments captured by the first principle component (PC1 (Lieberman-Aiden et al., 2009; Belaghzal, Dekker and Gibcus, 2017)), TADs, and CTCF-CTCF loops were intact in isolated nuclei and quantitatively similar to those in intact cells (Figure 2.3, and below).

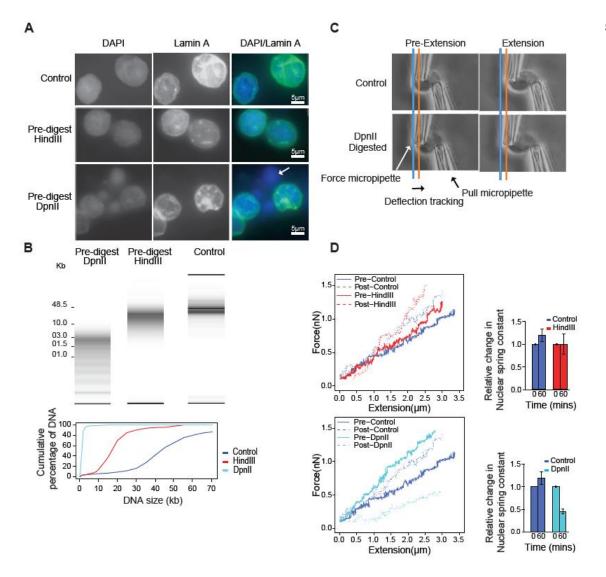


Figure 2.2 | Extensive fragmentation of chromatin leads to liquefied chromatin.

(A) Nuclear and chromatin morphology before and after chromatin fragmentation. Top row: control nuclei in restriction buffer, middle row nuclei digested for 4 hours with HindIII. Bottom row: nuclei digested for 4 hours with DpnII. Nuclei were stained with DAPI (left column), with antibodies against Lamin A (middle column). The right column shows the overlay of the DAPI and Lamin A stained images. HindIII digestion did not lead to major alteration in nuclear morphology and chromatin appearance, while DpnII digestion led to the appearance of DAPI stained droplets (arrow) exiting the nuclei. (B)Top: DNA purified from undigested nuclei, and nuclei pre-digested with DpnII and HindIII was run on a Fragment Analyzer. Bottom: cumulative DNA length distributions calculated from the

Fragment Analyzer data. (**C**) Micromanipulation of single nuclei. Isolated nuclei 52 were attached to two micropipettes at opposite ends. Nuclei were extended by moving the right micropipette (Extension micropipette) and the force required was calculated from the deflection of the calibrated "force" (left) pipette. Blue and orange lines indicate the position of the force pipette before and after extension for control nuclei. After digestion of nuclei with DpnII (bottom) extension required less force as indicated by the much smaller deflection of the force pipette as compared to control nuclei. (**D**) Force-extension plots (left) for control nuclei before and 60 minutes after incubation in restriction buffer (pre- and postcontrol), for nuclei before and after digestion with DpnII, and for nuclei before and after HindIII digestion. Right panel: relative change in nuclear spring constants, calculated from the slopes of the force-extension plots shown on the left. Bars indicate standard error of the mean (n = 5 DpnII pre-digested nuclei, and n = 4 HindIII pre-digested nuclei).

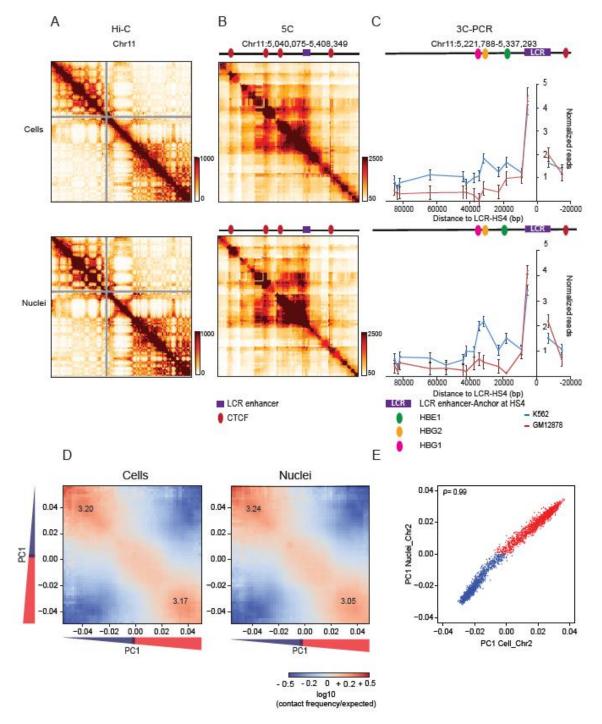


Figure 2.3 | Chromosome conformation in isolated nuclei. (A) Hi-C 2.0 intra-chromosomal interaction maps for K562 cells display

chromosomal compartments and TADs. Top: cells. Bottom: purified nuclei. (B) 5C interaction map of 1 Mb region surrounding the beta-globin locus in K562 cells. Top: cells. Bottom: purified nuclei. CTCF-mediated interactions are preserved in purified nuclei. Red circles: positions of CTCF sites, purple square Beta-globin locus control region (LCR). (C) 3C-PCR for a 44120 kb region surrounding the beta-globin LCR on chromosome 11, detects at high resolution the known looping interactions between the LCR and the expressed gammaglobin genes (HBE1, HBG2) in K562 cells. Looping interactions are not detected in GM12878 cells that do not express these genes. Top: cells. Bottom: purified nuclei. (D) Compartmentalization saddle plots: average intra-chromosomal interaction frequencies between 100 kb bins, normalized by genomic distance. Bins are sorted by their PC1 value derived from Hi-C data obtained with K562 cells. In these plots preferential B-B interactions are in the upper left corner, and preferential A-A interactions are in the lower right corner. Numbers in the corners represent the strength of AA interactions as compared to AB interactions and BB interactions over BA interactions. Left: cells. Right: purified nuclei. (E) Spearman correlation of PC1 in cells vs PC1 in nuclei for chromosome 2 at 100kb resolution $(\rho = 0.99).$

Extensive chromatin fragmentation leads to the formation of liquid

chromatin

We incubated purified nuclei for four hours with restriction enzymes that digest chromatin with different frequencies. Digestion with HindIII resulted in fragments that ranged in size from ~10-25 kb (Figure 2.2, B). A minority of molecules was over 25 kb (<15%), indicating that most of the genome was fragmented to a similar extent. Digestion with DpnII resulted in fragments that ranged in size between ~1 and ~6 kb, with less than 6% of fragments >6 kb (Figure 2.2, B). Microscopic inspection of nuclear morphology by DAPI and Lamin A immunofluorescence staining showed that fragmentation of chromatin with HindIII had only minor effects on nuclear morphology (Figure 2.2, A). In contrast, fragmentation of chromatin with DpnII led to large-scale alteration of

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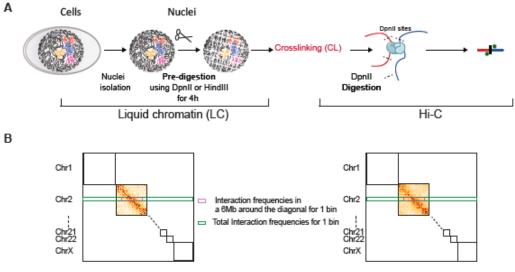
nuclear morphology as detected by DAPI staining, and large buds of apparently 55 liquid chromatin (not surrounded by Lamin A) protruding from the nuclear periphery (Figure 2.2, A, arrow). After spinning down nuclei we detected no DNA in the supernatant, indicating that the liquified chromatin remains largely within the nuclear envelope.

We next tested whether different chromatin fragmentation levels had an effect on nuclear stiffness, which reflects the integrity of chromatin interactions inside the nucleus. Nuclei were isolated from K562 cells, attached to two micropipettes at opposite ends, and the whole nucleus was extended by moving an extension micropipette (Methods). The deflection of a force micropipette provides a measure of the force (Figure 2.2, C). This data provides a force vs. extension plot (Figure 2.2, D, plots on the left), in which the slope of the line fitted to the data is the nuclear spring constant in nN/µm (Figure 2.2, D, bar plots on the right). Extension between 0 - 30% strain measures the chromatin-dominated regime of nuclear force response (Banigan, Stephens and Marko, 2017; Stephens et al., 2017, 2018). Isolated single nuclei were measured for their native spring constant before treatment. We find the stiffness can vary somewhat between individual nuclei. We then measured the stiffness of the same nuclei again 60 minutes post-treatment. Nuclei treated with control conditions (only restriction buffer added to the media) showed a slight stiffening of the nucleus (Figure 2.2, D). Treatment of nuclei with HindIII did not significantly decrease the stiffness as compared to their stiffness pre-treatment. In contrast, DpnII-treated

nuclei displayed a significant decrease (~75%) in stiffness, consistent with previous experiments (Stephens *et al.*, 2017). We conclude that chromosome and nuclear organization can tolerate fragmentation to 10-25 kb segments. In contrast, fragmenting the genome to smaller than 6 kb segments results in extensive loss of chromatin-mediated stiffness.

Compartmental segregation requires chromatin fragments larger than 6 kb

To determine how chromosome folding and compartmentalization is altered as a function of chromatin fragmentation level, we applied Hi-C before (conventional Hi-C) and after chromatin liquefication (liquid chromatin Hi-C). Nuclei were digested with either HindIII or DpnII for 4 hours followed by formaldehyde fixation and Hi-C analysis (Figure 2.4, A). Liquid chromatin Hi-C interaction maps obtained from nuclei that were pre-digested with HindIII were remarkably similar to those obtained with nuclei that were not pre-digested (Figure 2.5, A). The relationship between interaction frequency and genomic distance was largely unaffected (Figure 2.5, B). The ratio of intra- vs. interchromosomal interactions was also highly similar to that in untreated nuclei (Figure 2.5, B). Compartments (Figure 2.5, D) were readily detectable and captured by the first principle component (PC1). Compartment positions were unaffected (Spearman $\rho = 0.99$).



Cis_Control = (Interactions in 6 Mb / total interactions) * 100 Loss of structure (LOS) = (Cis_Control - Cis_Pre-digested) / Cis_Control



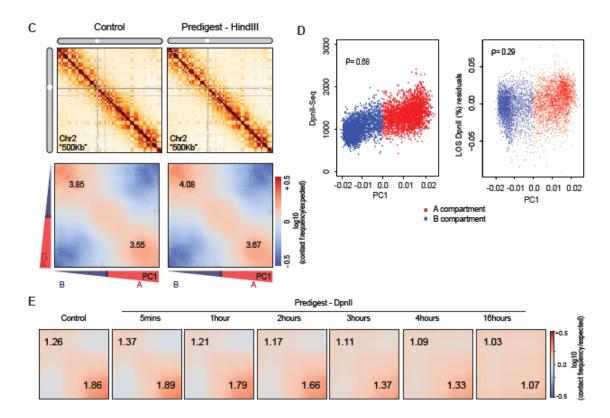


Figure 2.4 | Chromosome conformation dissolution upon chromatin fragmentation.

(A) Workflow for Liquid chromatin Hi-C. (B) Illustration of loss of structure metric using a pre-digested sample and a control. (C) Hi-C interaction maps and

compartmentalization saddle plots for a second replicate of control nuclei (incubated for 4 hours in restriction buffer) and nuclei pre-digested with HindIII for 4 hours. (**D**) Left: Spearman correlation of DpnII restriction digestion efficiency (DpnII-seq) and PC1 for chromosome 2 at 40 kb resolution. Right: Partial correlation of LOS (LOS residuals) with PC1 after controlling for restriction efficiency (DpnII-seq), for chromosome 2 at 40kb resolution. Spearman correlation is indicated. (**E**) compartmentalization saddle plots for the corresponding conditions. Numbers indicate strength of A-A and B-B interactions for inter-chromosomal interactions.

58

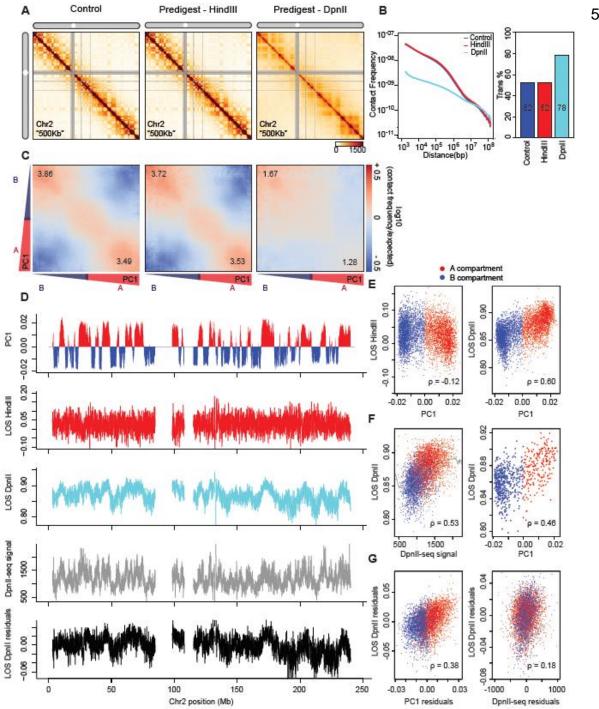


Figure 2.5 | Hi-C analysis reveals chromosome disassembly upon chromatin liquefication.

(A) Hi-C interaction maps of chromosome 2 binned at 500 kb. Left: interaction map for control nuclei in restriction buffer for 4 hours. Middle: nuclei pre-digested for 4 hours with HindIII prior to Hi-C. Right: nuclei digested for 4 hours with DpnII prior to Hi-C (see Figure 2.4, A). (B) Left: genome-wide interaction frequency as function of genomic distance for control nuclei (dark blue), nuclei pre-digested with HindIII (red), and nuclei pre-digested with DpnII (cyan). Right: percentage of inter-chromosomal (trans) interaction frequencies. (C) Compartmentalization saddle plots: average intra-chromosomal interaction frequencies between 40 kb bins, normalized by expected interaction frequency based on genomic distance. Bins are sorted by their PC1 value derived from Hi-C data obtained with control nuclei. In these plots preferential B-B interactions are in the upper left corner, and preferential A-A interactions are in the lower right corner. Numbers in corners represent the strength of AA interactions as compared to AB interaction and BB interactions over BA interactions (Figure 2.10,B). (D) Top plot: Eigenvector 1 values (PC1, 40 kb resolution) across a section of chromosome 2, representing A (red) and B (blue) compartments. Second plot: Loss of pair-wise interactions "LOS" (Methods and Figure 2.4, B) along chromosome 2 at 40 kb resolution for nuclei pre-digested with HindIII. Third plot: LOS for nuclei pre-digested with DpnII. Fourth plot: DpnII-seg signal along chromosome 2 at 40 kb resolution. Bottom plot: LOS-residuals for nuclei pre-digested with DpnII after correction for DpnII signal. (E) Correlation between LOS for nuclei pre-digested with HindIII (left) or DpnII (right) and PC1 (for chromosome 2, Spearman correlation values are indicated). (F) Left: correlation between LOS for nuclei pre-digested with DpnII and DpnII-seg signal (for chromosome 2). Grey line indicates moving average used for residual calculation. Right: correlation between LOS for nuclei pre-digested with DpnII and PC1 for loci cut to the same extent by DpnII (1000-1100 DpnII-seq reads/ 40 kb bin; for chromosome 2). Spearman correlation values are indicated. (G) Left: partial correlation between residuals of LOS for nuclei pre-digested with DpnII and residuals of PC1 after correcting for correlations between LOS and DpnII-seq and PC1 and DpnII-seq signal. Right: partial correlation between residuals of LOS for nuclei pre-digested with DpnII and residuals of DpnII-seq signal after correcting for correlations between LOS and PC1 and DpnII-seq signal and PC1. Spearman correlation values are indicated.

Chromosome compartment strength can be visualized and quantified by

plotting interaction frequencies between pairs of 40 kb loci arranged by their

values along the first eigenvector (PC1) to obtain compartmentalization saddle

plots (Figure 2.5, C). In nuclei pre-digested with HindIII, the strength of preferential A-A and B-B interactions (the ratio of the frequency of A-A and B-B interactions divided by the frequency of A-B interactions) was very similar to untreated nuclei (Figure 2.5, C; see Figure 2.4, C for a replicate).

Much more extensive changes were observed when nuclei were predigested for 4 hours with the frequent cutting enzyme DpnII (Figure 2.5, A) followed by formaldehyde fixation and Hi-C analysis. We observed a considerable redistribution of interactions with loss of short range (<10 Mb) intrachromosomal interactions, and a gain of longer range (>10 Mb) interactions and inter-chromosomal interactions (Figure 2.5, B). The gain in inter-chromosomal interactions appeared to be the result of random mixing of As and Bs from different chromosomes as the preference for interchromosomal A-A and B-B interactions decreased (Figure 2.4, E). Moreover, compartment strength in cis was greatly reduced with a greater relative reduction evident in the A compartment (Figure 2.5, C). These observations show that fragmentation in <6 kb fragments leads to loss of spatial segregation of A and B compartments with more extensive loss of the A compartment.

Quantification of chromosome conformation dissolution

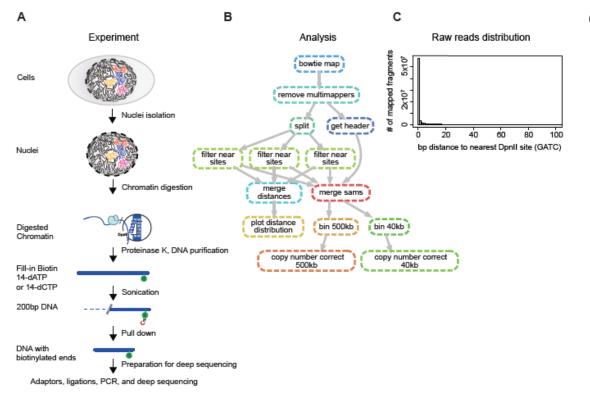
Loss of chromosome conformation and dissolution of chromosomal compartments will result in random mixing of previously spatially separated loci both in cis and in trans. In Hi-C, this effect will be apparent by a redistribution of contacts from short-range interactions towards longer range and interchromosomal interactions. We developed a metric which represents the percentage change in short range intra-chromosomal interactions (up to 6 Mb), and concomitant increase in long-range and interchromosomal interactions after fragmentation relative to control nuclei, which we call "loss of structure" (LOS) (Figure 2.4, B).

We first calculated LOS after 4 hours for chromatin fragmented with HindIII. We observe that in general short-range interactions are only somewhat reduced (less than 5%; Figure 2.5, B). When LOS is plotted along chromosomes (Figure 2.5, D), we observed that LOS was very weakly negatively correlated with PC1 (Figure 2.5, D and E left panel).

We performed the same analysis for nuclei pre-digested with DpnII for 4 hours. We find extensive loss of chromosome conformation, with LOS generally >80%. LOS varies along chromosomes and is strongly positively correlated with PC1 with loci in the A compartment displaying the largest loss (Figure 2.5, D and E). These results show that chromatin fragmentation to <6 kb fragments leads to extensive genome-wide dissolution of chromosome conformation, loss of spatial segregation of A and B compartments, with the A compartment affected the most.

After correcting for differential fragmentation LOS remains highly correlated with compartment status

One explanation for the greater effect of fragmentation on chromatin interactions in the A compartment could be that DpnII cuts more frequently in the A compartment producing smaller fragments. We performed two experiments to assess differential fragmentation. First, we determined the cutting frequency of DpnII in isolated nuclei across the genome by sequencing the ends of the DNA fragments (DpnII-seq; Figure 2.6, see Methods). Second, we directly determined the average fragment size along the genome by purifying DNA of different sizes after pre-digestion with DpnII, sequencing the ends and using the data to calculate for each 40 kb bin the average fragment size (see Methods). The average fragment size for most 40 kb bins ranged from 2.7 to 3.7 kb and was on average slightly smaller for A compartments compared to B compartments (3.1 kb and 3.2 respectively). Cutting frequency and average fragment size are both correlated with PC1 and with LOS (Figure 2.5, D and F left panel, Figure 2.4, D, Figure 2.7).



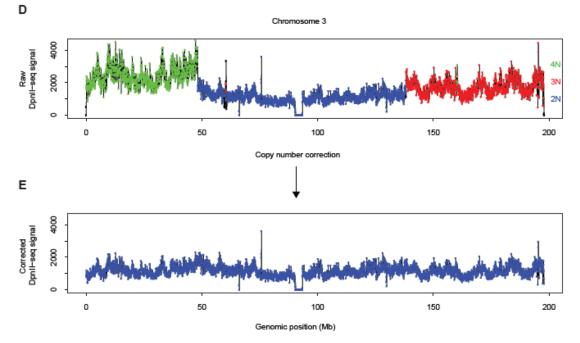


Figure 2.6 | Experimental protocol and computational workflow for DpnIIseq.

(A) Schematic of DpnII-seq experimental protocol for recovering DNA fragments digested by the restriction enzyme DpnII. (B) Directed graph of DpnII-seq computational pipeline (C) Histogram of distance to nearest DpnII recognition site for each recovered DpnII digested fragment. (D) Raw DpnII-seq signal displaying multiple copy number states (2N, 3N, 4N) within chromosome 3 (data binned at 40 kb). (E) Copy number corrected DpnII-seq signal displaying single copy number state (2N) across chromosome 3.

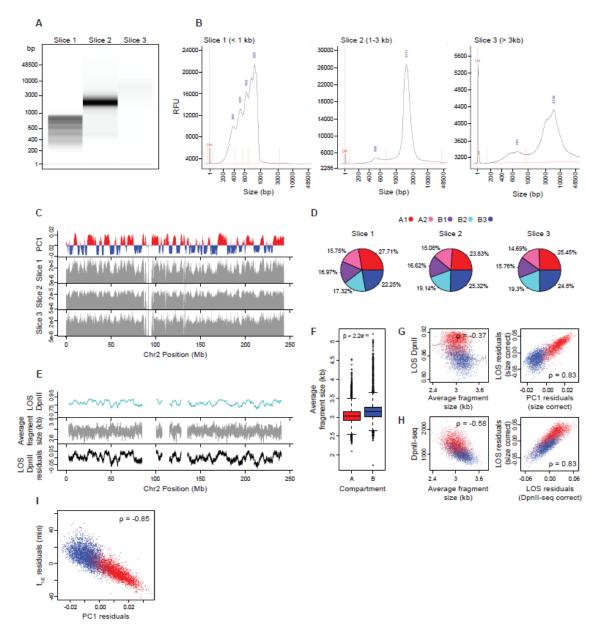


Figure 2.7 | Average fragment size per bin and correlation with chromatin stability.

(A) DNA purified from nuclei pre-digested with DpnII for 4 hours were separated into slices of three sizes and run on a Fragment Analyzer. (B) Fragment Analyzer distributions of DNA fragment sizes for the three separated slices (RFU: relative fluorescence unit, LM: lower marker, fragment sizes at distribution peaks are given in blue). (C) Top plot: Eigenvector 1 values (PC1, 40 kb resolution) across a section of chromosome 2, representing A (red) and B (blue) compartments. Bottom three plots: Normalized coverage of fragments from given slice size across a section of chromosome 2. (D) Percentages of fragments mapped to

each subcompartment for given slice size. (E) Top plot: LOS along chromosome 67 2 at 40 kb resolution for nuclei pre-digested with DpnII. Middle plot: Average fragment size estimated for every 40kb bin after pre-digestion with DpnII (Methods). Bottom plot: LOS-residuals for nuclei pre-digested with DpnII after correction for average fragment size. (F) Boxplot of average fragment size for A compartment and B compartment bins. Significance determined by two-sample two tailed t-test (p < 2.2e-16). (G) Left plot: correlation between LOS for nuclei pre-digested with DpnII and average fragment size. Grey line indicates moving average used for residual calculation. Right plot: partial correlation between residuals of LOS for nuclei pre-digested with DpnII and residuals of PC1 after correcting for correlations between LOS and average fragment size and PC1 and average fragment size (for chromosome 2, Spearman correlation values are indicated). (H) Left plot: Correlation between DpnII-seq signal and average fragment size. Right plot: correlation between residuals of LOS after correcting for average fragment size and residuals of LOS after correcting for DpnII-seq signal (for chromosome 2, Spearman correlation values are indicated). (I) Partial correlation between residuals of $t_{1/2}$ and residuals of PC1 after correcting for correlations between t_{1/2} and average fragment size and PC1 and average fragment size.

Next, we corrected LOS for the differential efficiency of DpnII digestion by calculating the partial correlation between LOS and PC1 after correcting for the correlations of PC1 and LOS with DpnII digestion frequency (see Methods for details). We find that the residuals of PC1 and LOS, are still correlated (Spearman $\rho = 0.38$ for chromosome 2; Figure 2.5, G). Similarly, when we corrected LOS for differences in average fragment size we find that the residuals of LOS remain highly correlated with residuals of PC1 (Spearman $\rho = 0.83$ for chromosome 2, Figure 2.7). To illustrate the correlation between LOS and PC1 independent of fragmentation level directly we selected a set of loci along chromosome 2 that are all cut to the same extent (1000-1100 reads in the DpnII-seq dataset). When we plot LOS vs. PC1 for this set we find a strong correlation (Figure 2.5, F right panel, Spearman $\rho = 0.46$). Finally, we repeated the entire

liquid chromatin Hi-C procedure using a different restriction enzyme, Fatl, which has a different pattern of digestion across the genome as compared to DpnII but produces fragments that are similarly small (Figure 2.8). We calculated LOS and corrected for differential Fatl digestion along the genome using Fatl-seq, exactly as above for DpnII. We again observe a high correlation between residuals of LOS and PC1 (Spearman $\rho = 0.64$ for chromosome 2, Figure 2.8). We conclude that LOS is correlated with compartment status, and that this result is robust for different enzymes and different methods for correcting for digestion efficiency.

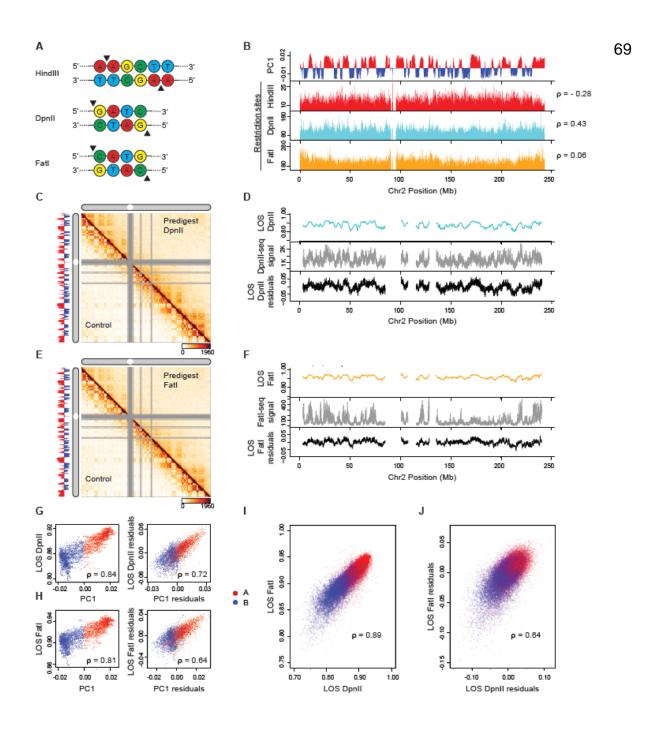


Figure 2.8 | Liquid chromatin Hi-C results are reproducible using the restriction enzyme Fatl.

(A) Restriction sites for the selected restriction enzymes. Black triangles denote cut sites. (B) Top plot: Eigenvector 1 values (PC1, 40 kb resolution) across a section of chromosome 2, representing A (red) and B (blue) compartments. Bottom three plots: Coverage of restriction sites (40kb resolution). Spearman

correlation between restriction site coverage and PC1 is given for each restriction 70 site track. (C) Third replicate of DpnII predigest liquid chromatin Hi-C. Hi-C interaction maps of chromosome 2 binned at 500 kb. Bottom left: control nuclei in restriction buffer for 4 hours. Top right: nuclei digested for 4 hours with DpnII prior to Hi-C. Left track: Eigenvector 1 values (PC1, 40 kb resolution) across a section of chromosome 2, representing A (red) and B (blue) compartments. (D) Top plot: LOS along chromosome 2 at 40 kb resolution for nuclei pre-digested with DpnII. Middle plot: DpnII-seq signal. Bottom plot: LOS-residuals for nuclei pre-digested with DpnII after correction for DpnII-seg signal. (E) Fatl predigest liquid chromatin Hi-C. Hi-C interaction maps of chromosome 2 binned at 500 kb. Bottom left: control nuclei in restriction buffer for 4 hours. Top right: nuclei digested for 4 hours with Fatl prior to Hi-C. Left track: Eigenvector 1 values (PC1, 40 kb resolution) across a section of chromosome 2, representing A (red) and B (blue) compartments. (F) Top plot: LOS along chromosome 2 at 40 kb resolution for nuclei pre-digested with FatI. Middle plot: FatI-seg signal. Bottom plot: LOSresiduals for nuclei pre-digested with Fatl after correction for Fatl-seg signal. (G) Left plot: Correlation between LOS for nuclei pre-digested with DpnII and PC1. Right plot: partial correlation between residuals of LOS for nuclei pre-digested with DpnII and residuals of PC1 after correcting for correlations between LOS and DpnII-seq and PC1 and DpnII-seq signal (for chromosome 2, Spearman correlation values are indicated). (H) Left plot: Correlation between LOS for nuclei pre-digested with Fatl and PC1. Right plot: partial correlation between residuals of LOS for nuclei pre-digested with Fatl and residuals of PC1 after correcting for correlations between LOS and Fatl-seg and PC1 and Fatl-seg signal (for chromosome 2, Spearman correlation values are indicated). (I) Correlation between LOS for nuclei pre-digested with Fatl and LOS for nuclei pre-digested with DpnII (genome wide, Spearman correlation values are indicated). (J) Correlation between residuals of LOS for nuclei pre-digested with Fatl and residuals of LOS for nuclei pre-digested with DpnII after correcting for correlations between Fatl LOS and Fatl-seq and DpnII LOS and DpnII-seq (genome wide, Spearman correlation values are indicated).

Dissociation kinetics of chromatin interactions and compartments

The loss of conformation after DpnII pre-digestion allowed us to measure

the dissociation kinetics of compartments and stability of chromatin interactions.

We first determined the kinetics of chromatin fragmentation (Figure 2.9, A, Figure

2.10, A). We digested nuclei with DpnII for 5 minutes up to 16 hours. After 5

minutes the size range of fragments was between 3 and 15 kb (80% of

fragments; Figure 2.11, A). After one hour 80% of DNA fragments were smaller 71 than 7 kb and after 16 hours 85% of fragments were smaller than 3.5 kb. We sequenced DNA ends to determine the distribution of DpnII cuts across the genome (Figure 2.11, B). At all timepoints the number of DpnII cuts per 40 kb bin was correlated with PC1 (Figure 2.11, B) and the pattern did not change over time (Figure 2.11, B, correlation matrix, Figure 2.9, A).

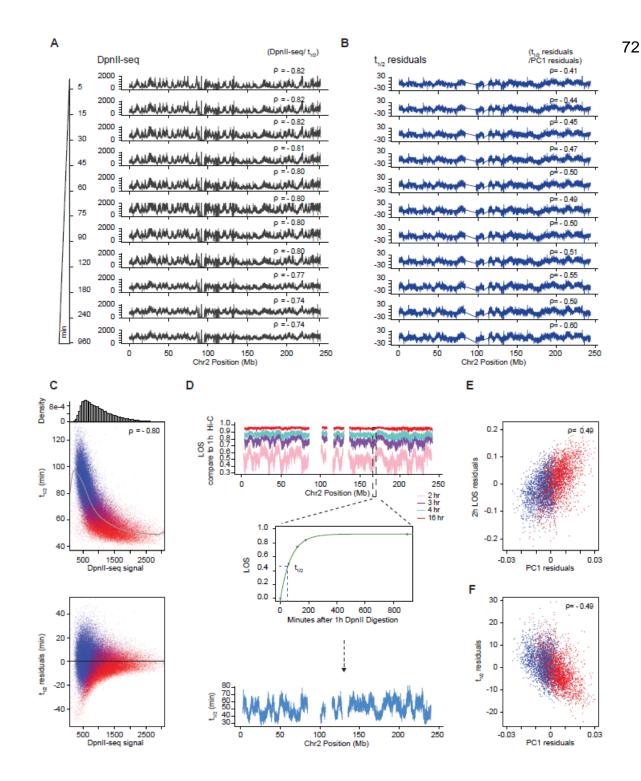
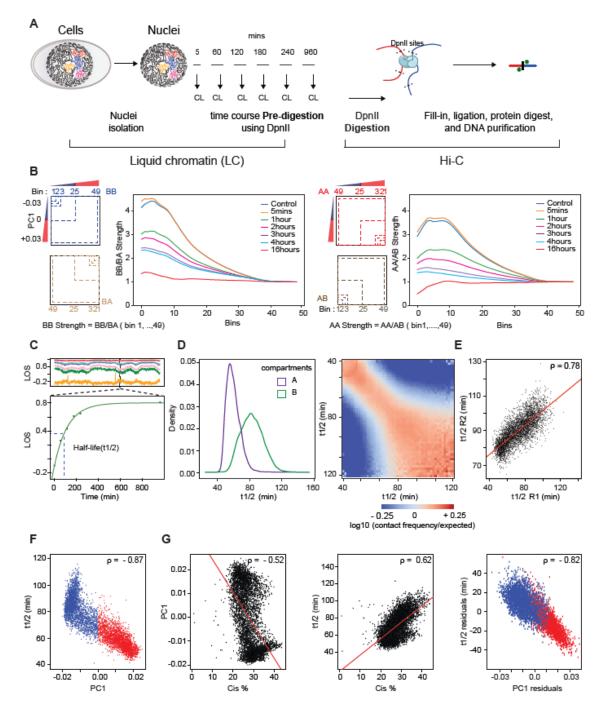


Figure 2.9 | Variations in Half-life and LOS are not explained by DpnII digestion kinetics.

(A) DpnII-seq signals along chromosome 2 after indicated times of digestion.

Spearman correlations between DpnII-seq and $t_{1/2}$ at each timepoint is indicated. 73 (B) $t_{1/2}$ residuals along chromosome 2 after correcting $t_{1/2}$ values by the correlation between t_{1/2} and DpnII-seq signals shown on the left obtained after the indicated times of digestion. Spearman correlation between t1/2 residuals and PC1 residuals are indicated. (C) Top: Genome wide scatterplot of t_{1/2} versus 1 hour DpnII-seq signal. Gray line: moving average. Bar plot above shows the number of loci displaying various levels of DpnII-mediated cuts. Bottom: residuals of t1/2 calculated by subtracting t1/2 from the corresponding average t1/2 (gray line in top plot) plotted vs. number of DpnII cuts. Red dots: loci in the A compartment; Blue dots: loci in the B compartment. The majority of loci have 500-1100 cuts. When comparing loci with similar number of DpnII cut we observe that loci in the A compartment have shorter t_{1/2} values as compared to loci in the B compartment. (D) Top: LOS along chromosome 2 at the indicated timepoints of digestion and calculated by comparison to Hi-C data obtained after 1 hour of digestion. Middle: calculation of t_{1/2} from LOS at different timepoints. Bottom: t_{1/2} along chromosome 2. This t_{1/2} is calculated using the Hi-C data obtained after 1 hour of pre-digestion as starting point. (E) Partial correlation between LOS and PC1 after correcting for their correlations with DpnII-seq. LOS (at 2 hours) is calculated as in panel C using the Hi-C data obtained after 1 hour of predigestion as starting point (F) Partial correlation between t_{1/2} and PC1 after correcting for their correlations with DpnII seq. t_{1/2} is calculated as in panel D using the Hi-C data obtained after 1 hour of pre-digestion as starting point. Spearman correlations are indicated.





(A) Workflow for Liquid chromatin Hi-C timecourse. CL = cross-linking step. (B) Compartment strength derived from compartment saddle plots (See Methods). Left: Diagram depicting compartment strength calculation for B-B interactions.

74

Plot to the right of diagram: B-B interaction strength as a function of bin number 75 for all timepoints of the time course. Right: Diagram depicting compartment strength calculation for A-A interactions. Plot to the right of diagram: A-A interaction strength as a function of bin number for all time points of the time course. (C) Top: LOS signal across a 40 Mb region on chromosome 2 calculated for indicated timepoints in the digestion timecourse. Line colors as in Figure 2.11, E. Bottom: Exponential curve fit to LOS timepoints for a single 40kb bin. t_{1/2} (dashed vertical blue line) representing time elapsed to reach half saturation of LOS signal. (D) Left: Density distributions of t_{1/2} for A and B compartments. Right: t_{1/2} saddle plots: average intra-chromosomal interaction frequencies between 40 kb bins, normalized by genomic distance. Bins are sorted by their t_{1/2} value derived from digestion timecourse. Bins with high t_{1/2} preferentially interact (bottom right of heatmap) and bins with low t1/2 preferentially interact (top left of heatmap). (E) Scatterplot of $t_{1/2}$ vs $t_{1/2}$ for two timecourse replicates (R1 and R2) on chromosome 2. Regression line (red). Spearman correlation is indicated. (F) Scatterplot of PC1 vs t_{1/2} for chromosome 2. A compartment (red); B compartment (blue). (G) Left: Scatterplot of percent interactions occurring in cis within a 6 Mb distance out of total genome wide interactions for each 40 kb bin in control Hi-C map (Cis %) vs PC1. Middle: Cis% vs t1/2. Right: Scatterplot of partial correlation between PC1 and t_{1/2} controlled by Cis %. A compartment (red); B compartment (blue). Solid red lines are regression lines. Spearman correlations are indicated.

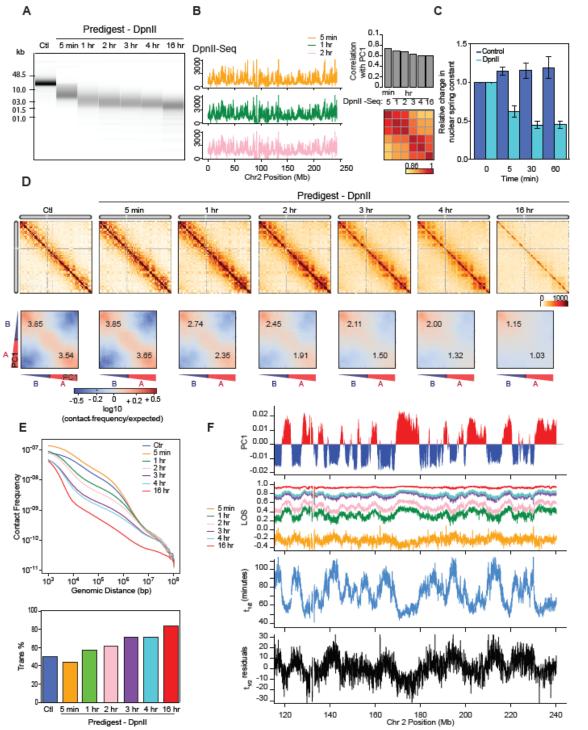


Figure 2.11 | Kinetics of chromatin fragmentation and chromatin dissolution. (A) DNA purified from undigested nuclei, and nuclei pre-digested with DpnII for different time points were run on a Fragment Analyzer. (B) Left:

DpnII-seq signals along chromosome 2 binned at 40 kb resolution after digestion 77 for 5 minutes, 1 hour and 2 hours. Right: correlation between DpnII-seg signals and PC1 and between DpnII-seq signals at different time points. (C) Relative change in nuclear spring constant (nN/µm) after DpnII fragmentation at different time points. Spring constant is significantly decreased after 5 minutes and at background level by 1 hour (p = 0.002, two-tailed t-test). (**D**) Top row: Hi-C interaction maps of chromosome 2 binned at 500 kb. Control: nuclei in restriction buffer for 4 hours. Pre-digest DpnII: nuclei were pre-digested with DpnII for 5 minutes up to 16 hours. (Figure 2.10, A). Bottom row: compartmentalization saddle plots for the corresponding conditions. Numbers indicate strength of A-A and B-B interactions. (E) Top: genome-wide interaction frequency as function of genomic distance for Hi-C data shown in panel (D). Bottom: percentage of interchromosomal (trans) interactions genome-wide for control nuclei and for nuclei pre-digested with DpnII for up to 16 hours. (F) Top: PC1 along a section of 120 Mb of chromosome 2. Second plot: LOS along chromosome 2 at 40 kb resolution for all time points (Figure 2.4, B). Third plot: half-life (t_{1/2}) values derived from the exponential fit of the six time-points for every 40 kb bin (Figure 2.10, C). Bottom plot: residuals of t_{1/2} after correcting for correlations between t_{1/2} and DpnII-seq (Dpnll-seq data for t = 1 hour).

Micromanipulation was again used to measure the nuclear spring constant corresponding to nuclear stiffness. Nuclei displayed a significant loss in stiffness within 5 minutes. Loss of stiffness leveled off at 30 minutes of digestion and showed a 60% decrease in nuclear rigidity, similar to previous experiments in other cell types ((Stephens *et al.*, 2017), Figure 2.11, C). Combined these analyses show that the bulk of DNA fragmentation and chromatin liquefication occurs within the first hour.

Next, we performed liquid chromatin Hi-C where nuclei were pre-digested with DpnII for 5 minutes up to 16 hours (Figure 2.10, A). Interestingly, after 5 minutes of pre-digestion chromosome conformation and compartmentalization are intact, even though chromatin was fragmented to 3-15 kb segments before fixation and nuclear stiffness was significantly reduced (Figure 2.11, C and D). The percentage of intra-chromosomal interactions especially for loci separated by <1 Mb was increased (Figure 2.11, E).

At subsequent time points, when most chromatin fragments are <7 kb long we observe increased loss of intra-chromosomal interactions and concomitant increased inter-chromosomal interactions genome-wide (Figure 2.11, D and E). Compartmentalization, as quantified by the preference of A-A and B-B interactions over A-B interactions, is progressively lost (Figure 2.11, D lower row of heatmaps, Figure 2.10, B). A-A interactions disappear faster than B-B interactions. After 16 hours, only a low level of preferential B-B interaction remains.

Quantification of the half-life of chromosome conformation across the genome

To quantify the kinetics of loss of chromosome conformation and compartmentalization, we calculated LOS genome-wide for each time point (Figure 2.11, F). At t = 5 minutes, LOS is generally negative indicating a gain in chromatin interactions: on average ~25% gain of intra-chromosomal interactions between loci separated by <6 Mb, consistent with the initial increase in overall intra-chromosomal interactions described above (Figure 2.11, E). LOS is inversely correlated with PC1, indicating that loci located within A compartments gain more intra-chromosomal interactions than loci located within B compartments (Spearman $\rho = -0.53$ for chromosome 2, Spearman $\rho = -0.49$

genome-wide). A "block copolymer" model predicts that partial DNA digestion can lead to a strengthening of compartmentalization by removing covalent linkages between A and B blocks, as long as the fragments are still large enough so that attractive forces between them are sufficient for phase segregation (see Methods). At subsequent time points, LOS is increasingly positive as intrachromosomal interactions are progressively lost and inter-chromosomal interactions are gained. LOS is the highest for loci located in the A compartment. At t = 16 hours, LOS is generally as high as 90%, intra-chromosomal interactions are low (<20% of total), and only preferential B-B interactions are still observed in the Hi-C interaction map (Figure 2.11, D). Similar results were obtained with an independent replicate time course experiment (see below).

Next we determined for each 40 kb locus at which time LOS has reached 50% of its maximal value at t = 16 hours. We refer to this time as the half-life, t_{1/2} (minutes), of chromatin interactions at each locus (Figure 2.11, F). To identify t_{1/2} we plotted LOS as a function of time for each 40 kb locus and fit the data to an exponential curve so that t_{1/2} could be determined when LOS equals 50% of its maximum (Figure 2.10, C). Examining t_{1/2} along chromosomes, we observe a strong inverse correlation with PC1 (Spearman ρ = -0.87, Figure 2.10, F): interactions in the A compartment dissolve relatively fast (t_{1/2} = 40-80 minutes) while interactions in the B compartment dissolve slower (t_{1/2} = 60-120 minutes, Figure 2.10, D). We also calculated t_{1/2} genome-wide for the second independent time course experiment and find a strong correlation between t_{1/2} calculated from

the two datasets (Spearman $\rho = 0.78$ for chromosome 2, Spearman $\rho = 0.76$ genome-wide, Figure 2.10, E). The value of t_{1/2} is proportional to a dissociation rate constant and thus independent of the initial level of intra-chromosomal interactions for a given locus. t_{1/2} remains highly correlated with PC1 after correcting for the initial level of intra-chromosomal (<6 Mb) interactions for each bin (Spearman $\rho = -0.82$, Figure 2.10, F and G).

Similar to LOS, t_{1/2} is correlated with DpnII digestion frequency at all timepoints (Figure 2.9, A). We calculated the partial correlation between t_{1/2} and PC1 after correcting for correlations between PC1 and t_{1/2} with DpnII cutting frequency. We find that t_{1/2} and PC1 remain strongly correlated (Figure 2.11, F), regardless of which DpnII fragmentation dataset (genome wide Spearman ρ ranging from -0.41 to -0.60, t = 5 min up to t = 16 hours) was used for the calculation of the partial correlation (Figure 2.9, A and B). Although loci in the A compartment are often cut more frequently than loci in the B compartment, when comparing loci cut with similar frequency, loci in the A compartments had shorter half-lives (Figure 2.9, C). Similar results were obtained when t_{1/2} was corrected for average fragment size for each bin (Figure 2.7, I, Spearman ρ = -0.85 for chromosome 2, Spearman ρ = -0.76 genome-wide).

We considered whether we could have overestimated the $t_{1/2}$ for loci in the B compartment because fragmentation of these loci could be slower than for loci in the A compartment. We reasoned that because after 1 hour incubation with DpnII digestion is largely complete, calculation of LOS using the Hi-C data at t =

1 hour as starting condition would provide an estimate of dissolution kinetics starting at a timepoint when A and B compartments are both extensively fragmented. We find that LOS, and t_{1/2} calculated this way are still strongly correlated with PC1, and this correlation remains strong after correcting for fragmentation level (Figure 2.9 D to F).

Dissociation kinetics of chromatin interactions at different sub-nuclear structures

The A1 sub-compartment is most enriched in active histone modifications and found near nuclear speckles (Chen *et al.*, 2018). B2 and B3 are located near the nuclear lamina (B2 and B3) and the nucleolus (B2) (Rao *et al.*, 2014; Chen *et al.*, 2018; Quinodoz *et al.*, 2018). B1 is enriched in the repressive H3K27me3 mark, which is often associated with polycomb binding. To relate subcompartment status to chromatin dissociation rates, we compared the residuals of t_{1/2} (after correcting for fragmentation level using DpnII-seq) for loci located in the 5 sub-compartments defined for K562 cells ((Xiong and Ma, 2018), Figure 2.12, A). We find that residual t_{1/2} varies greatly between sub-compartments: t_{1/2}(A1) ~ t_{1/2}(B1) < t_{1/2}(A2) < t_{1/2}(B2) ~ t_{1/2}(B3).

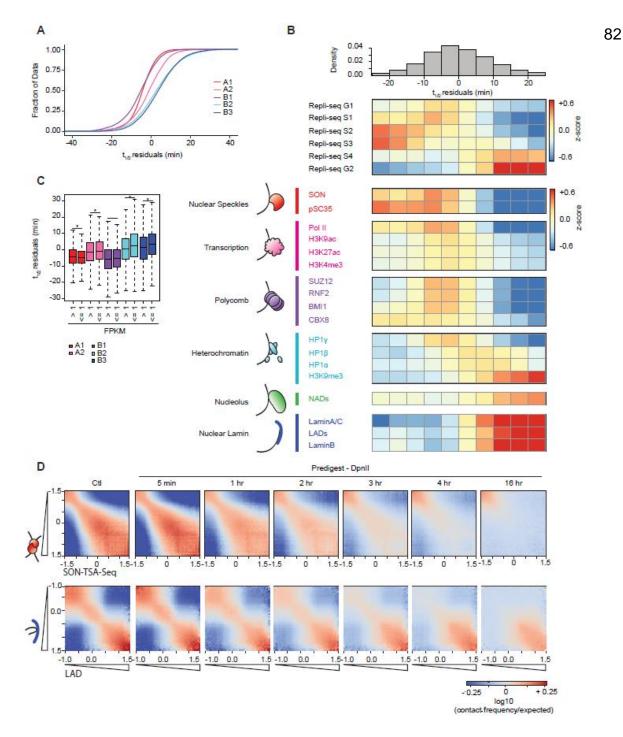


Figure 2.12 | Dissociation kinetics of chromatin interactions at different sub-nuclear structures.

(A) Cumulative distributions of residuals of $t_{1/2}$ (in minutes) for each of the five annotated sub-compartments. (B) Top: the genome was split into 10 bins, where each bin corresponds to sets of loci that share the same $t_{1/2}$ residual interval.

Middle: For each $t_{1/2}$ residual interval a heatmap of mean z-score signal of Repli-Seq data in different phases of the cell cycle G1, S1-4, G2. Bottom: For each $t_{1/2}$ residual interval a heatmap of mean z-score signal enrichment was quantified for various markers of sub-nuclear structures (See Methods). (**C**) Boxplot of $t_{1/2}$ residuals for bins with expressed genes (mean FPKM > 1) and bins with low or no expression (mean FPKM <=1) stratified by sub-compartment. Significance determined by two-sample two tailed t-test *(p < 0.003). (**D**) Homotypic interaction saddle plots for loci ranked by their association with speckles (as detected by SON-TSA-seq, top 3) and by their association with the nuclear lamina. Preferential pair-wise interactions between loci associated with the lamina can still be observed after several hours, whereas preferential pair-wise interactions between loci associated with speckles are lost more quickly.

It is noteworthy that interaction dissociation rates for loci in the polycomb related B1 sub-compartment are as high or higher (residuals of t_{1/2} as or more negative) than those for loci in the active and open A1 sub-compartment. Interactions between Lamin-associated loci in the B3 sub-compartments dissociate the slowest while interactions between loci in the B2 sub-compartment dissociate somewhat faster. These observations indicate that loci associated with different sub-nuclear structures display a range of interaction stabilities.

We coarse-grained $t_{1/2}$ residuals by splitting them into 10 intervals and then assigned these intervals genome-wide. We then explored the enrichment for varying chromatin features for each $t_{1/2}$ residual interval (Figure 2.12, B). Chromatin interactions for early replicating domains had short half-lives, while interactions for loci in later replicating domains were more stable (Figure 2.12, B). Interestingly, loci with the shortest $t_{1/2}$ residuals replicate in the middle of Sphase. We find that loci near the speckle-associated proteins pSC35 or SON are engaged in the most unstable interactions. Similarly, transcriptionally active loci, identified by ChIP-seq for a range of histone modifications and factors associated with open chromatin such as H3K4me3 and RNA PoIII, were also involved in relatively unstable chromatin interactions.

Interactions for loci bound by polycomb complexes (a subset of which are in the B1 sub-compartment) were as unstable as active speckle associated loci (Figure 2.12, B, Figure 2.13, B). Half-lives differed for loci bound by different polycomb subunits. Loci with the shortest t_{1/2} residual values are enriched for binding the CBX8 subunit. An example of a large polycomb-bound domain in K562 cells is the HoxD cluster. The cluster is ~100 kb and covered by polycomb subunits Suz12, RNF2, CBX8 and BMI1 and the histone modification H3K27me3 (Figure 2.13, C). The half-life of chromatin interactions for loci in the HoxD cluster is relatively short.

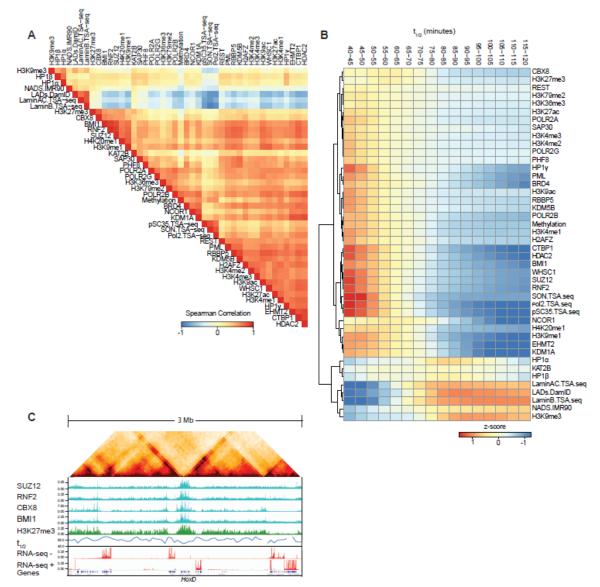


Figure 2.13 | Associations between sub-nuclear structures and chromatin interaction stability.

(A) Spearman correlation matrix between signals for various chromatin state markers of various sub-nuclear structures, chromatin remodellers and histone modifications with row order determined by hierarchical clustering. (B) The genome was split into 16 bins, where each bin corresponds to sets of loci that share the same $t_{1/2}$ interval. For each $t_{1/2}$ interval the mean z-score signal enrichment for various markers of sub-nuclear structures, chromatin remodellers and histone modifications was calculated and shown as a heatmap. Row order determined by hierarchical clustering. (C) 3 Mb region surrounding HoxD locus. Top: Hi-C contact map for K562 control nuclei showing the position of the HoxD

locus. Tracks: ChIP-seq tracks for polycomb subunits (cyan) and the polycomb associated histone modification H3K27me3 (green). t_{1/2} (blue). Minus strand and plus-strand signal of total RNA-seq (red). Refseq Genes (blue/black). The polycomb-bound domain displays shorter half-life compared to expressed genes in flanking regions.

Silent and closed chromatin loci around the nucleolus or at the nuclear lamina were engaged in the most stable interactions (Figure 2.12, B). Chromatin interactions for loci associated with HP1y (CBX3) were relatively unstable while interactions for loci associated with HP1 β (CBX1) or HP1 α (CBX5) were more stable. This variation is in agreement with the chromosomal locations and dynamics of these three HP1 proteins. HP1y is associated with active chromatin and mobile, while HP1 α and HP1 β are typically found in constitutive heterochromatin near (peri) centromeres and are much less mobile (Dialynas et al., 2007).

For each sub-compartment, we split loci into expressed (FPKM>=1) or not expressed (FPKM<1) categories (Figure 2.12, C). We find that sub-compartment status is the major determinant of chromatin interaction stability, irrespective of transcriptional status. However, transcriptional status modulates t_{1/2} to some extent: in general, loci located in B2 and B3 sub-compartments are engaged in relatively stable chromatin interactions, but interactions that involve loci that are expressed have shorter half-lives. The expression status of loci located in the A1, A2 sub-compartments had only very minor effect on the t_{1/2}.

The differential stability of pair-wise chromatin interactions at different subnuclear structures can be quantified by plotting interaction frequencies between pairs of 40 kb loci arranged by their level of factor binding to obtain homotypic interaction saddle plots (Figure 2.12, D). In these plots, pair-wise interactions between loci enriched in factor binding are shown in the lower right corner, and pair-wise interactions between loci not bound by the factor are shown in the upper left corner. After chromatin fragmentation we observe loss of preferential interactions between speckle associated loci, while preferential interactions between non-speckle associated loci can be observed even after 16 hours. Conversely, preferential interactions between lamin-associated loci remain detectable even at late time points, while interactions between loci not at the lamina disappear relatively fast.

Chromatin loops dissociate upon chromatin fragmentation

Enriched point-to-point looping interactions are detected as "dots" in Hi-C interaction maps. The majority of these represent cohesin-mediated interactions between pairs of convergent CTCF sites (Rao *et al.*, 2014). We aggregated Hi-C data at pairs of sites that had previously been shown to engage in looping interactions in K562 cells (Rao *et al.*, 2014). We readily detected these loops in intact purified nuclei (Figure 2.14, A). After fragmentation with HindIII for 4 hours, loops appeared to become slightly stronger. Fragmenting chromatin with DpnII resulted in loss of loops over time.

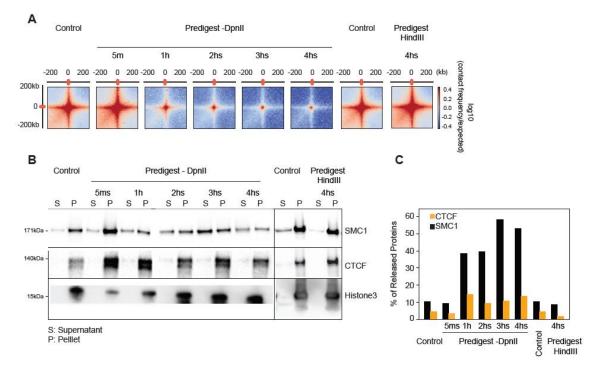


Figure 2.14 | Chromatin loop dissociation upon fragmentation.

(A) Aggregated distance-normalized Hi-C interactions around 6,057 loops detected in K562 cells by HiCCUPS (Rao *et al.*, 2014) at 10 kb resolution, for control nuclei and nuclei digested with DpnII up to 4 hours, and for nuclei digested with HindIII for 4 hours. (B) Western blot analysis of CTCF, cohesin and Histone H3 abundance in soluble and chromatin-bound fractions obtained from control nuclei and from nuclei pre-digested with DpnII up to 4 hours and HindIII for 4 hours. (C) Quantification of the data shown in panel B. Percentage of released protein is the ratio of protein level in the soluble fraction divided by the sum of the levels in the soluble and chromatin-bound fractions.

We assessed whether CTCF and cohesin binding to chromatin is affected

by chromatin fragmentation. We fractionated proteins in chromatin-bound and

soluble fractions ((Liang and Stillman, 1997), Methods). In intact nuclei, most of

the CTCF and cohesin is associated with chromatin (Figure 2.14, B and C).

Digesting chromatin with HindIII did not lead to dissociation of CTCF or cohesin.

However, fragmenting chromatin with DpnII led to dissociation of cohesin after 1 89 hour, while CTCF binding was only weakly affected. We conclude that DNA fragmentation to <6 kb fragments, but not to 10-25 kb fragments, leads to loss of cohesin binding and loss of looping interactions. These results are consistent with earlier observations that showed that in yeast stable chromatin binding by cohesin requires intact DNA (Ciosk *et al.*, 2000). These data can be interpreted in the context of the model where cohesin rings encircle DNA (pseudo-) topologically (Srinivasan *et al.*, 2018). Possibly, when DNA is fragmented, the cohesin ring can slide off nearby free ends.

Discussion

Using liquid chromatin Hi-C we obtained a view of the dynamics of chromatin interactions throughout the nucleus and the genome (Figure 2.15, A). Previously, live cell imaging experiments found differences in mobility dependent on sub-nuclear position and chromatin state and activity (Marshall *et al.*, 1997; Hediger *et al.*, 2002; Thakar, Gordon and Csink, 2006; Bronshtein *et al.*, 2009, 2015; Therizolsa *et al.*, 2010; Shinkai *et al.*, 2016; Nagashima *et al.*, 2019). In such experiments the movement detected is strongly constrained by the fact that loci are part of very long chromosomes. A previous study, which inspired the current work, aimed to identify factors that determine intrinsic locus-locus interactions and locus mobility by removing the polymeric constraint due to linkage (Gartenberg *et al.*, 2004). In that work a silent locus was excised from the chromosome (Gartenberg *et al.*, 2004) and its mobility and preference for

association with other silent loci and the nuclear periphery was found to depend 90 on specific silencing complexes. In our liquid chromatin Hi-C experiments, the polymeric constraint on movement is removed for all loci simultaneously, in effect performing a genome-wide variant of the experiments performed by Gartenberg et al.

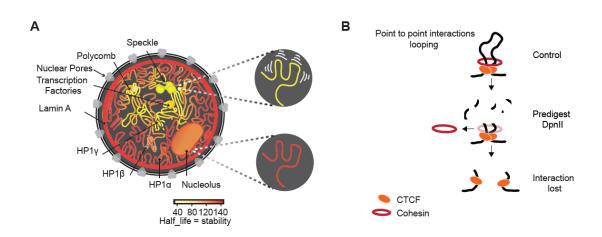


Figure 2.15 | Illustration of chromatin interaction dynamics in the nucleus and model for cohesin loss after chromatin digestion.

(A) Left: Schematic representation of varying chromatin interactions dynamics at different sub-nuclear domains. Shortest half-life reflects the least stable interactions (yellow), while longest half-life reflects the most stable interactions (dark orange). Nuclear subdomains differ greatly in their stability. Top right: Chromatin anchored at speckles is driven by the most dynamic interactions. Bottom right: Chromatin anchored at the nuclear lamina involves the most stable interactions. (B) Model for how cohesin rings stabilize CTCF-CTCF loops by encircling loop bases. Top: Cohesin ring encircles loop bases at convergent CTCF sites. Middle: Pre-digestion with DpnII cuts loop into chromatin fragments <6 kb, and the cohesin ring can slide off nearby ends. Bottom: CTCF remains bound to digested chromatin fragments but interactions between CTCF-bound sites are lost</p>

Chromosomal compartmentalization tolerates genome-wide fragmentation

with HindIII in >10-25 kb fragments. Micro-mechanical measurements also show

that chromosomes remain mechanically fully connected. We conclude that stable 91 chromosome conformation and phase segregation can occur when blocks of a particular chromatin state are at least 10 kb. Our results obtained with DpnII digestion where the genome is fragmented in <6 kb (average \sim 3.1 kb) fragments show that these fragments are too short to maintain phase-segregated domains. The stability of interactions between <6 kb fragments depends on their chromatin state and association with sub-nuclear structures: interactions at the nuclear lamina are relatively stable, those near nuclear speckles and polycomb complexes are highly unstable, while interactions for loci associated with different heterochromatin proteins and the nucleolus displayed a range of intermediate stabilities. The dynamics of associations between loci are therefore determined by chromatin-associated factors, and may also be determined directly by the biochemical properties of histone tail modifications. For instance, the Rosen lab found that chromatin fragments can form droplets in vitro and that the dynamics of chromatin fragments within these droplets are dependent upon both H1 binding and histone acetylation (Gibson et al., 2019).

Loci associated with the three different HP1 proteins display different dissociation kinetics. Interestingly, these differences correlate with different dynamics and sub-nuclear locations of the HP1 proteins in the nucleus. HP1 γ (CBX3) binds relatively transiently to euchromatin, which may explain the dynamic nature of chromatin interactions between loci bound by this protein (Dialynas *et al.*, 2007). On the other hand, interactions between loci enriched in

HP1 α (CBX5) are more stable, which likely is the result of more stable HP1 α binding to heterochromatin (Strom *et al.*, 2017). Our finding that lamin-associated and HP1 α -associated loci are engaged in the most stable interactions is consistent with recent modeling of inverted nuclei where heterochromatin is located in the center of the nucleus. Such inverted arrangement can only occur when heterochromatic interactions are much more stable or abundant than interactions between active chromatin loci (Falk *et al.*, 2019).

Liquid chromatin Hi-C showed differences in chromatin interaction stability between facultative heterochromatic domains marked by polycomb and constitutive heterochromatic domains marked by lamina association or binding of HP1 α /HP1 β proteins. While many chromatin contacts in constitutive heterochromatin were maintained even after 16 hours of digestion, the half-life for chromatin contacts at polycomb-bound regions was short, on a scale similar to active regions of the genome. The compacted states of polycomb and HP1 α bound chromatin appear to form via a similar phase-separation mechanism mediated by multivalent interactions between specific CBX homologs. In vitro and *in vivo*, both CBX2 (polycomb subunit) and CBX5 (HP1 α) are capable of forming condensates of polycomb bodies and constitutive heterochromatin, respectively (Larson et al., 2017; Plys et al., 2019; Tatavosian et al., 2019). Our data indicate that these different condensates and associated chromatin have very different properties: the stability of interactions between loci mediated by these factors is distinct, possibly related to differences in affinity between CBX

proteins and chromatin: the binding affinity of CBX5 (HP1 α) for its preferential histone modification H3K9me3 is higher than the affinity of CBX2 for its preferential mark H3K27me3 (Kaustov *et al.*, 2011). This differential could have consequences for the ability of cells to regulate genes embedded within these different types of heterochromatin.

Our results allow a crude estimate of the Flory-Huggins χ parameter for A/B segregation of chromatin in the nucleus. Given that HindIII and DpnII cut chromatin into segments of approximately 17 ± 7 kb and 3 ± 1 kb respectively (with Fatl also generating approximately 3 kb average sized fragments), a reasonable estimate of the minimum length of fragments necessary to drive A/B segregation is $N^* = 10 \pm 4$ kb. Given that these fragments are small compared to the A/B compartmentalization scale of a few Mb, the fragments will be essentially A- or Btype (euchromatin or heterochromatin) homopolymers. The similar ~1.2 Mb average size of the A and B compartments, as well as the remarkably similar ~ 3 kb fragment sizes observed for DpnII digestion of A and B regions (Figure 2.7, F) indicate that the simple Flory-Huggins model can be applied to a concentrated solution of symmetrical (equal length and concentration) A and B homopolymers. For homopolymers, the Flory-Huggins model predicts a critical length needed for phase separation of N*=2/ χ (de Gennes, 1979), indicating $\chi = 0.20 \pm 0.07$ /kb ($\chi =$ 0.036±0.013 /nucleosome). Given that χ is in k_BT units (1 k_BT = 0.6 kcal/mol at physiological temperatures), its small value indicates that the effective demixing interaction between nucleosomes is weak, consistent with a liquid-like phaseseparation picture for A and B compartments, where regulation of chromatin organization and compartmentalization is possible by relatively small changes in nucleosome interactions (e.g., via histone modifications). While extremely crude (e.g., we have used the χ estimate for a dense polymer melt rather than for a concentrated solution) our data clearly indicate a weak value for χ , and that the interactions that are driving compartmentalization are a small fraction of a kcal/(mol·nucleosome). Our result is within a factor of 2 of a recent theoretical estimate (critical size for demixing of 20 kb, (MacPherson, Beltran and Spakowitz, 2018)). We note that we have been able to rather use a simple application of Flory-Huggins theory by virtue of the similar A/B fragment and compartment sizes. We emphasize that these properties do need to be examined before similar estimates can be done for other cell types or species.

We also note that our estimated χ assumes that the fragments are able to equilibrate their positions in both the DpnII and HindIII cases. It is conceivable that entanglements, cross-bridging or other effects may strongly retard or preclude demixing in the HindIII case. Such effects would be a confounding factor in estimation of χ , but the DpnII data do solidly constrain the critical size for demixing to be larger than 3 kb (i.e., χ < 0.66/kb). Further experiments on the kinetics of fragment demixing would be very interesting in this regard, as they would shed further light on the physical processes underlying A/B compartment formation in vivo. It is important to point out that during the liquid chromatin Hi-C procedure 95 some chromatin factors and RNAs may dissociate from the purified nuclei, and this could affect the locus mixing behavior we observe. The current work analyzed the intrinsic chromatin interaction strengths and dissolution kinetics of chromosome conformation within otherwise inactive nuclei. Future work should focus on how these kinetic properties change in cells or nuclei, where active processes such as transcription, replication, chromatin compaction and condensation, and loop extrusion are also acting, and on determining the roles of RNAs, protein complexes, and histone modifications in modulating the attractive forces between loci and the dynamics of genome folding in general.

Tables

		ENCODE Accession		
Chromatin Feature	· ·	ID	Output type	PubMed ID
H3K36me3	ChIP-seq	ENCFF223BKS	fold change over control	
H3K27ac	ChIP-seq	ENCFF006RIO	fold change over control	
H3K4me1	ChIP-seq	ENCFF463AQS	fold change over control	
H3K4me2	ChIP-seq	ENCFF778DNU	fold change over control	
H3K4me3	ChIP-seq	ENCFF330TJF	fold change over control	
H4K20me1	ChIP-seq	ENCFF242XLB	fold change over control	
H2AFZ	ChIP-seq	ENCFF430FSQ	fold change over control	
H3K9me1	ChIP-seq	ENCFF526UWC	fold change over control	
H3K9me3	ChIP-seq	ENCFF700FQH	fold change over control	
H3K9ac	ChIP-seq	ENCFF527JUP	fold change over control	
H3K27me3	ChIP-seq	ENCFF936BVT	fold change over control	
H3K79me2	ChIP-seq	ENCFF619JRY	fold change over control	
HP1β	ChIP-seq	ENCFF985MBQ	fold change over control	
HP1γ	ChIP-seq	ENCFF630YDI	fold change over control	
HP1α	ChIP-seq	ENCFF014XHT	fold change over control	
EHMT2	ChIP-seq	ENCFF982RMW	fold change over control	
CBX8	ChIP-seq	ENCFF206WVX	fold change over control	
RNF2	ChIP-seq	ENCFF071CIY	fold change over control	
BMI1	ChIP-seq	ENCFF514QBY	fold change over control	
SUZ12	ChIP-seq	ENCFF363DWX	fold change over control	
RBBP5	ChIP-seq	ENCFF058KTS	fold change over control	
CTBP1	ChIP-seq	ENCFF112LWM	fold change over control	
KAT2B	ChIP-seq	ENCFF164NLF	fold change over control	
BRD4	ChIP-seq	ENCFF260JHC	fold change over control	
NCOR1	ChIP-seq	ENCFF292YLV	fold change over control	
KDM5B	ChIP-seq	ENCFF293III	fold change over control	
HDAC2	ChIP-seq	ENCFF915GWT	fold change over control	
SAP30	ChIP-seq	ENCFF816KCQ	fold change over control	
WHSC1	ChIP-seq	ENCFF864WOR	fold change over control	
PHF8	ChIP-seq	ENCFF427QTV	fold change over control	
REST	ChIP-seq	ENCFF518QUW	fold change over control	
KDM1A	ChIP-seq	ENCFF734YKJ	fold change over control	
POLR2A	ChIP-seq	ENCFF749YKR	fold change over control	
POLR2B	ChIP-seq	ENCFF452WGO	fold change over control	
POLR2G	ChIP-seq	ENCFF015NSS	fold change over control	
PML	ChIP-seq	ENCFF157IUB	fold change over control	
Methylation	WGBS	ENCFF867JRG	methylation state at CpG	
NADS.IMR90	aCGH		NAD state	28575119
SON	TSA-seq		log2(pull-down / input)	30154186
pSC35	TSA-seq		log2(pull-down / input)	30154186
LaminA/C	TSA-seq		log2(pull-down / input)	30154186

LaminB	TSA-seq		log2(pull-down / input)	30154186	97
Pol2	TSA-seq		log2(pull-down / input)	30154186	
LADs	DamID		log2(Dam-LaminB1/Dam)	30154186	
G1	Repli-seq	ENCFF001GRX	percentage normalized signal		
S1	Repli-seq	ENCFF001GSF	percentage normalized signal		
S2	Repli-seq	ENCFF001GSJ	percentage normalized signal		
S3	Repli-seq	ENCFF001GSN	percentage normalized signal		
S4	Repli-seq	ENCFF001GSP	percentage normalized signal		
G2	Repli-seq	ENCFF001GSB	percentage normalized signal		

Table 2.1 | Public datasets used to associate liquid chromatin Hi-Cmeasured stability with various chromatin features

Data used to associate $t_{1/2}$ with various chromatin histone modifications, transcription factors, DNA-binding proteins, sub-nuclear structures, and replication timing (Figure 2.12, B and D, Figure 2.13, A and B).

Materials and methods

Digestion, cross-linking and copolymer architecture and

hetero/euchromatin phase separation

Chromatin in the G1 nucleus can be considered as a set of blocks of euchromatin and heterochromatin (the A and B compartments consisting of regions of predominantly euchromatin vs heterchromatin, respectively), which are constrained to be near each other by being part of the same linear chromosomes, i.e., effectively being long many-block copolymers. We suppose that the A and B heterochromatin/euchromatin monomers have a weak tendency to repel one another (or equivalently that A-A or B-B attract one another, for example via protein-mediated nucleosome-nucleosome interactions acting preferentially on euchromatin or heterochromatin, or even via physio-chemical effects such as relative hydrophobicity of more methylated nucleosomes).

If we suppose the A and B blocks to be on average N monomers long (roughly nucleosomes for the sake of this discussion), then under melt-like conditions the standard Flory theory of polymer phase separation predicts that if we were to cut the polymers into pure A and B blocks at the block boundaries (i.e., at a spacing of N monomers commensurate with the block sizes), they would phase separate for a segment-segment interaction strength stronger than $\chi^* = 2/N$ (de Gennes, 1979). Note that this level of interaction (given approximately in k_BT units) is proportional to 1/N where N is crudely in nucleosome units; for 200 kilobase blocks, we have approximately N=1000, indicating that small fractions of a k_BT in effective A/B repulsion or A-A or B-B attraction is sufficient to drive strong euchromatin/heterochromatin phase separation (Marko and Siggia, 1997).

Now if we were to instead cut less frequently than this, say at every second block boundary, so as to arrive at a system of AB linear diblock copolymers each of length 2N (N monomers of A followed by N monomers of B), the constraint that the A and B blocks be connected suppresses phase separation, increasing the critical interaction (all other factors held constant) to χ^* = 5.3/N (Leibler, 1980). In this case bulk phase separation cannot occur, but instead local, or "microphase separation" occurs, with formation of micelle-like or layered phase-separated structures. Nevertheless, for $\chi >> \chi^*$, strong segregation of the A and B monomers can still occur.

If we were to not cut at all, but rather to suppose that the chromosomes are very long multiblock copolymers, with many blocks each of N monomers alternating between A and B ("ABABABAB... multiblock copolymers"), the critical interaction strength will rise with increasing number of blocks, approaching the limit $\chi^* = 7.5/N$ for many blocks (Matsen and Schick, 1994). Therefore, starting from this limit, the tendency for chromosome domains to phase separate will be enhanced by cutting the chromosomes up into successively smaller pieces: as chromatin cutting increases from no cutting, we expect to see intensification of A/B compartment contrast in the Hi-C map.

Now, if we cut too frequently, when the cuts become spaced smaller than 100 the block size (cut spacing M < N monomers), we will have the situation that the critical interaction strength will become $\chi^* = 2/M > 2/N$, i.e., the cuts are frequent enough to suppress phase separation by decreasing the amount of interaction enthalpy per polymer "molecule". Therefore we expect that overly frequent cutting will cause a reduction in A/B compartment Hi-C map contrast, i.e., for some intermediate level of cutting similar to the sizes of the A and B blocks, one will see a maximum level of A/B compartment contrast.

There is also likely an effect of "crosslinking" ("chromatin cross-bridging"), which provides an additional level of constraint suppressing phase separation, above the linear-multiblock architecture of chromosomes. For example, taking linear diblock copolymers (N A monomers followed by N B monomers) and circularizating them raises the critical interaction for microphase separation from 5.3/N to 8.9/N, nearly a factor of 2 (Marko, 1993).

Similarly, if we start with A and B homopolymers each of length N, constraining them to have their ends at a flat surface, thus forcing them to mix at the surface, increases the critical interaction for phase separation from 2/N up to 4.5/N (Marko and Witten, 1991), with microphase separation again occurring in the constrained case. Releasing chromatin crosslinking/cross-bridging constraints (which also will occur for chromatin cutting) will in general also reduce the interaction strength needed to drive phase separation, increasing A/B compartment contrast in Hi-C maps.

In conclusion, basic polymer phase separation theory predicts that gradually increasing the cleavage of chromatin will gradually increase the intensity of A/B compartment contrast in Hi-C maps until the cuts are spaced by approximately one A or B block; further cutting will reduce the intensity of phase separation and A/B compartment contrast. Notably, the nature of the segregation can be expected to be "microphase segregation" rather than bulk phase separation, until the number of cuts is sufficient to liberate A or B "homopolymer" segments.

K562 nuclei purification

Three sucrose cushions were made before starting nuclei purification. 30 mL of 30% sucrose [10 mM PIPES pH 7.4, 10 mM KCl, 2 mM MgCl2, pH adjusted to 7.4 using 1 N KOH, 30% sucrose, 1 mM DTT (added prior to use), 1:100 protease inhibitor (Thermo Fisher 78438) (added prior to use)] was transferred to a 50 mL tube, then 5 mL of 10% sucrose [10 mM PIPES pH 7.4, 10 mM KCl, 2 mM MgCl2, 10% Sucrose, 1 mM DTT (added prior to use), 1:100 protease inhibitor (added prior to use)] was slowly loaded in top of 30% sucrose, and the tubes were incubated at 4°C until needed. K562 cell pellets (100 million cells) were lysed using the following nuclear isolation procedure. After the cells were spun, the pellets were washed twice with 10 mL HBSS, then pelleted after each wash at 300 rpm for 10 min at 4°C. Cell pellets were dissolved in 15 mL nuclear isolation buffer [10 mM PIPES PH 7.4, 10 mM KCl, 2 mM MgCl2, 1 mM DTT (added prior to use), 1:100 protease inhibitor (added prior 10 min at 4°C. Cell pellets were dissolved in 15 mL nuclear isolation buffer [10 mM PIPES PH 7.4, 10 mM KCl, 2 mM MgCl2, 1 mM DTT (added prior to use), 1:100 protease inhibitor (added prior to use)], pH

adjusted to 7.4 using 1 M KOH]. Then, cells were lysed on ice in a 15 mL Dounce 102 homogenizer with pestle A (KIMBLE Kontes 885002-0015) by moving the pestle slowly up and down 20 times, followed by incubation on ice for 20 min and another 20 strokes. Next, each 5 mL of lysed extract was loaded slowly on top of a sucrose cushion prepared earlier. Then the tubes were spun for 15 min at 800 g at 4°C. The supernatant was removed carefully for a good recovery of the nuclei pellet in the bottom of the tube. Nuclei pellets were resuspended in 1 mL of HBSS, then spun for 5 min at 5,000 g at 4°C using a benchtop refrigerated centrifuge. Then, the nuclei pellet was resuspended in 3 mL HBSS, and 1 µL was taken to quantify the nuclei before the 3 mL was split over two microfuge tubes and spun for 5 min at 5,000 g at 4°C using a benchtop refrigerated centrifuge. Finally, the nuclei pellet was dissolved into an adequate total volume to obtain 1 million nuclei per 0.1 mL of Nuclei storage buffer (NSB) [10 mM PIPES pH 7.4, 10 mM KCl, 2 mM MgCl2, 50% glycerol, 8.5% sucrose, 1 mM DTT (added prior to use), 1:100 protease inhibitor (added prior to use)]. Each 0.5 mL of NSB containing 5 million nuclei was transferred to a microfuge tube and stored at -80°C.

3C (Chromosome Conformation Capture)

3C was performed as described in "From cells to chromatin: Capturing snapshots of genome organization with 5C technology" (Ferraiuolo *et al.*, 2012). Crosslinking: 1.25 mL of 37% formaldehyde was added to 40 mL of HBSS. 50 million cells or nuclei were washed twice using 20 mL of HBSS and then pelleted

at 500 g for 10 min. The pellet was resuspended in 5 mL HBSS and then added 103 to 41.25 mL of HBSS and formaldehyde (final formaldehyde concentration was 1%). The sample was incubated at RT for 10 min on a rocking platform. Afterward, to stop cross-linking 2.5 mL of 2.5 M glycine was added and samples were incubated at RT for 5 min on a rotating platform. To pellet the crosslinked cells or nuclei the sample was centrifuged at 800 g for 10 min at 4°C. After discarding the supernatant the pellet was washed twice using HBSS. Next, the pellet was either processed immediately as described below or was stored at -80 °C after flash freezing using liquid nitrogen.

Cell lysis: (This step was included when cells are used, but was skipped for 3C with purified nuclei).Cells were lysed by adding 2 mL of cold lysis buffer [10 mM Tris-HCI (pH=8.0), 10 mM NaCI, 0.2% Igepal CA-630 (NP40)] and 20 μ L of 100x Protease inhibitors. The sample was incubated on ice for 15 min to let the cells swell. The cells were lysed on ice using the homogenizer with pestle A (KIMBLE Kontes 885300-0002) by moving the pestle slowly up and down 30 times and incubating on ice for 1 min followed by another 30 strokes. The sample was transferred to two 1.5 mL microcentrifuge tubes, spun at 5,000 g at RT for 5 min using a benchtop centrifuge.

Digestion: each pellet was washed using 1 mL cold 1X NEBuffer 2.1, then spun at 5,000 g for 5 min at RT using a benchtop centrifuge, afterward each pellet was resuspended in 250 μ L of 1X NEB2.1 buffer, and the two pellets were pooled (~ 500 μ L). 50 μ L aliguots of the suspension were transferred to 10 new 1.5 mL

microfuge tube and 292 μ L of 1x NEBuffer 2.1 was added to each tube. Next, 38 104 μ L of 1% SDS was added per tube and mixed well, the samples were incubated at 65°C for 10 min, then placed on ice. 44 μ L of 10% Triton X-100 was added to each tube to quench SDS. Finally, 400 U of EcoRI (NEB R0101L) was added per tube and incubated at 37°C overnight on a thermocycler (with 900 rpm for 30 sec every 4 min).

Ligation: 86 μ L of 10% SDS was added to the digested samples and the samples were then incubated at 65°C for 30 min for EcoRI inactivation after which the tubes were placed on ice. Each sample was then transferred to a 15 mL conical tube and 7.69 mL of ligation mix was added [820 μ L 10% Triton X-100, 820 μ L 10x ligation buffer (500 mM Tris-HCI pH7.5, 100 mM MgCl2, 100mM DTT), 82 μ L 10 mg/mL BSA, 82 μ L 100 mM ATP and 5.886 μ L ultrapure distilled water]. Finally, 10 U of T4 ligase (Invitrogen 15224090) was added per tube before incubation at 16°C for 2 hr on a thermocycler (with 900 rpm for 30 sec every 4 min).

Reverse Crosslinking: 50 μ L of 10 mg/mL proteinase K (Fisher BP1750I-400) was added per tube, the sample was incubated at 65°C for 4 hr followed by a second addition of 50 μ L 10 mg/mL Proteinase K and overnight incubation at 65°C on a thermocycler (with 900 rpm for 30 sec every 4 min).

DNA purification: Tubes were cooled at room temperature, at this stage each tube contains ~ 8.21 mL final volume. The samples from every two tubes were

combined to a 50 mL conical tube (~16,42 mL) to have five tubes in total. DNA was extracted by adding an equal volume of 17 mL of saturated phenol pH8.0: chloroform (1:1) (Fisher BP1750I-400) and vortexing for 3 min. Then the mix was transferred to a 15 mL phase-lock tube (Quiagen 129073) followed by spinning tubes at 5,000 g for 10 min. The upper phase was taken to a 50 mL tube to start the second extraction. We added an equal volume of 17 mL saturated phenol pH 8.0: chloroform (1:1), vortexing for 1 min. Then the upper phase was transferred to a 15 mL phase-lock tube, and tubes were centrifuged at 5,000 g for 10 min. We pooled all the upper phases from all 5 tubes ~ 85 mL into a single 300 mL high-speed centrifuge tube to precipitate the DNA. 8.5 mL (1/10 volume) of 3M sodium acetate pH 5.2 was added and brief vortexing was performed, then 212 mL (2.5 volumes) of ice-cold 100% ethanol was added, and the tube was inverted slowly several times and incubated at -80° C for 1 hr. Afterward, the DNA was pelleted at 16,000 g for 30 min at 4°C. The supernatant was discarded and the pellet was dissolved in 500 µL 1X TLE and transferred to a 0.5 mL AMICON Ultra Centrifuge filter (UFC5030BK EMD Millipore). The column was centrifuged for 5 min at 14,000 g and the flow-through was discarded. The column was washed 4 times using 450 µL of 1X TLE for desalting DNA. After the final wash, the library remaining in the column (~50 μ L) was eluted in 30 μ L of 1XTLE, the column was flipped upside down into a new tube to collect DNA by centrifugation for 3 min at 4,000 g. RNA was degraded by adding 1 µL of 10 mg/mL RNAase A and incubation for 30 min at 37°C.

105

Quality control assessment: to test the quality of the 3C library we used PCR to 106 amplify a specific ligation product formed by two nearby restriction fragments, using the following primers:

GPF33: GACCTCTGCACTAGGAATGGAAGGTTAGCC

GPF23: GACTAATTCCTGACACTACTTGAGGGATAC

The amplicon was digested with EcoRI to assess the efficiency of 3C ligation.

BAC library for 3C-PCR

BAC DNA was generated as described (Dostie *et al.*, 2006). A control ligation library covering the Beta-globin locus (ENCODE region ENm009) was generated using BACs overlapping the region. Starting with a mixture of DNA of seven BACs (CTC-775N13, RP11-715G8, CTD-3048C22, CTD3055E11, CTD-2643I7, CTD-3234J1, and RP11-589G14) (Invitrogen), mixed in equimolar ratios, we used the same steps described in the 3C protocol above starting from the digestion step. BAC clones were digested with EcoRI, then randomly ligated, and the DNA was purified. The BAC ligation library reflects random ligation of EcoRI fragments throughout the beta-globin locus, so any difference in PCR signal for 3C primer pairs along the beta-globin locus due to differences in primer efficiency can be corrected by normalizing the amount of PCR product obtained with the 3C library to the amount obtained with the BAC ligation library.

Chromosome Conformation Capture Carbon Copy (5C)

Experimental design

Probes were designed as described (Dostie *et al.*, 2006). 213 5C probes were designed for a ~1 Mb region (chr11:4730996 -5729937; hg18) around the Beta-globin locus at EcoRI restriction sites using publicly available 5C primer design tools (Lajoie *et al.*, 2009). Probes were designed according to a single alternating scheme exactly as described before (Lajoie *et al.*, 2009) and the genomic uniqueness of all primers was verified with the SSAHA algorithm. For each EcoRI fragment at the 1 Mb target region a primer was designed. 104 5' forward (FOR) and 109 5' reverse (REV) primers were designed.

Generation of 5C libraries

5C libraries were generated as described before (Ferraiuolo *et al.*, 2012), with three modifications. First, we skipped the gel purification after the adaptor ligation and replaced this with a 1:1 Ampure step to remove unligated DNA and adaptors. Second, barcoded Illumina adaptors were used. Third, we performed the final PCR using TruSeg DNA LT kit Set A (REF 15041757).

Annealing: The 5C probes were pooled and combined with the 3C template each reaction contained 800,000 genome copies of 3C template and 0.2 fmol per 5C probe [800,000 genome copies of 3C template, 2 μ L of 10X NEB4 (NEB B7004S), 2.75 μ L of Salmon Sperm DNA (250 ng; (Invitrogen T 15632011), 0.25 μ L of 1 fmol/ μ L probes , up to 20 μ L ultrapure distilled water]. We set up 8

annealing reactions for each library in a 96-well PCR plate. We then incubated the samples in a PCR machine and ran the following program [95°C for 9 min, Ramp 0.1°C/sec to 55 C, then keep at 55°C for 12 hr].

Ligation: We ligated 5C probe pairs, which represent a specific ligation junction in the 3C library, by adding 20 µL of ligation mix 2 µL of [10X Tag DNA ligase buffer (NEB B0208S), 0.25 µL Tag DNA ligase (NEB M0208S), 17.75 uL ultrapure distiller water] while the samples are kept in the PCR block at 55°C. We then incubated the reactions for 1 hr at 55°C followed by a 10 min incubation at 65°C; samples were then cooled to 4°C. Negative controls (no ligase, no template, no 5C oligonucleotide) were included to ensure the absence of any contamination.

PCR amplification: Universal emulsion primers were used for amplification of the ligated product by using 5C forward and reverse emulsion primers [Forward_primer: CCTCTCTATGGGCAGTCGGTGAT. Reverse_primer : CTGCCCCGGGTTCCTCATTCTCT] for 25 PCR cycles [6 µL of ligation product, 2.5 µL of 10XPCR (600 mM Tris-SO4, pH 8.9, 180 mM (NH4)2SO4), 1.8 mM MgCl2, 0.2 mM dNTP, 0.5 µL F-emulsion primer (80 µM), 0.5 µL R-emulsion primer (80 µM), 0.225 µL AmpliTagÒ Gold DNA polymerase, ultrapure distilled water to bring volume up to 25 μ L]. We then amplified DNA using this PCR program: [95° 9 min, 25 cycles (95°C 30 s, 65°C 30 s, 72° 30 s), 95°C 30 s, 65°C 30 s, 72°C 8 min, 4°C].

We pooled all the PCR reactions for the same library together and concentrated 109 the DNA to 50 μ L using 0.5 mL AMICON Ultra Centrifuge filter (UFC5030BK EMD Millipore). DNA was then loaded on a 2% agarose gel, along with a low molecular weight ladder, and the gel was run in a 4°C room at 200 volts for 90 min. The 150 bp DNA that corresponded to the ligated 5C probes was isolated from the gel using the QIAquick Gel Extraction Kit Protocol (QIAGEN 28115). DNA was finally eluted in 32 μ L of 1XTLE.

A-tailing: A dATP was added to the 3' ends of the 5C library by adding 18 μ L of A-tailing mix [5 μ L NEB buffer 2.1, 10 μ l of 1 mM dATP, 3 μ L Klenow exo (NEB M0212S)] to the 32 μ L of DNA sample from the previous step. The reaction was then incubated in a PCR machine [at 37°C for 30 min, then at 65°C for 20 min, and finally cooled down to 4°C]. Next, the tube was placed on ice immediately. 1:1 Ampure was used to remove unligated adaptors. The DNA was finally eluted in 40 μ L 1X T4 DNA Ligase buffer (Invitrogen).

Illumina adapter ligation and paired-end PCR: For this step, we used the TruSeq DNA LT kit Set A (REF 15041757). 10 μ L of ligation mix [5 μ L Illumina pairedend adapters, 3 μ L T4 DNA ligase Invitrogen, 2 μ L 5x T4 DNA ligase buffer (Invitrogen 5X)] was added to the 40 μ L sample from the previous step. The ligation sample was then incubated at RT for 2 hours on a tube rotator. Afterward, the sample was run on a 2% agarose gel in a cold room 4°C at 150 volts for 120 min along with a low molecular weight ladder. The 270 bp band that corresponds to 5C products (150 bp) ligated to the two adaptors (64 bp) was extracted from the gel and isolated using the QIAquick Gel Extraction Kit 110 (QIAGEN 28115). DNA was finally eluted in 30 µL 1XTLE.

Pre-digestion of nuclei (liquify chromatin)

Purified nuclei as described above (K562 nuclei purification) were placed on ice and 1 mL of HBSS was added to each 0.5 mL of 5 million frozen nuclei. After thawing, nuclei were centrifuged 5 min at 5,000 g. The nuclei pellet was washed twice with 1XNEB3.1 for nuclei that would be digested with DpnII or 1XNEB2.1 for nuclei that would be digested with HindIII. The nuclei were pelleted for 5 min at 5,000 g after each wash.

Isolated nuclei: a sample of 5 million nuclei was resuspended in 1,250 µL of 1X NEB3.1 as control, and then processed immediately for Hi-C starting at the crosslinking step (see below Hi-C 2.0 protocol).

Undigested nuclei: Each sample of two million nuclei was resuspended in 500 µL of 1X NEB3.1 on ice, as and control for the pre-digestion and then treated as described immediately below.

DpnII pre-digestion: Each sample of two million nuclei was resuspended in 500 µL of 1X NEB3.1 on ice. Next, 120 U of DpnII (NEB R0543S) was added to the sample in order to obtain 10 U DpnII/µg DNA and then treated as described immediately below.

FatI pre-digestion: Each sample of two million nuclei was resuspended in 500 μ L 111 of 1X NEB3.1 on ice. Next, 120 U of FatI (NEB R0650L) was added to the sample in order to obtain 10 U FatI/ μ g DNA and then treated as described immediately below.

HindIII pre-digestion: Each sample of two million nuclei was resuspended in 500 μ L of 1X NEB2.1 on ice. Next, 600 U of HindIII (NEB R0104T) was added to the sample in order to obtain 50 U HindIII/ μ g of DNA and then treated as described immediately below.

Next, control and pre-digestion samples were incubated at 37°C on a thermocycler (900 rpm for 30 sec every 4 min) for 5 min up to 16 h. Afterward, samples were placed on ice for 10 min.For DpnII-seq and assessment of fragmentation level , a final volume of 10 mM of EDTA was added to inactivate the endonuclease, followed immediately by the DpnII-seq protocol (details of protocols below. DpnII-Seq) or DNA purification for fragment analyzer analysis. For Hi-C, we proceeded immediately to the first step of the protocol (crosslinking as described below). For microscopy, nuclei samples were cross-linked with a 4% final concentration of paraformaldehyde.

Hi-C 2.0

Hi-C was performed as described (Belaghzal, Dekker and Gibcus, 2017) with some modifications in the crosslinking and lysis step as described below. Hi-C was performed following the exact same protocol for mock treated and for predigested nuclei, even though for pre-digested nuclei chromatin was already fragmented prior to fixation. There are several reasons to perform the full Hi-C procedure even for pre-digested nuclei. First, pre-digestion (e.g. with DpnII) leads to partial digestion. During the subsequent Hi-C procedure chromatin is digested with DpnII again, but this time the chromatin has been opened with SDS treatment. This allows more complete digestion. Second, by performing the full Hi-C procedure for pre-digested nuclei allows direct comparison of data obtained with mock treated nuclei, HindIII pre-digested nuclei, DpnII pre-digested nuclei and Fat1 pre-digested nuclei.

Crosslinking: isolated, undigested, and pre-digested (with liquified chromatin) nuclei were not pelleted after the pre-digestion step above but were crosslinked immediately as follows: for each sample 1,250 μ L volume of nuclei in the digestion buffer was transferred to a 21.875 mL mix [625 μ L of 37% formaldehyde + 21.25 mL of HBSS]. For intact cells: 5 million K562 cells or nuclei were washed twice with 15 mL of HBSS and pelleted at 300 g for 10 min, then resuspended in 2.5 mL of HBSS. The sample was transferred to 20.625 mL crosslinking mix [625 μ L of 37% formaldehyde + 20 mL of HBSS].

All samples were incubated at RT for 10 min on a rocking platform. Next, to stop cross-linking 1.25 mL of 2.5 M glycine was added to each sample and the mix was incubated at RT for 5 min on a rocking platform. To pellet the crosslinked cells/nuclei, the sample was centrifuged at 1,000 g for 10 min at 4°C. The

supernatant was discarded and the pellet was washed twice with HBSS before 113 going to the next step or storing samples at -80°C.

Cells lysis: This step is not needed for isolated, undigested, and pre-digested (with liquified chromatin) nuclei. For Hi-C with intact cells: the 5 million crosslinked cells were lysed by adding 1 mL cold lysis buffer [10 mM Tris-HCl (pH=8.0), 10 mM NaCl, 0.2% Igepal CA-630 (NP40)] and 10 μ L of 100X Protease inhibitors. The sample was incubated on ice for 15 min to let the cells swell. The cells were lysed on ice using a dounce homogenizer with pestle A (KIMBLE Kontes 885300-0002) by moving the pestle slowly up and down 30 times and incubating on ice for 1 min followed by another 30 strokes. The sample was transferred to a new 1.5 mL microcentrifuge tube, and the sample was centrifuged at 5,000 g at RT for 5 min.

Digestion: from each sample (isolated undigested, and pre-digested (with liquified chromatin) nuclei and lysed cells) the pellet was resuspended in 500 μ L of ice-cold 1X NEBuffer 3.1, and pelleted for 5 min at 4,000 g. The pellet was washed twice using 500 μ L of ice-cold 1X NEBuffer 3.1. After the last wash, the pellet was resuspended in 350 μ L 1X NEBuffer 3.1, and 8 μ L was taken and kept at 4°C to assess the DNA integrity later. 38 μ L of 1% SDS was added to 342 μ L (380 μ L total volume), and the mixture was resuspended and incubated for 10 min at 65°C. The tube was placed on ice immediately afterward. Next 43 μ L of 10% Triton X-100 was added and the sample was mixed gently by pipetting. The

tubes were placed at room temperature and 12 μL of 10X NEBuffer 3.1 was added. Then 400 U of DpnII (R0543L) was added and mixed gently before an overnight incubation at 37°C on a thermocycler (with 900 rpm for 30 sec every 4 min).

Biotin Fill-in: After overnight digestion, the sample was incubated at 65°C for 20 min in order to inactivate the restriction enzyme. Then, 10 μ L of the digested sample was taken and kept at 4°C to assess the digestion efficiency later. DNA ends were marked with biotin-14-dATP by adding 60 μ L of biotin fill-in master mix [1XNEB 3.1, 0.25 mM dCTP, 0.25 mM dGTP, 0.25 mM dTTP, 0.25 mM biotin-dATP (ThermoFisher#19524016), 50U Klenow polymerase Polymerase I (NEB M0210L)]. Next, the sample was incubated for 4 h at 23°C on a thermocycler (with 900 rpm for 30 sec every 4 min). Finally, the sample was placed on ice immediately for 15 min before proceeding to the next step.

Ligation: After fill-in, the total sample volume was ~535 μ L. Ligation was performed by adding 665 μ L of ligation mix [240 μ L of 5x ligation buffer (1.8X) (Invitrogen), 120 μ L 10% Triton X-100, 12 μ L of 10 mg/mL BSA, 50 μ L T4 DNA ligase (Invitrogen 15224090), and 243 μ L ultrapure distilled water (Invitrogen)], to make a total volume of 1,200 μ L. The reaction was then incubated at 16°C for 4 hours in a Thermomixer with interval shake.

Reverse Crosslinking: 50 μ L of 10 mg/mL proteinase K (Fisher BP1750I-400) was added after ligation, the sample was incubated at 65°C for 4 hr followed by a second addition of 50 μ L 10 mg/mL Proteinase K and overnight incubation 65°C

DNA purification: Reactions were cooled to room temperature and the 1.3 mL total volume was transferred to a 15 mL tube. The DNA was extracted by adding an equal volume of 1.3 mL of saturated phenol pH 8.0: chloroform (1:1) (Fisher BP1750I-400) and vortexing for 1 min. Then the total volume of 2.6 mL was transferred to a 15 ml phase-lock tube (Quiagen #129065) and tubes were centrifuged at 5,000 g for 10 min. The upper phase was transferred to a 15 mL tube to start the second extraction. An equal volume of 1.3 mL saturated phenol pH8.0: chloroform (1:1) was added and the sample was vortexed for 1 min. Then the mix was transferred to 15 ml phase-lock tube (Quiagen #129065) followed by spinning tubes at 5,000 g for 10 min. The upper phase of ~1.3 mL was transferred to a 15 mL tube (high speed) to precipitate the DNA. 1/10 volume (130 µL) of 3 M sodium acetate pH 5.2 was added and the sample was briefly vortexed. Then, 2.5 volumes of ice-cold 100% ethanol 3.25 mL was added, the tube was inverted slowly several times and then incubated at -80° C for 1hr. Next, the DNA was pelleted at 16,000 g for 30 min at 4°C. The supernatant was discarded and the pellet was dissolved in 500 μ L 1X TLE and transferred to a 0.5 mL AMICON Ultra Centrifuge filter (UFC5030BK EMD Millipore). The column was spun for 5 min at 14,000 g and the flow-through was discarded. The column was washed 4 times using 450 µL of 1X TLE for desalting of DNA. After the final

wash the DNA remaining in the column (~50 μ L) was eluted in 52 μ L of 1XTLE. 116 The column flipped upside down into a new tube to collect DNA and spun for 3 min at 4,000 g, the volume was adjusted to 102 μ L. RNA was degraded by adding 1 μ L of 10 mg/mL RNAase A and incubation for 30 min at 37°C. To quantify the DNA concentration, 2 μ L of the final DNA sample along with the first 8 μ L sample taken before digestion, the 10 μ L sample taken after digestion, and various amounts of the 1 kb ladder (NEB#N3232s) were run on 1% Agarose gel.

Removal of Biotin from unligated ends: To remove biotinylated nucleotides at DNA ends that did not ligate, the Hi-C sample was treated with T4 DNA polymerase. For each Hi-C sample, we assembled the following reaction: [up to 5 μ g of Hi-C library, 5 μ L 10x NEBuffer 3.1, 0.025 mM dATP, 0.025 mM dGTP and 15 U T4 DNA polymerase (NEB # M0203L). The samples were brought up to 50 μ L total volume adding ultrapure distilled water . Reactions were incubated at 20°C for 4 hours, the enzyme was then inactivated by incubation of the reaction for 20 mins at 75°C and placed at 4°C. Next, the samples were pooled and the volume was brought up to 130 μ L 1XTLE in preparation for sonication.

Sonication: the DNA was sheared to a size of 100-300 bp using a Covaris instrument [Duty cycle 10%, Intensity 5, Cycles per Burst 200, set Mode Frequency sweeping, continuous degassing, process time 60 sec, Number of cycles] for 3 cycles. The volume was brought up to 500 µL using TLE for Ampure fractionation.

Size fractionation using AMpure XP: 500 µL AMpure beads (Beckman Coulter 117 A63881) were added to a 1.5 mL tube labeled as 1.1X. Then the tube was placed on the Magnetic Particle Separator (MPS) for 5 min, and the supernatant was removed. Beads were resuspended in 150 µL AMpure mixture in order to make the 1.1X solution. 400 µL of AMpure mixture was added to 500 µL of sonicated DNA from the previous step and the tube was labeled 0.8X. The sample was vortexed and spun down briefly followed by incubation at RT for 10 min on a rotating platform. Then the tube was placed on the MPS for 5 min at RT. The supernatants were collected and added to the 1.1X tube, the tube was briefly vortexed and spun down followed by incubation at RT for 10 min on a rotating platform. Then the tube was placed on the MPS for 5 min at RT. The supernatant was discarded and the beads in 0.8X and 1.1X tubes were washed twice with 1 mL 70% ethanol. Beads were reclaimed by the MPS for 5 min. Beads were then air-dried on the MPS until ethanol had evaporated completely. Next, 51 µL of 1XTLE was added to the 0.8X and 1.1X tubes to resuspend the DNA from the beads. Tubes were incubated at RT on a rotating platform for 10 min. Then the tubes with AMpure beads from both 0.8X and 1.1X tubes were placed on the MPS for 5 min. Finally, the supernatants were transferred to 1.7 mL tubes labeled 0.8X and 1.1X. Our sample with DNA that ranges from 100-300 bp is in the 1.1X sample, the 0.8X sample was kept in case more DNA was needed. DNA from both samples 0.8X and 1.1X were quantified by running 1 μ L on a 2%

agarose gel along with different amounts of low molecular weight DNA ladder 118 (100 ng, 200 ng, 400 ng).

End Repair: 50 µL of Hi-C sample was transferred to a PCR tube, then 20 µL of the end-repair mix [3.5X NEB ligation buffer (NEB B0202S), 17.5 mM dNTP mix, 7.5 U T4 DNA polymerase (NEBM0203L), 25 U T4 polynucleotide kinase (NEB M0201S), 2.5 U Klenow polymerase Polymerase I (NEB M0210L)] was added. The 70 µL total volume reaction was then incubated at 37°C for 30 min, followed by incubation at 65°C for 20 min to inactivate Klenow polymerase, and then the sample was put at 4°C. The volume was brought up to 400 µL using 1X TLE for the next step.

Biotin pull-down: All the following steps were performed with 1.5 mL loBind tubes (Eppendorf 22431021). 15 µL of MyOne streptavidin C1 beads mix (Thermo Fisher 65001) was transferred to a 1.5 mL tube. The beads were washed twice by adding 400 µL of TWB [5 mM Tris-HCl pH8.0, 0.5 mM EDTA, 1 M NaCl, 0.05% Tween20] followed by incubation for 3 min at RT. The tube was then placed on an MPS for 1 min and the supernatant was removed. After the washes, the beads were resuspended in 400 µL of 2X Binding Buffer (BB) [10 mM Tris-HCl pH8, 1 mM EDTA, 2 M NaCl] and mixed with the 400 µL DNA from the previous step in a new 1.5 mL tube. The mixture was incubated for 15 min at RT with rotation, the tube was then placed on the MPS for 1 min and the supernatant was removed. The DNA bound to the beads was washed by adding 400 µL of 1X BB and transferred to a new tube. The beads were reclaimed

against the MPS for 1 min, and the supernatant was discarded. The second wash used 100 μ L of 1X TLE, beads were reclaimed against MPS for 1 min, and the supernatant was discarded. Finally, the DNA bound to the beads was eluted in 32 μ L of 1X TLE.

A-tailing: A dATP was added to the 3' ends by adding 18 μ L of A-tailing mix [5 μ L NEB buffer 3.1, 10 μ L of 1 mM dATP, 3 U Klenow exo (NEB M0212S)] to the 32 μ L of DNA sample from the previous step. The reaction was incubated in a PCR machine [at 37°C for 30 min, then at 65°C for 20 min, followed by cool down to 4°C]. Next, the tube was placed on ice immediately. The sample was transferred to a 1.5 mL loBind tube, the tube was placed on the MPS for 1 min and the supernatant was removed. The streptavidin beads bound to DNA were washed twice using 100 μ L 1X T4 DNA Ligase Buffer (Invitrogen). Finally, streptavidin beads bound to DNA were resuspended in 40 μ L 1X T4 DNA Ligase buffer (Invitrogen).

Illumina adapter ligation and paired-end PCR: For this step, the TruSeq DNA LT kit Set A (REF#15041757) was used. 10 μ L of ligation mix [5 μ L Illumina paired-end adapters, 3 μ L T4 DNA ligase Invitrogen, 2 μ L 5x T4 DNA ligase buffer (Invitrogen 5X)] was added to the 40 μ L Hi-C sample from the previous step. The ligation sample was then incubated at RT for 2 hours on a rotator. The sample was transferred to a 1.5 mL loBind tube, the tube was placed on the MPS for 1 min and the supernatant was removed. The streptavidin beads bound to DNA

were washed twice with 400 μ l of TWB, then twice using 100 μ L 1X TLE. Finally, 120 the sample was resuspended in 20 μ L 0f 1XTLE.

Illumina Truseg Kit for PCR: We performed three trial PCR reactions as follows [2.5 µL DNA bound to beads, 2 µL of Primers mix (TruSeg DNA LT kit Set A 15041757)), 10 µL Master Mix (TruSeq DNA LT kit Set A 15041757), 10.5 µL of ultrapure distilled water (Invitrogen)]. We split the 25 µL over three PCR tubes (5 μ L, 5 μ L, 15 μ L per tube). Each of the three samples was then amplified with different numbers of PCR cycles (6, 8, 10 respectively) to assess the Hi-C library quality: [30 sec at 98°C, n cycles of (30 sec at 98°C, 30 sec at 65°C, 30 sec at 72°C), 5 min at 72°C, hold at 10°C]. 10 µL was taken from the 15 µL sample (with 10 PCR cycles), the 10 μ L sample was then digested with Clal for 1 h by adding 10 μ L of digestion mix [1.5 μ L 10x NEB Cutsmart buffer, 1.5 μ L Clal (NEB R0197S), 7 µL ultrapure distilled water]. The 5 µL of each PCR cycle sample along with the 20 µL digested sample, and titration of the low molecular ladder (100 ng, 200 ng, 400 ng) (NEB) were run on a 2% Agarose gel. After digestion with Clal, a downward shift of the amplified DNA to smaller sizes is expected, which indicates DNA ends were correctly filled in and ligated (creating a Clal site). The number of PCR cycles to generate the final Hi-C material for deep sequencing was chosen based the minimum number of PCR cycles in the PCR titration that was needed to obtain sufficient amounts of DNA for sequencing using the remaining 17.5 μ L Hi-C sample.

Dpnll-Seq

For each DpnII-Seq library, 10 million nuclei were used right after the predigestion procedure described above (Pre-digestion of nuclei). The pre-digested nuclei were then treated as follows:

Proteinase K: 50 μL of 10 mg/mL proteinase K (ThermoFisher # 25530) was added to each 500 µL pre-digested nuclei sample (2 million nuclei) (See Methods: Pre-digestion) and the 5 tubes were incubated at 65°C for 3 hours. DNA purification: Tubes were cooled to room temperature and all 5 samples were pooled in a single 15 mL tube (2.75 mL total volume). The DNA was extracted by adding an equal volume of 2.75 mL of saturated phenol pH8.0: chloroform (1:1) (Fisher BP1750I-400), followed by vortexing for 1 min. The sample (5.5 mL) was transferred to a 15 mL phase-lock tube (Quiagen #129065) followed by centriguation at 5,000 g for 10 min. The upper phase was transferred to a 15 mL tube to start the second extraction. An equal volume of 2.75 mL saturated phenol pH8.0: chloroform (1:1) was added, followed by vortexing for 1 min. Then the mix was transferred to a 15 mL phase-lock tube (Quiagen #129065) followed by centrifugation at 5,000 g for 10 min. The upper phase of ~ 2.75 mL was transferred to a 15 mL tube (high speed), 1/10 volume (275 μ L) 3M sodium acetate pH 5.2 was added followed by brief vortexing and then 2.5 volumes of ice-cold 100% ethanol (6.875 mL) were added. The tube was inverted slowly several times, incubated at -80°C for 1 hr and then DNA was pelleted by

centrifugation at 16,000 g for 30 min at 4°C. The supernatant was discarded and 122 the pellet was dissolved in 500 μ L 1X NEB3.1 and transferred to a 0.5 mL AMICON Ultra Centrifuge filter (UFC5030BK EMD Millipore). The column was centrifuged for 5 min at 14,000 g and the flow-through was discarded. The column was washed 4 times using 450 μ L of 1X NEB3.1 for desalting of DNA. After the final wash, the library remaining in the column (~50 μ L) was eluted in 450 μ L of 1XNEB3.1; the column was flipped upside down into a new tube to collect DNA and centrifuged for 3 min at 4,000 g. ~500 μ L of DNA was recovered. RNA was degraded by adding 1 μ L of 10 mg/mL RNAase A and incubation for 30 min at 37°C. The amount of DNA was estimated by running an aliguot on a 1% Agarose gel along with a 1kb ladder (NEB#N3232s).

Biotin Fill-in: 1XNEB3.1 was added the reaction to a final volume of 680 μ L, and then the 680 μ L was split over 2 1.5 mL tubes. DNA ends were filled in and marked with biotin-14-dATP. To each tube 60 μ L of biotin fill-in master mix was added: [1xNEB2.1, 0.25 mM dCTP, 0.25 mM dGTP, 0.25 mM dTTP, 0.25 mM biotin-dATP (ThermoFisher#19524016), 50 U Klenow polymerase Polymerase I (NEB M0210L)]. Samples were incubated at 37°C in a Thermocycler for 75 mins. Next, the tubes were placed on ice immediately for 15 mins, and samples from the 2 tubes were combined to obtain a final volume ~800 μ L. Amicon filters were used to reduce the volume of the final sample from 801 μ L to 130 μ L.

Sonication: DNA was sonicated to a size of 100 – 300 bp using a Covaris instrument (Duty Cycle 10%, Intensity 5, Cycles per Burst 200, set Mode

Frequency sweeping, continuous degassing, process time 60 sec, Number of cycles) for 4 cycles. The 130 μ L of sonicated DNA was transferred to a 1.5 mL tube and 1XTLE was added to a total volume of 500 μ L. DNA fragment size was determined by running 2 μ L of DNA along with low molecular ladder (NEB) on a 2% agarose gel.

Size fractionation using AMpure XP: 500 µL AMpure beads (Beckman Coulter A63881) were added to a 1.5 mL tube labeled as 1.1X. The tube was placed on the MPS for 5 min, and the supernatant was removed. Beads were resuspended in 150 µL AMpure mixture in order to make 1.1X. 400 µL of AMpure mixture was added to 500 µL of sonicated DNA from the previous step and the tube was labeled 0.8X. The sample was vortexed and centrifuged briefly using a tabletop small centrifuge followed by incubation at RT for 10 min on a rocking platform. Then the tube was placed on the MPS for 5 min at RT. The 0.8X supernatants were collected and added to the 1.1X tube, the tube was briefly vortexed and centrifuged followed by incubation at RT for 10 min on a rocking platform. The tube was placed on the MPS for 5 min at RT, and the supernatant discarded. Beads in the 0.8X and 1.1X tubes were washed twice with 1 mL 70% ethanol, reclaiming beads against the MPS for 5 min. Beads on the MPS were then dried until ethanol had evaporated completely. Next, 51 µL of 1XTLE was added to the 0.8X and 1.1X tubes to elute DNA from the beads. Tubes were incubated at RT on a rocking platform for 10 min. The 0.8X and 1.1X tubes were placed on the MPS for 5 min. Finally, the supernatants were transferred to 1.7 mL tubes

labeled 0.8X and 1.1X. The 1.1X sample contains DNA that ranges in size from 124 100-300 bp. The DNA in the 0.8X sample was kept in case more DNA was required, in which case the DNA would be sonicated using 2 cycles followed by a similar round of size fractionation as described above. The amount of DNA from both samples 0.8X and 1.1X was quantified by running 1 μ L on a 2% agarose gel along with a titration of low molecular weight DNA ladder (100 ng, 200 ng, 400 ng).

End Repair: 50 µL from the 1.1X sample was transferred to a PCR tube, and 20 µL of end repair mix was added: [3.5X NEB ligation buffer (NEB B0202S), 0.875 mM dNTP mix, 0.375 U/µL T4 DNA polymerase, 1.25 U/µL T4 polynucleotide kinase, 0.125 U/µL Klenow DNA polymerase]. The 70 µl total volume reaction was incubated for 30 min at 20°C in a PCR machine and then placed on ice. The DNA was purified by 1:2 Ampure, by adding 140 µL 2X Ampure solution to the 70 µL DNA sample followed by incubation for 5 min at RT. The tube was placed on the MPS for 4 min to reclaim the beads and the supernatant was discarded. The beads were washed twice with 1 mL of 70% ethanol while on the MPS. After beads were dried DNA was eluted in 32 µL TLE (pH 8.0) and incubation for 10 min at RT. The supernatant was transferred to a 1.5 mL tube.

A-tailing: A dATP was added to the 3' ends by adding 18 μ L of A-tailing mix [5 μ L NEB buffer 3.1, 10 μ L of 1 mM dATP, 3 U Klenow exo (NEB M0212S)] to the 32 μ L of DNA sample from the previous step. The reaction was then incubated in a PCR machine at 37°C for 30 min followed by incubation 65°C for 20 min and

cooling down to 4°C. The tube was placed on ice. The volume was brought to 100 μ L by adding 1X NEB2.1. The DNA was then purified by adding 1:2 Ampure mix (200 μ L of Ampure was added to the 100 μ L final DNA volume). Finally, the DNA was eluted in 40 μ L of 1X T4 DNA ligase buffer (Invitrogen 5X).

Illumina adapter ligation and paired-end PCR: For this step we used the TruSeq DNA LT kit Set A (REF#15041757). 50 μ L of ligation mix [25 μ L Illumina pairedend adapters, 15 μ L T4 DNA ligase Invitrogen, 10 μ L 5X T4 DNA ligase buffer (Invitrogen 5X)] was added to the 40 μ L sample from the previous step. The ligation sample was then incubated at RT for 2 hours on a rotator. Next, the DNA was purified by adding 1:1 Ampure solution (180 μ L of Ampure mix was added to the 90 μ L sample), the supernatant was discarded and beads were washed twice with 1 mL of 70% ethanol. After the last wash step, the beads were resuspended in 400 μ L of 1X TLE and incubated at RT on a rocking platform for 10 mins. The tube was placed on the MPS for 4 mins. Finally, the 400 μ L supernatant was transferred to a new tube.

Biotin pull-down: All the following steps are done using 1.5 mL loBind tube (Eppendorf 22431021). 15 μ L of MyOne streptavidin C1 beads mix (Thermo Fisher 65001) was transferred to a 1.5 mL tube. The beads were washed twice with 400 μ L of TWB [5 mM Tris-HCl pH8.0, 0.5 mM EDTA, 1 M NaCl, 0.05% Tween20] by incubation for 3 min at RT. After each wash, the tube was placed on the MPS for 1 min and the supernatant was removed. After the washes, the beads were resuspended in 400 μ L of 2X Binding Buffer (BB) [10 mM Tris-HCl

pH8.0, 1 mM EDTA, 2 M NaCl] and mixed with the 400 µL DNA from the previous step in a new 1.5 mL. The mixture was incubated for 15 min at RT with rotation. The tube was then placed on the MPS for 1 min and the supernatant was removed. The DNA bound to the beads was washed first by adding 400 µL of 1X BB and transferring to a new tube. The beads were reclaimed against the MPS for 1 min, and the supernatant discarded. 100 µL of 1X TLE was added and the beads were reclaimed against the MPS for 1 min, then the supernatant was discarded. Finally, the DNA bound to the beads was eluted in 32.5 µL of 1X TLE. PCR optimization: The Illumina Truseq Kit (DNA LT kit Set A (REF#15041757)) was used for PCR amplification of DNA for DpnII-Seq. The trial PCR reaction was set up as follows: [2.5 µL DNA bound to beads, 2 µL of Primers mix (Truseq kit), 10 µL Master Mix (Truseq kit), 10.5 µL of ultrapure distilled water (Invitrogen)]. The 25 µL was split over four PCR tubes (5 µL/per tube). Each of the four samples was incubated for different PCR cycles (6, 8, 10, or 12 cycles): [30 sec at 98°C, n cycles of (30 sec at 98°C, 30 sec at 65°C, 30 sec at 72°C), 7 min at 72°C, hold at 10°C]. The optimal PCR cycle number needed to get enough DNA for sequencing was determined by running the 4 PCR reactions on a 2% agarose gel along with low molecular ladder titration (100 ng, 200 ng, 400 ng). Three PCR reactions of 50 µL volume were then performed: [5 µL DNA bound to beads, 4 µL of Primers mix (Truseq kit), 20 µL Master Mix (Truseq kit), 21 µL of ultrapure distilled water (Invitrogen)]. The 3 PCR reactions were pooled together to obtain 150 µL total volume. The samples were reclaimed against the MPS for

126

1 min, then the PCR products (supernatant) were taken to new 1.5 mL tubes. 1:1 127 Ampure was performed for removal of primer dimers (150 μ L of Ampure and 150 μ L DNA sample). Finally, beads were resuspended in 35 μ L of TLE to elute the DNA. DNA that remained bound to beads was saved after a first wash using TBW followed by two washes with 1X TLE and then resuspended in 30 μ L of 1X TLE.

DpnII Pre-digestion size assessment

4 million cells were pre-digested for 4 hours using DpnII procedure described above (Pre-digestion of nuclei). The pre-digested nuclei were then treated as follows:

Proteinase K: 50 µL of 10 mg/mL proteinase K (ThermoFisher # 25530) was added to each 500 µL pre-digested nuclei sample (2 million nuclei) (See Methods: Pre-digestion) and the 2 tubes were incubated at 65°C for 3 hours. DNA purification: Tubes were cooled to room temperature and all 2 samples were pooled in a single 15 mL tube (1.1 mL total volume). The DNA was extracted by adding an equal volume of 1.1 of saturated phenol pH8.0: chloroform (1:1) (Fisher BP1750I-400), followed by vortexing for 1 min. The sample (2.2 mL) was transferred to a 15 mL phase-lock tube (Quiagen #129065) followed by centriguation at 5,000 g for 10 min. The upper phase was transferred to a 15 mL tube to start the second extraction. An equal volume of 1.1 mL saturated phenol pH8.0: chloroform (1:1) was added, followed by vortexing for 1

min. Then the mix was transferred to a 15 mL phase-lock tube (Quiagen #129065) followed by centrifugation at 5,000 g for 10 min. The upper phase of ~ 1.1 mL was transferred to a 15 mL tube (high speed), 1/10 volume (110 μ L) 3M sodium acetate pH 5.2 was added followed by brief vortexing and then 2.5 volumes of ice-cold 100% ethanol (2.75 mL) were added. The tube was inverted slowly several times, incubated at -80°C for 1 hr and then DNA was pelleted by centrifugation at 16,000 g for 30 min at 4°C. The supernatant was discarded, and the pellet was dissolved in 500 µL 1X NEB3.1 and transferred to a 0.5 mL AMICON Ultra Centrifuge filter (UFC5030BK EMD Millipore). The column was centrifuged for 5 min at 14,000 g and the flow-through was discarded. The column was washed 4 times using 450 µL of 1X NEB3.1 for desalting of DNA. After the final wash, the DNA remaining in the column (\sim 50 µL) was eluted in 70 µL of 1XNEB3.1; the column was flipped upside down into a new tube to collect DNA and centrifuged for 3 min at 4,000 g. ~70 µL of DNA was recovered. RNA was degraded by adding 1 µL of 10 mg/mL RNAase A and incubation for 30 min at 37°C. The amount of DNA was estimated by running an aliguot on a 1% Agarose gel along with a 1kb ladder (NEB#N3232s).

Blunting overhang: DNA ends were filled in by adding 60 μ L of fill-in master mix was added: [1xNEB2.1, 0.25 mM dCTP, 0.25 mM dGTP, 0.25 mM dTTP, 0.25 mM dATP (ThermoFisher#19524016), 50 U Klenow polymerase Polymerase I (NEB M0210L)]. Samples were incubated at 37°C in a Thermocycler for 75 mins. Next, the tubes were placed on ice immediately for 15 mins.

Gel Extraction for size-selection: a 1% gel was prepared. 25µL of loading dye (blue dark) was added to the 130µL DNA sample from the previous step. 25µL of 1kb DNA ladder was loaded in one well and all sample in the remaining wells. After elecrophoresis the DNA was isolated from the gel in three size intervals: less than 1kb, 1kb-3kb, and larger than 3kb.

DNA purification from agarose gel: The DNA was extracted from the agarose gel using GFX[™] PCR DNA and Gel Band Purification Kit (GE28-9034-70 Millipore Sigma). After DNA extraction the sample was washed five times with TLE buffer using a 1.5 Amicon column followed by elution with 50 µl TLE. Aliquots of each DNA samples was analyzed on a fragment analyzer.

Sonication: DNA from each size fraction was sonicated to a size of 100 - 300 bp using a Covaris instrument (Duty Cycle 10%, Intensity 5, Cycles per Burst 200, set Mode Frequency sweeping, continuous degassing, process time 60 sec, Number of cycles) for 4 cycles. The 130 µL of sonicated DNA was transferred to a 1.5 mL tube and 1XTLE was added to a total volume of 500 µL. DNA fragment size was determined by running 2 µL of DNA along with low molecular ladder (NEB) on a 2% agarose gel.

Size fractionation using AMpure XP: 500 μ L AMpure beads (Beckman Coulter A63881) were added to a 1.5 mL tube labeled as 1.1X. The tube was placed on the MPS for 5 min, and the supernatant was removed. Beads were resuspended in 150 μ L AMpure mixture in order to make 1.1X. 400 μ L of AMpure mixture was

added to 500 µL of sonicated DNA from the previous step and the tube was labeled 0.8X. The sample was vortexed and centrifuged briefly using a tabletop small centrifuge followed by incubation at RT for 10 min on a rocking platform. Then the tube was placed on the MPS for 5 min at RT. The 0.8X supernatants were collected and added to the 1.1X tube, the tube was briefly vortexed and centrifuged followed by incubation at RT for 10 min on a rocking platform. The tube was placed on the MPS for 5 min at RT, and the supernatant discarded. Beads in the 0.8X and 1.1X tubes were washed twice with 1 mL 70% ethanol, reclaiming beads against the MPS for 5 min. Beads on the MPS were then dried until ethanol had evaporated completely. Next, 51 µL of 1XTLE was added to the 0.8X and 1.1X tubes to elute DNA from the beads. Tubes were incubated at RT on a rocking platform for 10 min. The 0.8X and 1.1X tubes were placed on the MPS for 5 min. Finally, the supernatants were transferred to 1.7 mL tubes labeled 0.8X and 1.1X. The 1.1X sample contains DNA that ranges in size from 100-300 bp. The DNA in the 0.8X sample was kept in case more DNA was required, in which case the DNA would be sonicated using 2 cycles followed by a similar round of size fractionation as described above. The amount of DNA from both samples 0.8X and 1.1X was quantified by running 1 µL on a 2% agarose gel along with a titration of low molecular weight DNA ladder (100 ng, 200 ng, 400 ng).

End Repair: 50 μ L from the 1.1X sample was transferred to a PCR tube, and 20 μ L of end repair mix was added: [3.5X NEB ligation buffer (NEB B0202S), 0.875

mM dNTP mix, 0.375 U/µL T4 DNA polymerase, 1.25 U/µL T4 polynucleotide kinase, 0.125 U/µL Klenow DNA polymerase]. The 70 µl total volume reaction was incubated for 30 min at 20°C in a PCR machine and then placed on ice. The DNA was purified by 1:2 Ampure, by adding 140 µL 2X Ampure solution to the 70 µL DNA sample followed by incubation for 5 min at RT. The tube was placed on the MPS for 4 min to reclaim the beads and the supernatant was discarded. The beads were washed twice with 1 mL of 70% ethanol while on the MPS. After beads were dried DNA was eluted in 32 µL TLE (pH 8.0) and incubation for 10 min at RT. The supernatant was transferred to a 1.5 mL tube.

A-tailing: A dATP was added to the 3' ends by adding 18 μ L of A-tailing mix [5 μ L NEB buffer 3.1, 10 μ L of 1 mM dATP, 3 U Klenow exo (NEB M0212S)] to the 32 μ L of DNA sample from the previous step. The reaction was then incubated in a PCR machine at 37°C for 30 min followed by incubation 65°C for 20 min and cooling down to 4°C. The tube was placed on ice. The volume was brought to 100 μ L by adding 1X NEB2.1. The DNA was then purified by adding 1:2 Ampure mix (200 μ L of Ampure was added to the 100 μ L final DNA volume). Finally, the DNA was eluted in 40 μ L of 1X T4 DNA ligase buffer (Invitrogen 5X).

Illumina adapter ligation and paired-end PCR: For this step we used the TruSeq DNA LT kit Set A (REF#15041757). 50 μ L of ligation mix [25 μ L Illumina pairedend adapters, 15 μ L T4 DNA ligase Invitrogen, 10 μ L 5X T4 DNA ligase buffer (Invitrogen 5X)] was added to the 40 μ L sample from the previous step. The ligation sample was then incubated at RT for 2 hours on a rotator. Next, the DNA was purified by adding 1:1 Ampure solution (180 μ L of Ampure mix was added to 132 the 90 μ L sample), the supernatant was discarded and beads were washed twice with 1 mL of 70% ethanol. After the last wash step, the beads were resuspended in 400 μ L of 1X TLE and incubated at RT on a rocking platform for 10 mins. The tube was placed on the MPS for 4 mins. Finally, the 400 μ L supernatant was transferred to a new tube.

PCR optimization: The Illumina Truseq Kit (DNA LT kit Set A (REF#15041757)) was used for PCR amplification of DNA for DpnII-Seq. The trial PCR reaction was set up as follows: [2.5 µL DNA bound to beads, 2 µL of Primers mix (Truseq kit), 10 µL Master Mix (Truseq kit), 10.5 µL of ultrapure distilled water (Invitrogen)]. The 25 µL was split over four PCR tubes (5 µL/per tube). Each of the four samples was incubated for different PCR cycles (6, 8, 10, or 12 cycles): [30 sec at 98°C, n cycles of (30 sec at 98°C, 30 sec at 65°C, 30 sec at 72°C), 7 min at 72°C, hold at 10°C]. The optimal PCR cycle number needed to get enough DNA for sequencing was determined by running the 4 PCR reactions on a 2% agarose gel along with low molecular ladder titration (100 ng, 200 ng, 400 ng). Three PCR reactions of 50 µL volume were then performed: [5 µL DNA bound to beads, 4 µL of Primers mix (Truseq kit), 20 µL Master Mix (Truseq kit), 21 µL of ultrapure distilled water (Invitrogen)]. The 3 PCR reactions were pooled together to obtain 150 µL total volume. The samples were reclaimed against the MPS for 1 min, then the PCR products (supernatant) were taken to new 1.5 mL tubes. 1:1 Ampure was performed for removal of primer dimers (150 µL of Ampure and 150

 μ L DNA sample). Finally, beads were resuspended in 35 μ L of TLE to elute the DNA. DNA that remained bound to beads was saved after a first wash using TBW followed by two washes with 1X TLE and then resuspended in 30 µL of 1X TLE.

Lamin A Immunofluorescence and DAPI

For nuclei immunofluorescence, we prepared a coverslip by adding 1 mL of 0.1% Poly-L-lysine solution (Sigma SLBQ5716V) for 10 min, then coverslips were dried using Whatman papers. Each coverslip was transferred to a single well of an eight wells plate. The coverslips were washed twice using PBS. Next 500 µL of 30% sucrose with 1 mM DTT was added on top of the coverslips to protect nuclei from an abrupt contact with coverslip during spinning. 1 million control nuclei or nuclei after chromatin digestion were crosslinked for 20 min using a 4% final concentration of Paraformaldehyde immediately after predigestion. Next, nuclei were added slowly on top of he sucrose solutions on the coverslips and spun for 15 mins at 2,500 g at 4°C. Next, nuclei were assumed to be attached to the coverslips which were then transferred to a new 8 well plate. The coverslips were washed five times with 1% PBS. Next, non-specific binding of the primary antibody was blocked by adding 500 µL of the blocking buffer [3% BSA, 1X PBS, 0.1% Triton X-100 (Sigma 9002-93-1)] and incubating for 60 min at RT. Afterward, lamin A antibody (ab 26300) was diluted 1:1000 in blocking buffer, and the coverslip was incubated face-down on top of a 250 µL of lamin A antibody droplet that was placed on parafilm for 120 min at RT. Then, the

coverslip was placed back in the well of a new plate face-up and washed five times with washing buffer (1X PBS, 0.1% Triton X-100). The secondary antibody Goat Anti-Rabbit (ab150077) was diluted 1:1000 in blocking buffer, and the coverslip was incubated face-down on top of a 250 µL droplet of the secondary antibody (Goat Abti-Rabbit (ab150077) that was placed on parafilm for 60 min at RT. Next, the coverslip was placed back in the well of a new plate face-up and washed five times with washing buffer (1X PBS, 0.1% Triton X-100) and twice with 1X PBS. The slide was mounted and sealed using 10 µL antifade mountant with DAPI (Invitrogen P36931).

For image acquisition, we used a Nikon Eclipse Ti microscope. Imaging was performed using an Apo TIRF, N.A. 1.49, 60X oil immersion objective (Nikon), and a Zyla sCMOS camera (Andor). Z-series of 0.2 μm slices were acquired using Nikon Elements software (Version 4.4).

Chromatin fractionation assay

Chromatin-bound proteins were isolated and separated from free proteins. A sample of 2 million control nuclei or pre-digested nuclei (obtained as described above "Pre-digestion of nuclei") was centrifuged at 5,000 g for 5 min at 4°C. The supernatant was transferred to an Amicon column to reduce the volume from 500 μ L to 100 μ L by centrifugation for 4 min at 14,000 g. This sample contains the free protein fraction. Next, 26 μ L of glycerol and 1.3 μ L of 100X protease inhibitor cocktail were added to the 100 μ L free proteins sample. The pellet containing the nuclei was resuspended in 100 μ L of nuclei purification buffer with Triton (10 mM

PIPES pH 7.4, 10 mM KCl, 2 mM MgCl2, 0.25% Triton, 1% Protease inhibitor, 1mM DTT) and incubated for 10 min on ice. Then, in order to protect protein structure during sonication, 25 µL of glycerol was added to the 100 µL pellet sample to have 20% final glycerol concentration. The sample was sonicated using a Covaris instrument at 4°C as follows: (Duty Cycle 10%, Intensity 5, Cycles per Burst 200, set Mode Frequency sweeping, continuous degassing, process time 60 sec, 4 cycles). The pellet sample contains chromatin-bound proteins, was transferred to a 1.5 mL tube. All samples were stored at -20. These samples contain the protein bound CTCF and cohesin. Note: when these samples were centrifuged after the triton solubilization, we found that no SMC3 or CTCF could be detected in the supernatant. These results indicate that nonchromatin-bound proteins exit the nuclei and were recovered in the supernatant prior to triton solubilization step.

For analysis of CTCF and SMC1 chromatin binding: 15 μ L from each protein sample (supernatant or pellet) was mixed with 5 μ L of 5X Lane Marker Reducing Sample Buffer (Thermo Fisher 39000), then the mix was boiled for 10 min. The samples were cooled down to RT before loading them on a 3-8% Tris-Acetate Protein Gels (Invitrogen EA0375PK2). Next, the gel was run in 1X Tris-Acetate SDS Running Buffer (Invitrogen LA0041) for 75 min at 150V. For Histone H3: 1 μ L of protein sample was mixed with 14 μ L of PBS containing 1% Protease inhibitor, 5 μ L of 5X Lane Marker Reducing Sample Buffer was added to the mix and boiled for 10 min. The samples were cooled down to RT before loading them

in Tris-Base 4-12% (Invitrogen NP0322BOX), then the gel was run in 1X MES-136 SDS running buffer (Invitrogen B0002) for 60 min at 150V. The proteins were transferred from the gel to nitrocellulose membrane using 1X western blot transfer buffer (Thermo science 35040). The transfer was 120 min for SMC1 and CTCF and 75 min for H3. The nitrocellulose membranes were washed using 1X TBST [50 mM Tris-Cl, pH 7.6; 150 mM NaCl, 0.1 mL of Tween 20], then Blocked for 120 min using 5% milk (1 g milk in 20 mL 1X TBST). The membrane when then incubated overnight at 4°C with primary antibody diluted in 5% milk [1:1000 CTCF antibody cell signaling (activeMotif 61311), 1:2000 SMC1 (Bethyl Antibody, A300-055A), 1:4000 H3 Abcam (ab1791)]. Next, the membranes were washed 6 times for 10 min per wash using 1X TBST. The secondary antibody anti-rabbit IgG HRP from cell signaling was diluted using 5% milk for CTCF and SMC1 [1:1000 for CTCF, 1:2000 SMC1] and in 1% milk for H3 1:5000 dilution. Membranes were incubated for 120 min at RT. Finally, membranes were washed 6 times for 10 min using 1X TBST. Finally, the membranes were developed using luminol-based enhanced chemiluminescence(Thermo science 34076).

Micromanipulation force measurement and treatments of an isolated nuclei

Micromanipulation force measurements were conducted as described previously in Stephens et al. (Stephens *et al.*, 2017). K562 cells were grown in microscope slide wells and treated with 1 μ g/mL latrunculin A (Enzo Life Sciences) for ~45 min before single nucleus isolation. The nucleus was isolated by using small amounts of detergent (0.05% Triton X-100 in PBS) locally sprayed onto a living cell via an "isolation" micropipette. This gentle lysis allows the use of 137 a second micropipette to retrieve the nucleus from the cell, using slight aspiration and non-specific adherence to the inside of the micropipette. A third micropipette was then attached to the opposite end of the nucleus in a similar fashion. This last "force" micropipette was pre-calibrated for its deflection spring constant, which is on the order of 2 nN/µm. A custom computer program written in LabView was then run to move the "pull" micropipette and track the position of both the "pull" and "force" pipettes. The "pull" pipette was instructed to move 5 µm at 45 nm/sec. The program then tracked the distance between the pipettes to provide a measure of nucleus extension ~3 µm. Tracking the distance that the "force" pipette moved/deflected multiplied by the pre-measured spring constant provides a calculation of force exerted. Calculations were done in Excel (Microsoft) to produce a force-extension plot from which the best-fit slope of the line would provide a spring constant of the nucleus (nN/µm). Isolated nuclei were measured twice initially to establish the native spring constant prior to treatment. After 50 uL of buffer only (control), 100 units DpnII (GATC) with NEB buffer 3.1, or 100 units HindIII (A|AGCTT) with NEB buffer 2.1 was added to the 1.5 mL imaging well and mixed gently. Force measurements were performed 5 min, 30 min, and 60 min post-treatment.

3C-PCR

The human β-globin locus is an ideal region to examine looping interactions between enhancers and genes because of the strong looping

interactions between the LCR and HBG globin gene in the erythroleukemia cell 138 line K562, which highly expresses the globin genes (Dostie et al., 2006). 3C libraries were generated from: (1) K562 cells that have an LCR-HBG interaction, (2) GM12878 cells in which the LCR-HBG looping interaction is absent, and (3) beta-globin BAC (ENm009) control to normalize for primer bias. To investigate the interaction between the LCR and HBG gene, 3C primers from (Dostie et al., 2006) were used. 16 forward primers of 28-33 bp length were designed 40-60 bp upstream of each EcoRI site throughout a 110 kb region around the Beta Globin locus (chr11: 5221788- 5337325). The EcoRI fragment overlapping with the LCR (HS3,4,5) was used as an anchor to detect the interaction frequencies between the LCR and EcoRI fragments throughout the β -globin locus. For each primer pair, triplicate PCR reactions were set up, and the mean of the three was normalized to the BAC signal for the same primer pair before plotting normalized interaction frequency in the y-axis, the distance from EcoRI fragment overlapping with LCR to neighboring EcoRI fragments is plotted in the x-axis. Error bars are the standard error of the mean (SEM).

5C data processing

The fastq files for 5C sequencing data were processed as described in https://github.com/dekkerlab/5C-CBFb-SMMHC-

Inhib/blob/master/data_processing_steps.md

The Fastq files were mapped using novoalign to a reference genome built from the pool of all 277 probes. After mapping, we combined the read-pairs. The results were then transferred to a matrix format, and interactions were filtered as 139 previously described (Lajoie *et al.*, 2009; Sanyal *et al.*, 2012). First, interactions that belong to the same EcoRI fragment were removed. Second, outliers that are overrepresented as a result of overamplification were also removed. Outliers were defined as the interactions with a Z-score greater than 20 in all datasets. Third, probes that strongly over or underperform which leads to strongly enriched or depleted interactions in a whole row of interactions, were also removed. The four matrices were then scaled to the same number of total reads. Finally, data were binned at 20 Kb (median) with a sliding window with 2.5 Kb steps

Hi-C data processing

Hi-C read mapping, filtering, binning and matrix normalization were performed using the cMapping pipeline available at https://github.com/dekkerlab/cMapping (Lajoie, Dekker and Kaplan, 2015). In brief, Hi-C reads were mapped to reference human genome assembly hg19 using an iterative mapping strategy and Bowtie 2 (Langmead *et al.*, 2009). Successfully mapped reads were then filtered to remove reads mapping to the same restriction fragment and to remove PCR duplicates. Interaction frequency versus distance plots displayed high variance for interactions below 1 kb for all samples. Hence, after mapping of valid pair, we removed all pairs with a genomic distance less than 1 kb. The remaining valid read pairs were then binned to 500 kb, 40 kb, and 10 kb resolution matrices. Outlier bins of these matrices with low signal were assigned values of NA. Then as a bias correction step, matrices were normalized such that the sum of interactions in each row/column are approximately equivalent via an iterative correction procedure (ICE) (Imakaev *et al.*, 2012). Lastly, for comparison between samples, matrices were scaled such that the total interactions for a genome-wide matrix equals one billion for each sample. These ICEd scaled matrices were used for subsequent analyses.

A/B compartments

All reads from Hi-C in control K562 samples were pooled to identify A (active) and B (inactive) compartments in K562 cells. A/B compartments were identified at 40 kb resolution following the procedure described in (Lieberman-Aiden et al., 2009) using matrix2compartment.pl in https://github.com/dekkerlab/cworld-dekker. Briefly, each cis interaction matrix was first transformed into a z-score matrix followed by transformation into a correlation matrix. PCA was performed on the correlation matrix and the first eigenvector (PC1) of the PCA analysis was used to identify compartments for each chromosome. A/B compartments were assigned based on gene density such that the A-compartment was more gene-dense than the B-compartment. Positive PC1 values indicate gene-rich A compartments and negative PC1 values indicate gene-poor B compartments. For chromosome 9 the compartments were called for each chromosome arm separately as PC1 captured preferences for interactions within the same arm as opposed to canonical compartment preferences.

140

LOS and half-life calculation

To measure the 3D structure changes resulting from DpnII, HindIII, or Fatl pre-digestion we quantified the amount of cis interactions lost or gained in a 6 Mb window centered at every 40 kb bin genome wide. We note that we did not observe detectable amounts of DNA in the supernatant after chromatin fractionation indicating the large majority of liquefied DNA remains within the nuclei. Even if some DNA is lost, ICE balancing of Hi-C matrices ensures any biases in sequence coverage are removed. For each 40 kb bin, the percent of interactions occurring within its 6 Mb window (corresponding to interactions less than or equal to 3 Mb in distance either upstream or downstream from 40 kb bin) out of total interactions for the 40 kb bin (cis and trans) was calculated. These 6 Mb cis percentages were calculated for control, DpnII pre-digested, HindIII-pre-digested nuclei, and Fatl pre-digested nuclei. The change in 3D structure relative to control using these cis percentages was given by the following loss of structure (LOS) metric:

$$LOS = \frac{Control_{Cis\%} - Predigest_{Cis\%}}{Control_{Cis\%}}$$

Hence, LOS values in the range (0, 1) represent a loss in short range contacts after pre-digestion; LOS values < 0 represent an increase in short range contacts after pre-digest, and an LOS equal to zero would indicate no change in structure after pre-digestion. A window of 6 Mb was chosen as we sought here to quantify interactions disrupted by pre-digestion. Many longer range interactions increased after pre-digestion, potentially due to random ligations of cut fragments 142 that start to mix. Difference noted in A and B stability was preserved when LOS was calculated using cis percentages for entire chromosomes as opposed to a 6 Mb window, however the size of chromosomes did bias results by giving 40 kb bins in small chromosomes greater LOS. We note that any loci that may have been lost from the nuclei will not be included in the Hi-C dataset. LOS represents the relative redistribution of short-range interactions to longer-range and interchromosomal interactions for the set of loci that remained contained within the nucleus after pre-digestion and we assume this re-distribution would not be affected by any lost loci. Fatl-pre digested libraries were of lower sequencing coverage and hence had a lower signal to noise ratio compared with DpnII and HindIII-pre digested libraries. To reduce noise, we applied a loess based smoothing with an α smoothing parameter of 0.01 to the signal track of LOS for nuclei pre-digested using Fatl. Correlations between Fatl LOS and PC1 were evident both before and after smoothing. Correlations also remained evident before and after smoothing between Fatl LOS residuals and PC1 residuals corrected for digestion efficiency by Fatl-seq.

To quantify the timing of disrupted interactions we generated a half-life track utilizing the Hi-C matrices from the DpnII timecourses. For each 40 kb bin we fit a curve to the LOS of each timepoint following an exponential decay of the form (Figure 2.10, C):

$$LOS = a - (b \times e^{-c \times minutes})$$

such that a,b and c are parameters to fit. The half-life, t_{1/2}, was defined as the time required to reach half saturation, saturation being the 16 hour timepoint where maximal cis interactions have been lost. Half-life values were then computed for every 40 kb bin genome wide. To remove noisy and less reliable t_{1/2} data, we first removed all extreme outliers bins where the sum of squared residuals (SSR) for the exponential fit was greater than 0.1. Then all bins with an SSR greater than two standard deviations from the mean were deemed as outliers and also removed from analyses.

As LOS and t_{1/2} are both dependent on digestion efficiency we also generated residual LOS and t_{1/2} tracks to account for bin to bin variation in digestion efficiency. We used a moving average approach to calculate residuals for LOS as a function of DpnII-seq signal and also t_{1/2} as a function of DpnII-seq signal since the relationships between these variables were non-linear (Figure 2.5, F left, Figure 2.9, C). For both stability metrics LOS and t_{1/2}, a sliding window of 200 DpnII-seq signal with a step size of one was used to calculate mean LOS or t_{1/2} signal for each DpnII-seq signal increment (Figure 2.5, F left, Figure 2.9, C). Window and step size were selected by manual inspection of moving averages and compromising between over and underfitting. These moving averages were used to calculate residuals such that a positive LOS residual indicates less structure loss than expected. As t_{1/2} is inversely related to LOS, positive t_{1/2} residuals indicate less structure loss than

143

expected and negative t_{1/2} residuals indicate more structure loss than expected. 144 Moving averages were also used to generate residuals for DpnII-seq as a function of PC1 and LOS as a function of PC1 (Figure 2.5, G right).

Similar to the digestion efficiency correction by DpnII-seq, we also used estimated average fragment size as an independent measure to correct LOS and t_{1/2} for biases in digestion efficiency (Figure 2.7). For both stability metrics LOS and t_{1/2}, a sliding window of 200 bp with a step size of one was used to calculate mean LOS or t_{1/2} signal for each average fragment size bp increment (Figure 2.7, G). These moving averages were then used to calculate LOS residuals or t_{1/2} residuals as in the DpnII-seq correction approach described previously.

Dpnll-seq data analysis

Sequenced reads were mapped to the hg19 genome using the Bowtie read aligner (Langmead *et al.*, 2009) and reads mapping to multiple sites of the genome were removed. As expected, a high percentage of reads mapped precisely to their associated restriction cut site (Figure 2.6). To remove potential artificial biases, we filtered out paired-end reads from fragments whose start or end coordinate was more than three nucleotides from an appropriate restriction cut site. Filtered reads were then binned to 500 kb or 40 kb resolutions. The K562 cell line has a primarily triploid karyotype with regions of the genome in diploid and tetraploid states. Copy number state assignments for each 500 kb or 40 kb bin were assigned using publicly available K562 copy number data from the Catalogue of Somatic Mutations In Cancer (COSMIC) database (https://cancer.sanger.ac.uk/cell_lines/download). Copy number segments in the 145 COSMIC dataset were identified by PICNIC analysis of Affymetrix SNP6.0 array data (PMID:19837654). Read coverage files at 500 kb and 40 kb were corrected to a genome wide diploid state using the copy number state assignments and dividing coverage by appropriate correction value (diploid = 1, triploid =1.5, tetraploid = 2, etc.) per bin. (Figure 2.6, D and E). Final copy number corrected coverage files were used for all downstream analysis. DpnII-seq computational workflow is maintained at <u>https://github.com/tborrman/DpnII-seq</u>

Fatl-seq data analysis

Computational workflow for FatI-seq analysis was identical to previously described DpnII-seq analysis, with the exception that FatI restriction sites were used in the filtering step as opposed to DpnII restriction sites. The DpnII-seq workflow maintained at https://github.com/tborrman/DpnII-seq has options for analyzing restriction enzyme-seq experiments using the following enzymes: DpnII, HindIII, and FatI.

DpnII pre-digestion average fragment size analysis

Pre-digestion by DpnII leads to variable DNA fragment sizes across the genome. To estimate the average fragment size for a genomic bin after a 4-hour DpnII pre-digestion, we first separated 4-hour DpnII pre-digestion DNA into three slices: less than 1kb, 1kb-3kb, and larger than 3kb (See previously described above: DpnII Pre-digestion size assessment, Figure 2.7 A and B). DNA fragments purified from these slices were sequenced and sequenced read pairs were mapped to the hg19 genome using the Bowtie read aligner (Langmead *et* 146 *al.*, 2009). Mapped reads were then binned to 40 kb resolution, normalized for sequencing depth, and corrected for copy number state as in the DpnII-seq workflow (See previously described above: DpnII-seq data analysis). This resulted in a coverage track for each of the three DpnII pre-digested slices: less than 1 kb, 1 kb – 3 kb, and larger than 3kb (Figure 2.7, C).

To estimate average fragment size for a given genomic bin we used the following formula:

Average fragment size =
$$\frac{p_1q_1s_1 + p_2q_2s_2 + p_3q_3s_3}{p_1q_1 + p_2q_2 + p_3q_3}$$

such that px equals the percent of normalized slice x reads that mapped to bin, qx equals quantity of slice x fragments (ng/µL), and sx equals mean size of fragments from slice x (bp). The variable x represents one of the three slice intervals (1: less than 1 kb, 2: 1 kb - 3 kb, and 3: larger than 3 kb). Hence, the average fragment size for a given bin estimates the quantity of fragments from each slice size mapping to the bin over the total quantity of fragments mapped to the bin. The values for px are extracted from our coverage tracks and vary bin to bin, while the values for qx and sx are extracted from the Fragment Analyzer analysis and are constants (s₁ = 643 bp, s₂ = 2332 bp, s₃ = 5495 bp, q₁ = 1.6562 ng/µL, q₂ = 2.544 ng/µL, q₃ = 2.4632 ng/µL, Figure 2.7, B). The average fragment size track was then used as an independent metric for measuring 4-hour DpnII digestion efficiency as compared to the DpnII-seq signal track.

Subcompartments

Rao et al. (2014) divided the canonical A/B compartments into five primary subcompartments A1, A2, B1, B2, B3 based on each subcompartment's preferential Hi-C interactions in GM12878 cells. Subcompartments were annotated using high resolution (~1 kb) Hi-C data and were shown to display unique genomic and epigenomic profiles. K562 subcompartments were annotated in (Xiong and Ma, 2019) via the method SNIPER using lower resolution Hi-C data. In short, SNIPER infers subcompartments via a neural network approach to accurately annotate subcompartments using Hi-C datasets with moderate coverage (~500 million mapped read pairs). Xiong et al.'s K562 SNIPER subcompartments showed a substantial conservation with GM12878 annotations from Rao et al. (Rao et al., 2014) and were also enriched in similar epigenetic features, hence we utilized these SNIPER annotations to compare subcompartment status with chromatin stability. K562 SNIPER subcompartments were annotated at 100 kb resolution. To compare with our 40kb resolution liquid chromatin Hi-C data, we binned the 100 kb subcompartment annotations to 40kb such that any 40 kb bin overlapping a boundary of two separate subcompartments was assigned a value of NA. Upon piling up K562 subcompartment boundaries, we also found enrichment and depletion of various chromatin features consistent with those described in both Rao et al. (Rao et al., 2014; Xiong and Ma, 2019).

Sub-nuclear structures

To assess the effect of sub-nuclear structures on chromatin stability we utilized the extensive genetic and epigenetic data publicly available for K562 cells (Table 2.1).

Fold change over control ChIP-seq tracks for histone modifications, chromatin remodellers, and other various proteins were downloaded from the ENCODE Portal. To compare ChIP-seq data with t_{1/2}, or residuals of t_{1/2} after correction for DpnII-signal, we binned the ChIP-seq signal tracks into 40 kb such that each 40 kb bin represented the mean signal found across the bin. Bins with no overlapping signal were designated a value of NA.

To examine the association between methylation state and t_{1/2} or residuals of t_{1/2} after correction for DpnII-signal, we downloaded methylation state at CpG Whole-Genome Bisulfite Sequencing (WGBS) tracks from ENCODE. As the methylation data was mapped to hg38, we used the UCSC LiftOver program to convert coordinates to hg19. Then percentage methylation at CpG sites was binned to 40kb resolution using the mean.

As there is currently no nucleolus associated domains (NADs) data available for K562, we analyzed a binary NADs state track for the human embryonic fibroblast IMR90 cell line (Dillinger, Straub and Nemeth, 2017). Dillinger et al. annotated NADs via a two-state hidden Markov model of aCGH data from DNA of isolated nucleoli. Using these annotated NADs, coverage of each 40 kb bin for NADs was assessed and used for all our downstream analyses.

Mapping of nuclear speckle, nuclear lamina and PolII associated loci for K562 cells was accomplished recently via the TSA-seq protocol (Chen *et al.*, 2018). Signal tracks of log2(pull-down/input) were downloaded from GEO and binned to 40 kb as previously described for ChIP-seq files. Microarray data for LaminB1 associated domains identified through the DamID protocol was also available from that study. We used the UCSC LiftOver program to convert coordinates from hg18 to hg19. We then binned the log2(Dam-LaminB1/Dam) signal to 40 kb bins as previously described for ChIP-seq files.

To analyze cell cycle relationship with chromatin stability we downloaded Repli-seq data for K562 cells from ENCODE. Actively replicating regions are quantified as a percentage normalized signal for FACS sorted cells in G1 phase, four stages of S phase (S1-S4) and G2 phase. Signal tracks for Repli-seq data were binned to 40 kb as previously described for ChIP-seq files.

Binning of data was performed using the bedtools/v2.26.0 software. To assess the quality of the publicly downloaded data we generated the spearman correlation matrix of all binned signal tracks (Figure 2.13, A). Hierarchical clustering of rows of the correlation matrix position heterochromatic marks (H3K9me3, HP1 α , HP1 β , NADs, and LADs) near one another as expected. The majority active marks form a larger cluster, with the markers for polycomb regions (H3K27me3, CBX8, BMI1, RNF2, and SUZ12) representing facultative 150 heterochromatin clustered together segregating active from inactive marks.

Gene Expression

To assess the effect of gene expression on chromatin stability we utilized processed gene expression quantifications of total RNA-seq for K562 cells available from ENCODE (Accession ID: ENCFF782PCD). Gene locations were mapped using the hg19 ensGene table from UCSC Table Browser. To compare expression values with 40 kb resolution $t_{1/2}$ or residuals of $t_{1/2}$ after correction for DpnII-signal tracks, fragments per kilobase million (FPKM) values for each gene were binned to 40 kb such that each 40 kb bin represented the mean FPKM for all genes overlapping that bin. Bins without any genes were assigned a value of NA. Binned FPKM >=1 was determined to be a reasonable cutoff for expression by inspection of the full distribution of FPKM values.

Compartmentalization saddle plots

Saddle plot calculations were performed using tools in the cooltools repository: (https://github.com/hms-dbmi/hic-data-analysisbootcamp/tree/master/notebooks/04 analysis cooltools-eigenvectorsaddle.ipynb).

To measure the strength of compartments, intra-chromosomal interaction frequencies were first normalized by the average interaction frequency at a given genomic distance (observed/expected Hi-C maps) at a resolution of 40 kb. Then

the distance corrected interaction frequencies were sorted based on PC1 values 151 of a pair of bins that define a given interaction. Finally, sorted frequencies were aggregated into 50 by 50 groups according to their PC1 values and averaged to obtain a compartmentalization saddle plot. In a compartmentalization saddle plot, preferential B-B interactions are in the upper left corner, and preferential A-A interactions are in the lower right corner.

Homotypic interaction saddle plots

Intra-chromosomal interactions frequencies between 40 kb bins were normalized by the average interaction frequency at a given genomic distance (observed/expected Hi-C maps). Then, the distance corrected interaction frequencies were sorted based on signal values (TSA-seq, DamID) of a pair of bins that define a given interaction, for a given factor (SON, Lamin). Finally, sorted frequencies were aggregated into 50 by 50 groups according to their signal values and averaged, to obtain homotypic interaction saddle plots. In these plots, pair-wise interactions between loci enriched in factor binding are shown in the lower right corner, and pair-wise interactions between loci not bound by the factor are shown in the upper left corner.

Scaling plot

The script to generate scaling plots was adapted from cooltools (<u>https://github.com/mirnylab/cooltools/tree/master/cooltools</u>). Genome-wide curves of normalized contact frequency P(s) is plotted as a function of genomic

distance for all intra-chromosomal interactions. Each library was normalized by 152 total valid interactions

Mean z-score heatmap

Each genome wide 40kb signal vector for a sub-nuclear structure was cleaned for outliers above three standard deviations of the vector's mean. Each cleaned vector was z-score transformed and then partitioned based on the different $t_{1/2}$ residual intervals for associated bins. The mean z-score for all bins within a given $t_{1/2}$ residual interval is plotted as a square in the heatmap.

CHAPTER III: NEURON-SPECIFIC SIGNATURES IN THE CHROMOSOMAL CONNECTOME ASSOCIATED WITH SCHIZOPHRENIA RISK

Preface

This chapter comprises work published in Science by Prashanth Rajarajan, myself, Will Liao, Nadine Schrode, Erin Flaherty, Charlize Casiño, Samuel Powell, Chittampalli Yashaswini, Elizabeth A. LaMarca, Bibi Kassim, Behnam Javidfar, Sergio Espeso-Gil, Aiqun Li, Hyejung Won, Daniel H. Geschwind, Seok-Man Ho, Matthew MacDonald, Gabriel E. Hoffman, Panos Roussos, Bin Zhang, Chang-Gyu Hahn, Zhiping Weng, Kristen J. Brennand, and Schahram Akbarian. The publication reference is "Neuron-specific signatures in the chromosomal connectome associated with schizophrenia risk" *Science*. Vol. 362 Issue 6420 Dec. 2018 (Rajarajan *et al.*, 2018)

Cell culture work including Hi-C, 3C, RNA-seq, ATAC-seq, Cas9, and dCas9 (epi)genome editing was performed by Prashanth Rajarajan, Charlize Casiño, Chittampalli Yashaswini, Bibi Kassim, Behnam Javidfar, Samuel Powell, Elizabeth A. LaMarca, Bin Zhang, Seok-Man Ho, and Aiqun Li. Biocomputing, informatics, and genomic analyses was performed by myself, Will Liao, Nadine Schrode, Sergio Espeso-Gil, Erin Flaherty, Gabriel E. Hoffman., and Zhiping Weng. Specifically, I computationally processed Hi-C sequencing datasets, performed loop calling, performed loop analysis, developed differential loop calling method, performed gene ontology analyses, performed downstream RNA-154 seq analyses, and performed coregulation analyses. Materials were contributed by Seok-Man Ho, Erin Flaherty, Hyejung Won, and Daniel H. Geschwind. Research was supervised by Zhiping Weng, Panos Roussos, Kristen J. Brennand and Schahram Akbarian. Experiments were conceived and designed by Prashanth Rajarajan, Kristen J. Brennand and Schahram Akbarian. The paper was written and figures were produced by Prashanth Rajarajan, myself, Will Liao, Kristen J. Brennand, and Schahram Akbarian with contributions from all coauthors.

Abstract

To explore the developmental reorganization of the three-dimensional genome of the brain in the context of neuropsychiatric disease, we monitored chromosomal conformations in differentiating neural progenitor cells. Neuronal and glial differentiation was associated with widespread developmental remodeling of the chromosomal contact map and included interactions anchored in common variant sequences that confer heritable risk for schizophrenia. We describe cell type–specific chromosomal connectomes composed of schizophrenia risk variants and their distal targets, which altogether show enrichment for genes that regulate neuronal connectivity and chromatin remodeling, and evidence for coordinated transcriptional regulation and proteomic interaction of the participating genes. Developmentally regulated chromosomal conformation changes at schizophrenia-relevant sequences

disproportionally occurred in neurons, highlighting the existence of cell type-155 specific disease risk vulnerabilities in spatial genome organization.

Introduction

Spatial genome organization is highly regulated and critically important for normal brain development and function (Rajarajan et al., 2016). Many of the risk variants contributing to the heritability of complex genetic psychiatric disorders are located in noncoding sequences (Ripke et al., 2014), presumably embedded in "three-dimensional genome" (3DG) structures important for transcriptional regulation, such as chromosomal loop formations that bypass linear genome on a kilobase (or megabase) scale and topologically associated domains (TADs) (Dixon et al., 2012; Nora et al., 2012) that assemble in nested fashion across hundreds of kilobases (Bharadwaj et al., 2013, 2014; Won et al., 2016; Jiang et al., 2017). By linking noncoding schizophrenia associated genetic variants with distal gene targets, 3DG mapping with Hi-C (Lieberman-Aiden et al., 2009; Rao et al., 2014) and other genome-scale approaches could inform how higher-order chromatin organization affects genetic risk for psychiatric disease. To date, only a very limited number of Hi-C datasets exist for the human brain: two generated from bulk tissue of developing forebrain structures (Won et al., 2016) and adult brain (Schmitt et al., 2016) and one from neural stem cells (Dixon et al., 2015). Although such datasets have advanced our understanding of the genetic risk architecture of psychiatric disease (Won et al., 2016; de la Torre-Ubieta et al., 2018), 3DG mapping from postmortem tissue lacks cell type-specific resolution

and may not capture higher-order chromatin structures sensitive to the autolytic 156 process (Mitchell *et al.*, 2014). We monitored developmentally regulated changes in chromosomal conformations during the course of isogenic neuronal and glial differentiation, describing large-scale pruning of chromosomal contacts during the transition from neural progenitor cells (NPCs) to neurons. Furthermore, we uncovered an expanded

3DG risk space for schizophrenia—with a functional network of disease-relevant regulators of neuronal connectivity, synaptic signaling, and chromatin remodeling—and demonstrate neural cell type–specific coordination at the level of the chromosomal connectome, transcriptome, and proteome.

Results

Neural progenitor differentiation is associated with dynamic 3DG remodeling

We applied in situ Hi-C (Rao *et al.*, 2014) to map the 3DG of two male human induced pluripotent stem cell (hiPSC)–derived neural progenitor cells (NPCs) (Topol *et al.*, 2016), together with isogenic populations of induced excitatory neurons ("neuron") generated through viral overexpression of the transcription factor *NGN2* (Ho *et al.*, 2016) and differentiations of astrocyte-like glial cells ("glia") (Figure 3.1, A and B, and Table 3.1) (TCW *et al.*, 2017). Transcriptome RNA sequencing (RNA-seq) comparison with published datasets (Hoffman *et al.*, 2017) confirmed that the NPCs, but not glia, from subjects S1 and S2 clustered together with NPCs from independent donors, whereas S1 and S2 NGN2 neurons closely aligned with directed differentiation forebrain neurons 157 (Brennand *et al.*, 2011) and prenatal brain datasets (Figure 3.2, A and B). As with our transcriptomic datasets, hierarchical clustering of our Hi-C datasets after initial processing (Figure 3.3, A) also showed clear separation by cell type (Figure 3.1 A and Figure 3.3, B). Genome-scale interaction matrices were enriched for intrachromosomal conformations (Figure 3.3, C), with the exception of the negative control ("No Ligase") NPC library, in which we omitted the ligase step (Materials and methods) and observed an interaction map with no signal due to the loss of ligated chimeric fragments (Figure 3.3, D). Given the observed correlation between technical replicates of Hi-C assays from the same donor and cell type, and the correlation between cell type–specific Hi-C from the two donors (*R*technical replicates, range = 0.970 to 0.979; *R*subject1-subject 2 by cell type, range = 0.962 to 0.970), we pooled by cell type for subsequent analyses (Figure 3.3, E).

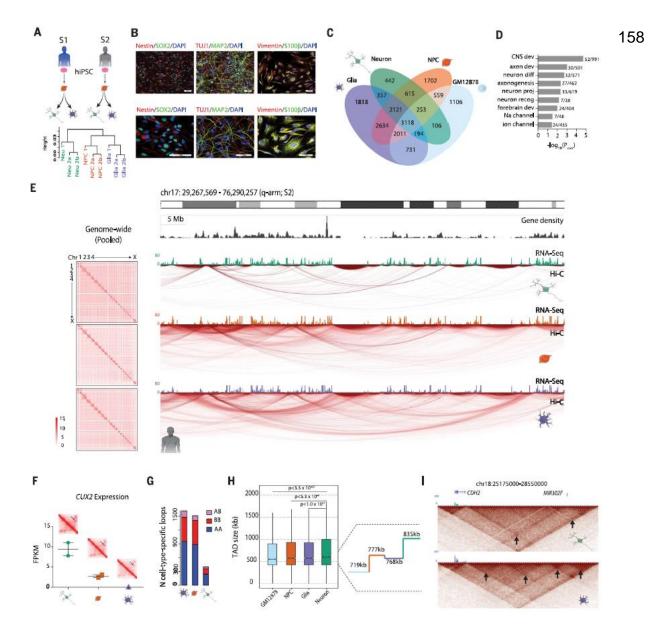
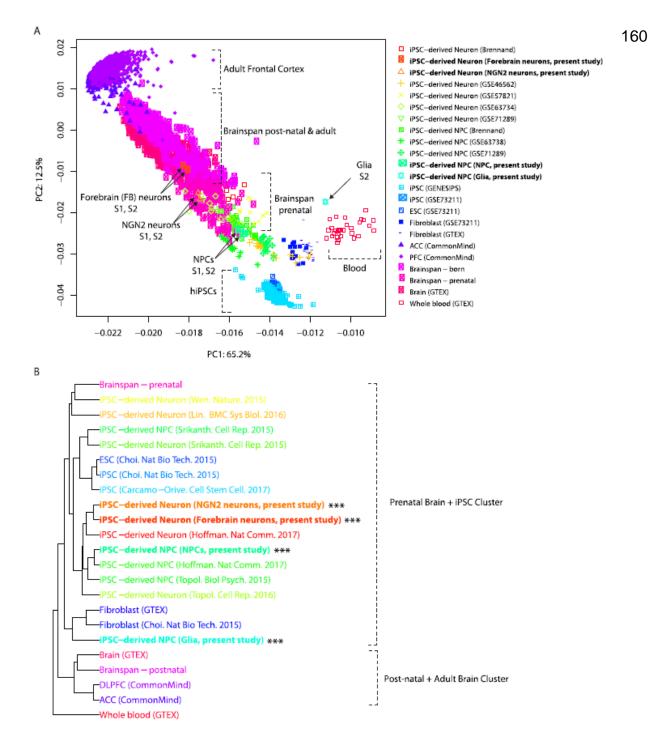


Figure 3.1 | Neural differentiation is associated with large-scale remodeling of the 3D genome.

(A) (Top) Derivation scheme of isogenic cell types from two male control cell lines. Pink oval, donor hiPSC; orange, NPC; green, neuron; purple, glia. (Bottom) Hierarchical clustering of intrachromosomal interactions (see Materials and methods) from six in situ Hi-C libraries. a and b are technical replicates of the same library; height corresponds to the distance between libraries (see Materials and methods) (Figure 3.3, B). (B) Immunofluorescent staining of characteristic cell markers for NPCs (Nestin and SOX2), neurons (TUJ1 and MAP2), and glia (Vimentin and S100 β). (C) Venn diagram of loop calls specific to and shared by different subsets of cells, including previously published GM12878 lymphoblastoid Hi-C data. (D) Gene ontology (GO) enrichment (significant terms 159 only) of genes overlapping anchors of loops shared by NPCs, neurons, and glia but absent in GM12878. (E) (Left) Cell-type pooled whole-genome heatmaps at 500-kb resolution (Figure 3.3, C). (Right) "Arc map" showing intrachromosomal interactions at 40-kb resolution of the q-arm of chr17 for isogenic neurons, NPCs, and glia, as indicated, from subject 2. RNA-seq tracks for each cell type shown on top of arc maps. Green, neuron; orange, NPC; purple, glia. (F) FPKM gene expression of CUX2 across three cell types with heatmap zoomed in on CUX2 loop (black arrow) (Figure 3.4). (G) Number of loops specific to each cell type (not shared with other cell types) with one anchor in an A compartment and another in a B compartment (pink), both in B compartments (red), or both in A compartments (blue). (H) (Left) Box and whisker distribution plot of TAD size across four cell types. (Right) Mean TAD length for each of the four cell types. (I) Heatmaps at 40-kb resolution for a 3-Mb window at the CDH2 locus on chr18. (Bottom) Nested TAD landscape in glia with multiple subTADs (black arrows) called, which (top) is absent from neuronal Hi-C. RNA-seq tracks: green, neuron; purple, glia (Figures 3.2 to 3.6).



3.2 | Cell- and tissue-specific gene expression profiles.

(A) Multidimensional scaling with samples colored as indicated in cell- and tissue-specific manner. Note that neuronal cultures and NPC from subjects S1 and S2 cluster together with fetal and perinatal brain tissue, intermingled in related cell types from other donors. (B) Pairwise distance matrix was computed

for all samples, and the median distance between all samples in each category 161 were used to create a summary distance matrix in order to perform the final clustering. Note that samples from the present study (marked by ***) align with independently generated datasets for same cell types from different donors.

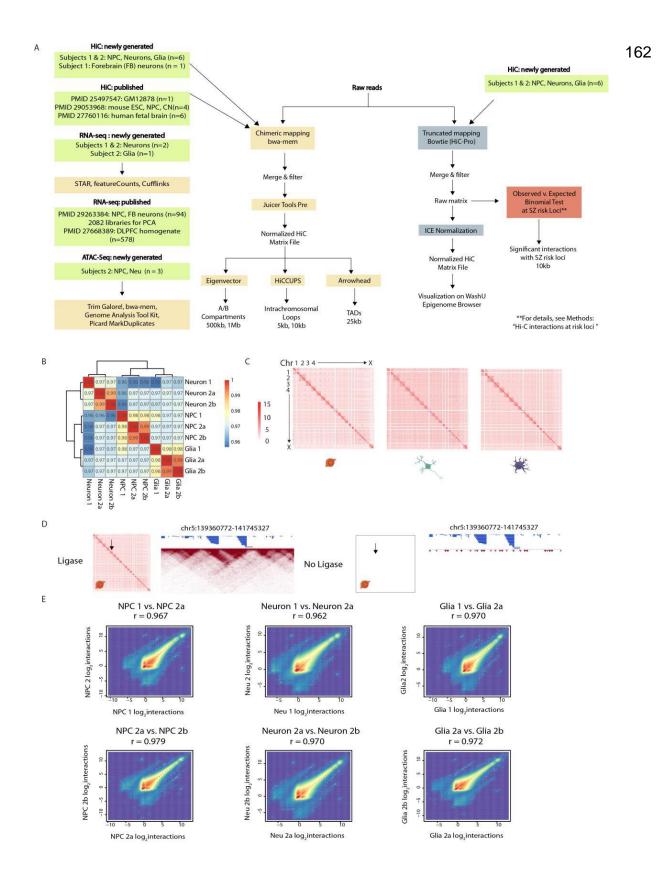


Figure 3.3 | Bioinformatic pipeline overview and basic library characteristics.

(A) Bioinformatic approaches involved in processing raw reads to arrive at Hi-C contact matrices, intrachromosomal loops and interactions, TADs, and compartments. Hi-C and other datasets used, both newly generated and previously published, are listed in green boxes. (B) Pearson correlation of logtransformed interaction bin counts for the 10% most variable intrachromosomal interactions at 500kb resolution, showing separation of libraries by cell type and not by individual identity. (See hierarchical clustering in Figure 3.1 A.) (C) Genome-wide Hi-C interaction matrix for NPC, neuron, and glia from donor S2 at 500kb resolution. (D) Genome-wide Hi-C heatmap of pooled NPC library (left) compared to No Ligase negative control library generated from NPC 1 (right), which shows a severely contact depleted map without the crucial ligation step. Black arrow points to chr5. Zoomed in TAD landscape at the clustered PROTOCADHERIN locus in pooled NPC library (left) and No Ligase NPC 1 (right). (E) Pearson correlation at 500kb resolution of intra- and interchromosomal interaction frequency between replicates, showing high level of correlation between different individuals ("1" v. "2") and technical replicates ("a" v. "b") within each cell type.

We first focused on intrachromosomal loop formations, which are

conservatively defined as distinct contacts between two loci in the absence of similar interactions in the surrounding sequences (Rao *et al.*, 2014). Our comparative analyses included published (Rao *et al.*, 2014) in situ Hi-C data from the B lymphocyte–derived cell line GM12878 (Table 3.1). When analyzed with the HiCCUPS pipeline (5- and 10-kb loop resolutions combined, subsampled to 372 million valid-intrachromosomal read pairs to reflect the library with the fewest reads after filtration) (Rao *et al.*, 2014), 17,767 distinct loops were called: n = 3118 (17.5%) were shared among all four cell types, whereas n = 5068 (28.5%) were specific to only one of the four cell types (Figure 3.1, C). Biologically relevant terms such as "central nervous system development," "forebrain development," and "neuron differentiation" were among the top gene ontology

(GO) enrichments from genes overlapping loops shared between NPCs, glia, and 164 neurons (brain-specific) but not identified in lymphocytes (Figure 3.1, D and Table 3.2), indicating strong tissue-specific loop signatures that were also confirmed in individual cell types (Figure 3.4, A and Tables 3.3 to 3.6).

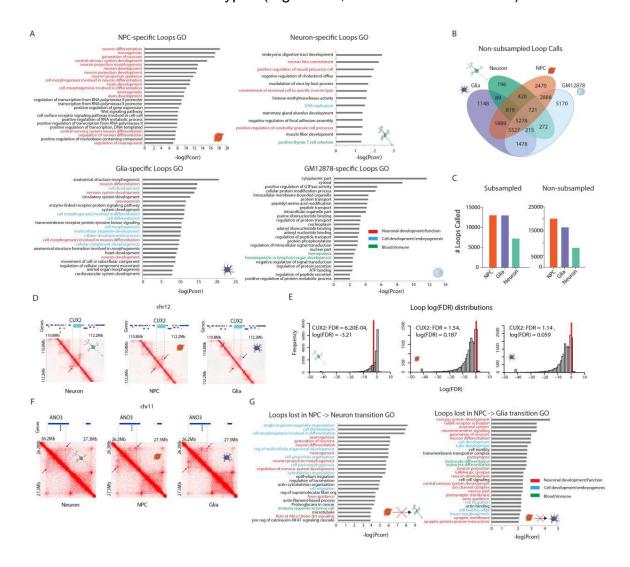


Figure 3.4 | Cell type-specific features of the 3D genome.

(A) GO enrichment of genes overlapping cell type-specific loops from nonsubsampled datasets, with genes overlapping loops specific to neural cell types involved in neurodevelopment while no such pattern is observed in GM12878. GO terms are color coded by functional categories (see legend). (B) Venn diagram of loops called from the non-subsampled datasets for each cell type. (C) Count of loops called in each cell type in the subsampled (to 372M cis contacts) and non-subsampled datasets, showing drastically reduced number of loops called in neurons. (**D**) Heatmaps at 10kb resolution with a neuron-specific loop (arrows; left) at CUX2 gene locus called in neurons but not in NPCs (middle) or glia (right), respectively. (**E**) Frequency distribution of log(FDR) of 17,767 loops (5kb and 10kb resolutions combined) in neurons (left), NPCs (middle), and glia (right). Vertical red line denotes the log(FDR) for the 10kb-resolution loop overlapping CUX2 in each cell type. Note that CUX2 loop is significant in neurons and not in NPCs or glia. (**F**) Representative example of a loop called in NPCs and glia but not in neurons. This loop spans 370kb, overlapping the gene ANO3. (**G**) GO enrichment of genes overlapping anchors of loops that are lost in the transition from NPC to neuron (left) and NPC to glia (right). GO terms are color coded by functional categories (see legend).

Unexpectedly, there was a reduction (~40 to 50% decrease) in the total number of chromosomal loops in neurons relative to isogenic glia and NPCs (Figure 3.4, B and C). Reduced densities of chromosomal conformations were also evident in genome browser visualization of chromosomal arms, including chr17q (Figure 3.1, E). Although both glia and NPCs harbored ~13,000 loop formations, only 7206 were identified in neurons (Figure 3.1, C; Figure 3.4, B and C; and table S1), including 442 neuron-specific loop formations. One such neuron-specific loop was at CUX2, a transcription factor whose expression marks a subset of cortical projection neurons (Gil-Sanz et al., 2015) and that is highly expressed in our NGN2-induced neurons (Figure 3.1, F and Figure 3.4, D and E). Examples of loops lost in neurons include one spanning the Ca²⁺ channel and dystonia-risk gene, ANO3 (Figure 3.4, F) (Charlesworth et al., 2012). Furthermore, NPCs, neurons, and glia had similar proportions of loops anchored in solely active (A) compartments, solely inactive (B) compartments, or in both, indicating no preferential loss of either active or inactive loops in neurons (Figure

3.1, G). However, among the genes overlapping anchors of loops that underwent 166 pruning during the course of the NPC-to-neuron transition, regulators of cell proliferation, morphogenesis, and neurogenesis ranked prominently in the top 25 GO terms with significant enrichment (Benjamini-Hochberg corrected $P_{range} = 10^{-3} - 10^{-8}$) (Figure 3.4, G and Table 3.4, B), which is consistent with a departure from precursor stage toward postmitotic neuronal identity (Wang *et al.*, 2017). Likewise, loops lost during NPC-to-glia transition were significantly enriched (Benjamini-Hochberg corrected $P_{range} = 10^{-2} - 10^{-5}$) for neuron-specific functions, including "transmission across chemical synapse," "g-aminobutyric acid (GABA) receptor activation," and "postsynapse" (Figure 3.4, G and Table 3.4, C), which is consistent with non-neuronal lineage commitment.

We defined "loop genes" as genes that either have gene body or transcription start site (TSS) overlap with a loop anchor (5- or 10-kb bins forming the points of contact in a chromatin loop). Genes with loop-bound gene bodies (one-tailed *Z* test, *Z*_{range} = 42.1 to 59.2, *P* < 10⁻³²⁴ for all) or loop-bound TSS (one-tail *Z*-test, *Z*_{range} = 15.2 to 28.8, *P*_{range} = 2.32×10^{-52} to 4.40×10^{-182}) both showed significantly greater expression [mean log₁₀(FPKM + 1); FPKM, fragments per kilobase of exon per million fragments mapped] than that of background (all genes for all brain cell types) (Figure 3.5, A), suggesting that looping architecture was associated with increased gene expression. Furthermore, 3% of loops shared by NPCs, neurons, and glia (brain-specific loops) interconnected a brain expression quantitative trait locus (eQTL) single nucleotide polymorphism (SNP) with its destined target gene(s), representing significant enrichment over background as determined with 1000 random distance- and functional annotation–matched loop samplings, (random sampling, one-sided empirical P = 0.012) (Materials and methods) (Figure 3.5, B).

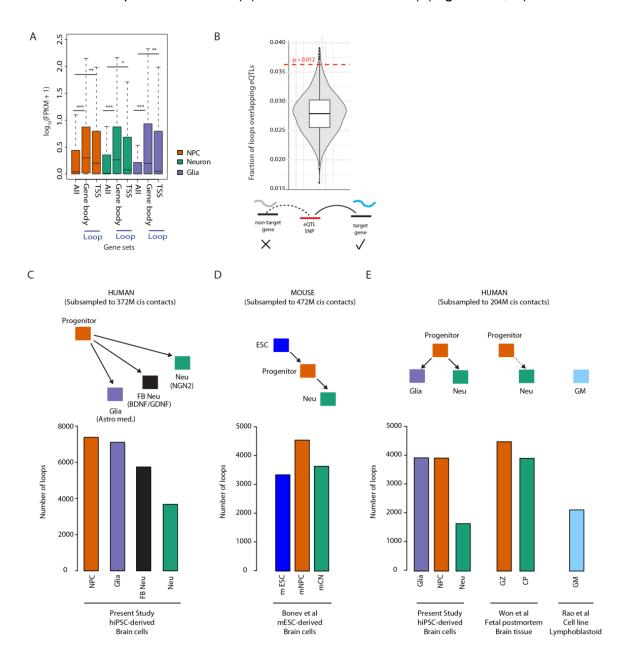


Figure 3.5 | Loop functional features and cell type-specific patterns in multiple model systems.

(A) Box-and-whisker plot comparing gene expression (log10(FPKM+1)) across all genes ("All"), the subset of genes located within the loop anchor ("Gene body"), and the subset of genes whose TSSs specifically are overlapping the loop anchor ("TSS"). * P < 10^{-50} , ** P < 10^{-150} , *** P < 10^{-300} . P < 10^{-324} for All v. Gene body across all 3 cell types (***); $P = 4.40 \times 10^{-182}$ All v. TSS in NPCs; P =2.32 x 10⁻⁵² All v. TSS in neurons; P = 8.23 x 10⁻¹⁷³ All v. TSS in glia (nonsubsampled datasets). (B) Violin plot of the distribution of fraction of overlaps between 1000 background loop sets (generated in silico) and brain eQTL SNPtarget gene pairs. An eQTL loop was counted if one bin contained the eQTL SNP (i.e., eSNP) and the other bin contained the target gene (bottom). Red dashed line indicates the fraction of overlap observed between brain-specific loops and brain eSNP-gene pairs. (C) Count of 10kb resolution loops called by HiCCUPS in hiPSC-derived libraries only, including directed differentiation forebrain neurons from S1 ("FB Neuron"), all subsampled to 372M valid cis contacts. (D) Count of loops called in mouse ESC-derived libraries that were all subsampled to 472M valid cis contacts. (E) Count of loops after all human libraries, including previously published fetal brain Hi-C, were subsampled to 204M valid cis contacts.

We aimed to confirm that the observed net loss of loop formations during the NPC to neuron transition could be replicated across a variety of independent cell culture and in vivo approaches and was not specific to our methodological choice of *NGN2*-induction. We conducted an additional Hi-C experiment on cells differentiated from hiPSC-NPCs by means of a non-*NGN2* protocol that used only differentiation medium and yielded a heterogeneous population of hiPSC forebrain-neurons in addition to a small subset of glia (Brennand *et al.*, 2011). In addition, we reanalyzed Hi-C datasets generated from a mouse model of neural differentiation, consisting of mouse embryonic stem cell (mESCs), mESC derived NPCs (mNPC), and cortical neurons (mCN) differentiated from the mNPCs via inhibition of the Sonic Hedgehog (SHH) pathway (Bonev *et al.*, 2017). To examine whether such genome-wide chromosomal loop remodeling also occurred in the developing brain in vivo, we reanalyzed Hi-C data from human fetal cortical plate (CP), mostly composed of young neurons, and forebrain germinal zone (GZ), primarily harboring dividing neural precursor cells in addition to a smaller subset of newly generated neurons (Won *et al.*, 2016). Across both the hiPSC-NPC-to-forebrain neuron and mESC-mNPC-mCN differentiation, in vitro neurons showed a 20% decrease in loops compared with their neural progenitors (Figure 3.5, C and D). Consistent with this, in vivo CP (neuron) compared with GZ (progenitor) showed a 13% decrease in loops genome-wide (Figure 3.5, E). The highly replicative cell types included here, mouse ESCs and human lymphoblastoid GM12878 cells, exhibited loop numbers very similar to their neuronal counterparts (Figure 3.5, D and E), suggesting that the changes in 3DG architecture from NPC to neurons do not simply reflect a generalized effect explained by mitotic potential.

Along with having fewer total loops, neurons exhibited a greater proportion of longer-range (>100 kb) loops than did NPCs or glia (two-sample two-tailed Kolmogorov-Smirnov test, *KS*_{range} = 0.1269 to 0.2317, *P* < 2.2 × 10⁻¹⁶ for three comparisons: Neu versus NPC/Glia/GM) (Figure 3.6, A). Likewise, in each of the alternative in vitro and in vivo analyses considered above, neurons exhibited a greater proportion of longer range (>100 kb) loops than did NPCs or glia [twosample two-tailed Kolmogorov-Smirnov test, *KS* = 0.0427, *P* = 1.5 × 10⁻⁵ for hiPSC-NPC versus forebrain neuron; *KS* = 0.0936, *P* = 1.1 × 10⁻¹⁶ for mESC-

NPC versus mCN; KS = 0.0663, $P = 2.04 \times 10^{-8}$ for fetal CP (neuron) compared 170 with GZ (progenitor)] (Figure 3.6, B, C, D, and E). Therefore, multiple in vitro and in vivo approaches comparing, in human and mouse, neural precursors to young neurons consistently show a reduced number of loops in neuron-enriched cultures and tissues, primarily affecting shorter-range loops.

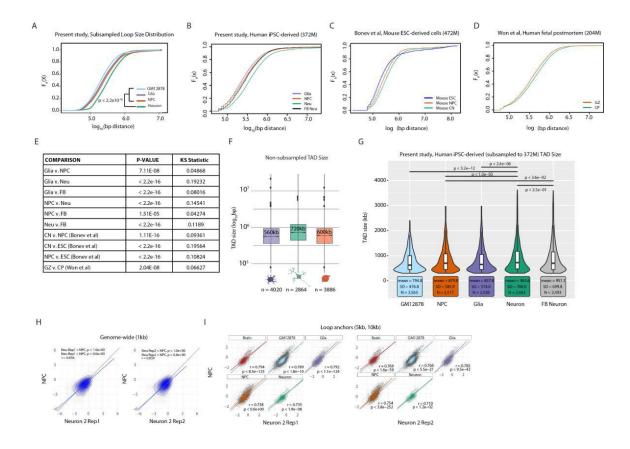


Figure 3.6 | Loop/TAD size comparisons across multiple datasets.

(A) Cumulative loop size distribution where $F_n(x)$ is the percent of loops whose size is less than or equal to the base pair distance (x). P-value is calculated using the Kolmogorov-Smirnov (KS) test. Neurons have a significantly larger proportion of loops that are longer in size than do other neural cells and non-neural GM12878 cells. (B) Cumulative loop size distribution of hiPSC-derived cell types subsampled to 372M cis contacts in the present study, including forebrain neurons. (C) Cumulative loop size distribution of mouse embryonic stem cells

(ESCs), ESC-derived neural progenitor cells (NPCs), and ESC-derived cortical 171 neurons (CN). (D) Cumulative loop size distribution of germinal zone (GZ) and cortical plate (CP) (E) Table of p-values and corresponding KS test statistics for each comparison presented in B-D. (F) Box-and- whisker plot of the distribution of TAD sizes across cell types in non-subsampled datasets, showing larger TADs in neurons than in the other two neural cell types. (G) Violin plot of TAD size across hiPSC-derived neural cell types, including directed differentiation FB neurons, and non-neural GM12878 cell lines. P-value is calculated using the Wilcoxon-Mann-Whitney test. (H) 2D density plots with coverage-balanced, log₂transformed ATAC signal for neurons on the x-axis and for NPCs on the y-axis. Open chromatin signatures of these two cell types were compared in 1kb bins genome-wide. P-values are reported from two-sided, paired t-tests. (I) 2D density plots with coverage-balanced, log2-transformed ATAC signal for neurons on the x-axis and for NPCs on the y-axis. Open chromatin signatures of these two cell types were compared in HiCCUPS loop anchors (5 and 10kb) that were shared by NPCs, neurons, and glia ("Brain"); specific to GM12878; or specific to only glia, NPCs, or neurons. P-values are reported from two-sided, paired t-tests.

Consistent with studies in peripheral tissues reporting conservation of the

overall TAD landscape across developmental stages, tissues, and species (when

considering syntenic loci) (Dixon et al., 2012, 2015), overall TAD landscapes

(Rao et al., 2014) remained similar between neurons, glia, and NPCs.

Nonetheless, TADs also showed a subtle (~10%) increase in average size in

neurons compared with isogenic NPCs, independent of the differentiation

protocol applied (Wilcoxon-Mann-Whitney test, $P < 3.6 \times 10^{-2}$) (Figure 3.1, H and

Figure 3.6, F and G), as highlighted here at a 3.4-Mb TAD at the *CDH*2 cell

adhesion gene locus (Figure 3.1, I). TAD remodeling may therefore reflect

restructuring of nested subdomains within larger neuronal TADs (Tables 3.7 and

3.8). To examine whether such developmental reorganization of the brain's

spatial genomes was associated with a generalized shift in chromatin structure,

we applied the assay for transposase accessible chromatin with high-throughput

sequencing (ATAC-seq) to map open chromatin sequences before and after *NGN2*-neuronal induction (Table 3.1). Genome-wide distribution profiles for transposase-accessible chromatin were only minimally different between NPCs and neurons (Figure 3.6, H) and further revealed that both NPCs and neurons showed low to moderate chromatin accessibility [$-2.5 < log_2(ATAC signal) < 1$] for $\geq 89\%$ of the anchor sequences comprising cell type–specific and shared "brain" loops in our cell culture system (Figure 3.6, I). These findings, taken together, point to widespread 3DG changes during the NPC-to-neuron transition and NPC-to-glia transition in human and mouse brain that are unlikely attributable to global chromatin accessibility differences. This includes highly cell type–specific signatures in gene ontologies of differentiation-induced loop prunings, reflecting neuronal and glial (non-neuronal) lineage commitment (Figure 3.4, A and G, and Table 3.4, B and C), and a subtle widening of average loop and TAD length in young neurons (Figure 3.1, H and Figure 3.6, A to G).

Chromosomal contacts associated with schizophrenia risk sequences

Because many schizophrenia risk variants lie in noncoding regions in proximity to several genes, we predicted that chromosomal contact mapping could resolve putative regulatory elements capable of conferring schizophrenia risk via their physical proximity (bypassing linear genome) to the target gene, as has been demonstrated in tissue in vivo (Won *et al.*, 2016; de la Torre-Ubieta *et al.*, 2018). We overlaid our cell type–specific interactions onto the 145 risk loci associated with schizophrenia risk (Ripke *et al.*, 2014; Pardiñas *et al.*, 2018).

Because only very few loops (defined as distinct pixels with greater contact frequency than neighboring pixels on a contact map) (Rao et al., 2014) were associated with schizophrenia risk loci (n = 212, 81, and 17 loops in NPC, glia, and neurons, respectively) (Table 3.9), we applied an established alternative approach to more comprehensively explore the 3DG in context of diseaserelevant sequences (Won et al., 2016). This approach defines interactions as those filtered contacts that stand out over the global background and applies binomial statistics to identify chromosomal contacts anchored at disease-relevant loci (Won et al., 2016). To begin, we examined the 40 loci with strongest statistical evidence for colocalization of an adult postmortem brain eQTL and schizophrenia genome-wide association study (GWAS) signal (Dobbyn et al., 2018). Chromosomal contacts were called for 29 of the 46 eQTLs present in the 40 loci, with 8 of 29 (28%) of the loci showing significant interactions (binomial test, $-\log q$ value range = 1.33 to 11.0) between the eQTL-SNPs (eSNPs) in the one contact anchor and the transcription start site of the associated gene(s) in the other anchor (Table 3.10). We conclude that ~30% of risk locus–associated eQTLs with strong evidence for colocalization with GWAS signal bypass the linear genome and are in physical proximity to the proximal promoter and transcription start site of the target gene, resonating with previous findings in fetal brain tissue that used a similar contact mapping strategy (Won et al., 2016).

Cell type–specific contact maps with 10-kb-wide bins, queried for the schizophrenia-associated loci, frequently revealed differential chromosomal

conformations in NPCs, glia, and neurons. For example, the risk locus upstream 174 of the PROTOCADHERIN cell adhesion molecule gene clusters (chromosome 5), which is critically relevant for neuronal connectivity in developing and adult brain (Figure 3.7, A) (Yagi, 2012; Chen and Maniatis, 2013), showed through both observed/expected interaction matrix (Durand, Robinson, et al., 2016) and global background-filtered contact mapping (Won et al., 2016) a bifurcated bundle of interactions in NPCs, with one bundle emanating to sequences 5' and the other bundle to sequences 3' from the risk locus. In neurons, the 3' bundle was maintained, but the 5' bundle was "pruned," whereas glia showed the opposite pattern; these differences between the three cell types were highly significant (observed/expected Wilcoxon rank sum $P < 10^{-9}$ to 10^{-15}) (Figure 3.8, A to C). Dosage of the noncoding schizophrenia risk-SNP (rs111896713) at the PCDH locus significantly increased the expression of multiple PROTOCADHERIN genes (PCDHA2, PCDHA4, PCDHA7, PCDHA8, PCDHA9, PCDHA10, and PCDHA13) in adult frontal cortex of a large cohort of 579 individuals, including cases with schizophrenia and controls (Figure 3.7, B and Table 3.11) (Fromer *et al.*, 2016). The affected genes were interconnected to the disease relevant noncoding sequence in neurons and NPCs but not in glia (Figure 3.7, C). Therefore, cell-type-specific Hi-C identified chromosomal contacts anchored in schizophrenia-associated risk sequences that affected expression of the target gene(s). On the basis of earlier chromosome conformation capture assays at the site of candidate genes, the underlying

mechanisms may include alterations in transcription factor and other nucleoprotein binding at loop-bound cis-regulatory elements (Bharadwaj *et al.*, 2014) or even local disruption of chromosomal conformations (Bharadwaj *et al.*, 2013).

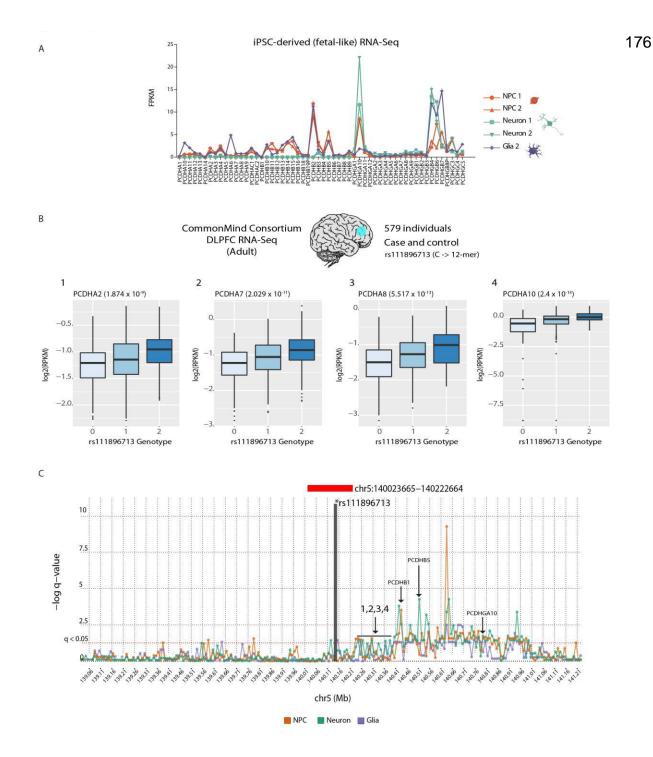


Figure 3.7 | Risk-associated chromosomal contact mapping.

(A) RNA-seq FPKM expression for all clustered *PCDH* genes in NPCs (orange), neurons (green), and glia (purple). (B) The association of expression of *PCDHA2* (1; $P = 1.874 \times 10^{-9}$), *PCDHA7* (2; $P = 2.029 \times 10^{-11}$), *PCDHA8* (3; $P = 5.517 \times 10^{-13}$), *PCDHA10* (4; $P = 2.4 \times 10^{-14}$) with schizophrenia risk allele at the GWAS index SNP rs111896713, upstream of the clustered *PCDH* genes, using the CommonMind Consortium (CMC) postmortem prefrontal cortex RNA-seq dataset consisting of 258 schizophrenia subjects and 279 controls. (C) Binomial interaction landscape with 10kb anchor bin (gray vertical box) containing rs111896713 within the larger schizophrenia risk locus (solid red horizontal box), showing significant (above horizontal red dotted line) interactions with the four PROTOCADHERIN genes no. 1 to no. 4 significantly affected by risk polymorphisms (shown in B) in neurons (green) and NPCs (orange), but not in glia (purple).

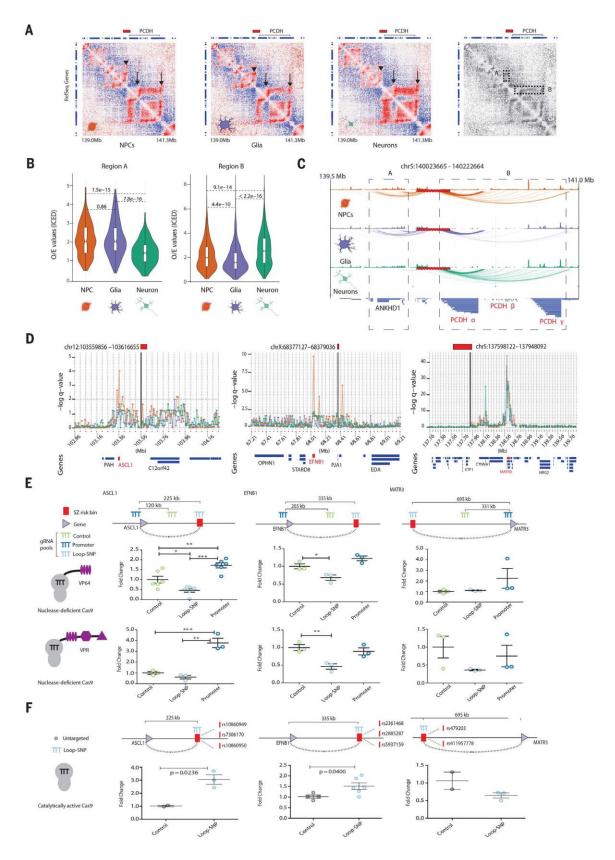


Figure 3.8 | Cell type–specific chromosomal contact maps at schizophrenia risk loci.

(A) Juicebox observed/expected interaction heatmaps at 10-kb resolution for the risk associated clustered PCDH locus chr5:140023665-140222664 for NPC. glia. and neurons as indicated. (Far right) Grayscale heatmap depicts areas of highly cell-specific contact enrichments: upstream genes including ANKHD1 (dotted rectangle "A" and arrowhead) and downstream PCDH gene clusters (dotted rectangle "B" and arrows). Clustered PCDH gene expression patterns are available in Figure 3.7, A. (B) Violin plots of observed/expected interaction values in the regions A and B highlighted in (A). (C) Map of contacts identified by binomial statistics. Red box with dashed black line represents the schizophrenia risk locus, dotted boxes regions "A" and "B" in heatmaps. (D) Cell-type resolved contact map of 10-kb bins (bold, black vertical lines) within risk sequences on chr12 (left), chrX (middle), and chr5 (right); NPC (orange), neuron (green), glia (purple); –log q value, significance of contact between schizophrenia risk locus and each 10-kb bin; gene models ("Genes") below with SNP-loop target gene highlighted in red. (E) Epigenomic editing (CRISPRa with nuclease-deficient dCas9 in NPCs) for three risk SNP-target gene pairs and their respective control sequences (top), measured with quantitative reverse transcription polymerase chain reaction (RT-PCR) (fold change from baseline) for VP64 (middle) and VPR (bottom) transcriptional activators. (F) Quantitative PCR gene expression changes upon directing catalytically active Cas9 to schizophrenia risk-associated credible SNPs (vertical red dashes with rsIDs) interacting via chromosomal contacts with promoters of ASCL1, EFNB1, and MATR3 in NPCs. Targeting strategy and contact distances depicted above: *P < 0.05, **P < 0.01, ***P < 0.0001 (Figures 3.7 and 3.9).

Transcriptional profiles of hiPSC derived NPCs and neurons most closely

resemble those of the human fetus in the first trimester (Brennand et al., 2015);

moreover, a portion of the genetic risk architecture of schizophrenia matches to

regulatory elements that are highly active during prenatal development (Gulsuner

et al., 2013). We surveyed in our Hi-C datasets seven loci encompassing 36

"credible" (potentially causal) schizophrenia-risk SNPs with known chromosomal

interactions in fetal brain to genes important for neuron development and function

(Won et al., 2016). We found that risk-associated chromosomal contacts were

conserved between our hiPSC NPCs and the published human fetal CP and germinal zone Hi-C datasets (Won et al., 2016) for five of the seven loci (71%) tested (CHRNA2, EFNB1, MATR3, PCDH, and SOX2, but not ASCL1 or DRD2) (Table 3.12). To test the regulatory function of these conserved risk sequence bound conformations, we performed single-guide RNA (sgRNA)-based epigenomic editing experiments on isogenic antibiotic-selected NPCs that stably express nuclease-deficient dCas9-VP64 (Maeder et al., 2013; Perez-Pinera et al., 2013) or dCas9-VPR (Chavez et al., 2016; Ho et al., 2017) transactivators (Table 3.13). Previous studies in peripheral cell lines succeeded in inducing gene expression changes by placing dCas9-repressor fusion proteins at the site of chromosomal contacts separated by up to 2 Mb of linear genome from the promoter target (Fulco et al., 2016). We tested ASCL1-, EFNB1-, MATR3-, and SOX2- bound chromosomal contacts separated by 200- to 700-kb interspersed sequences (Figure 3.8, D and E; Figure 3.9, A; and Table 3.14). Pools of five individual sgRNAs directed against a risk-associated noncoding sequence bypassing 225 and 355 kb of genome consistently resulted in significantly decreased expression of ASCL1 [one-way analysis of variance (ANOVA), $F_{VP64}(2, 15) = 22.20, P < 0.0001$; Dunnett's $P_{VP64} = 0.023$] and EFNB1 target genes [one-way ANOVA, $F_{VP64}(2, 6) = 14.47$, P = 0.0051, Dunnett's $P_{VP64} =$ 0.0356; $F_{VPR}(2, 6) = 1.46$, P = 0.0111, Dunnett's $P_{VPR} = 0.0088$], in comparison with positive (promoter bound) and negative (linear genome) control sgRNAs. Epigenomic editing of risk sequence 500 to 600 kb distant from the SOX2 and

MATR3 loci did not alter target gene expression (Figure 3.8, D and E, and Figure 181 3.9, A and B), which could reflect practical limitations in nonintegrative transfection based (as opposed to viral) methods, impact of epigenetic landscape, or suboptimal guide RNA positioning (Ho et al., 2017), further limited by the 10-kb contact map resolution. Because portions of the MATR3-bound risk sequences are embedded in repressive chromatin, we directed five sgRNAs for Cas9 nuclease mutagenesis toward a 138-base pair (bp) sequence within a MATR3 long-range contact that was enriched with trimethyl-histone H3K27me3, commonly associated with *Polycomb* repressive chromatin remodeling, in order to disrupt it (Figure 3.9, C to E). This strategy produced a significant increase in *MATR3* expression upon ablation of the putative repressor sequence, whereas targeting MATR3 (linear genome) control sequence remained ineffective (Figure 3.9, D and E). We conducted additional genomic mutagenesis assays, with sgRNAs directly overlapping credible SNPs participating in chromatin contacts with ASCL1, EFNB1, EP300, MATR3, PCDHA7, PCDHA8, and PCDHA10 (Table 3.13). Cas9 nuclease deletion of interacting credible SNPs significantly increased gene expression of ASCL1, EFNB1, and EP300 (Prange = 0.0053 to 0.04, trange = 2.449 to 4.265) (Figure 3.8, F and Figure 3.9, F). Similar targeting of four credible SNPs upstream of the clustered *PCDH* locus significantly decreased levels, by ~50 to 60%, of PCDHA8 and PCDHA10 (Prange = 0.0122 to 0.0124, trange = 4.326 to 4.343), two of the genes whose expression increased with dosage of the risk SNP rs111896713 in adult postmortem brain (Figure 3.7, B and Figure 3.9, G).

Taken together, our (epi)genomic editing assays (Figure 3.9, H) demonstrate that 182 chromosomal contacts anchored in schizophrenia risk loci potentially affect target gene expression across hundreds of kilobases, which is consistent with predictions from chromosomal conformation maps from hiPSC-derived brain cells described here, and from developing (Won *et al.*, 2016; de la Torre-Ubieta *et al.*, 2018) and adult (Bharadwaj *et al.*, 2014) human brain tissue.

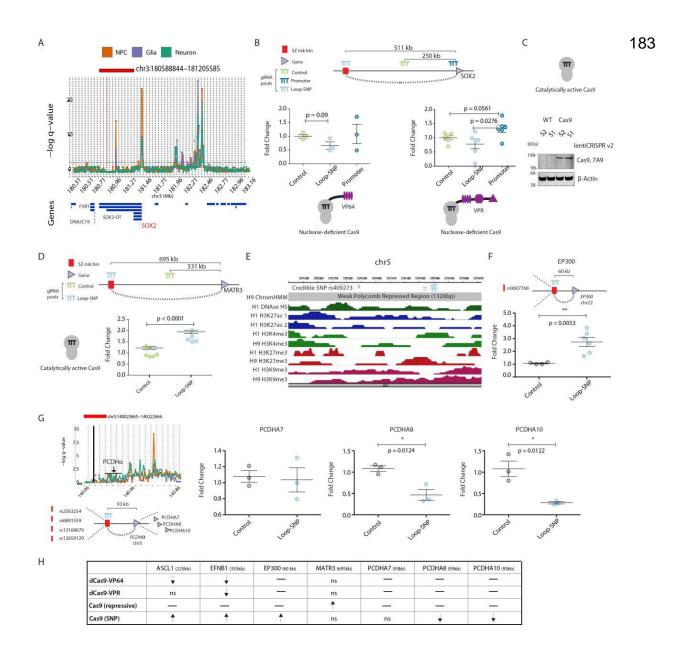


Figure 3.9 | Epigenomic and genomic editing at schizophrenia riskassociated chromosomal conformations.

(A) Interaction landscape at the *SOX2* locus on chr3, where highlighted (red) 10kb bin in *SOX2-OT* has a strong interaction with *SOX2*, a key transcription factor important in maintaining pluripotency in neural precursors. (B) *SOX2* epigenomic editing strategy (top) with results from VP64 (middle) and VPR (bottom) platforms, demonstrating that promoter-targeted gRNAs show increased gene expression and schizophrenia (SZ) risk SNP-targeted gRNAs potentially

decrease expression. (C) Western blot for Cas9 protein levels in wild type (left) 184 and NPCs transduced with lentiviral vector containing Cas9 (right). (D) CRISPR deletion strategy (top) for contact-associated schizophrenia risk region (top) and results (bottom), which shows increased MATR3 after noncoding deletion ~700kb from promoter. Note that no SNPs were targeted by sgRNAs in this experiment, and the closest risk-associated SNP was 400bp from the nearest sgRNA. Positions of sgRNA and of nearest risk SNP are shown in Panel E browser window. (E) ENCODE epigenomic landscape of H9/H1 NPCs at the targeted region in panel D, suggesting weak polycomb repressed region. (F) qPCR gene expression upon directing catalytically active Cas9 to schizophrenia riskassociated credible SNPs (vertical red dashes with rsIDs) interacting with the promoters of *EP300*. Targeting strategy and interaction distance depicted above. (G) PCDH interaction landscape of 4 credible SNPs (vertical red dashes) with members of the PCDH α cluster, spanning roughly a 93kb distance (left). qPCR expression results for PCDHA7, PCDHA8, and PCDHA10 after Cas9 mutagenesis of PCDH credible SNPs. (H) Summary table of CRISPR epigenomic and genomic editing experiments.

Cell type–specific schizophrenia-related chromosomal connectomes are associated with gene co-regulation and protein-protein association

networks

Having shown that the chromosomal contact maps anchored in sequences associated with schizophrenia heritability undergo cell type–specific regulation (Figure 3.8, A to C), are reproducible in neural cell culture and fetal brain (Table 3.12), frequently harbor risk-associated eQTLs (Table 3.10), and bypass extensive stretches of linear genome to affect target gene expression in genomic and epigenomic editing assays (Figure 3.8, D to F, and Figure 3.9), we investigated chromosomal contacts for all 145 GWAS-defined schizophrenia risk loci together (Pardiñas *et al.*, 2018).We refer to the resulting "network" of risk loci and their 3D proximal genes as the "schizophrenia-related chromosomal connectome."

Earlier studies in adult brain had shown that open-chromatin-associated 185 histone modification and other "linear epigenome" mappings strongly link the genetic risk architecture of schizophrenia specifically with neuronal, as opposed to non-neuronal, chromatin (Girdhar et al., 2018), which would suggest that similar cell-specific signatures may emerge in the risk-associated 3DG. Neurons and NPCs, but not the isogenic glia, showed a high preponderance of chromosomal contacts with schizophrenia-associated risk loci (Figure 3.10, A). There were 1203 contacts involving schizophrenia risk sequences that were highly specific to neurons (median distance between risk and target bins = 510kb), 1100 highly specific for NPCs (median distance between risk and target bins = 520 kb), whereas only 425 highly specific for glia (median distance between risk and target bins = 580 kb) (Figure 3.10, A; Figure 3.11, A and B). There were also unexpectedly robust cell type- and gene-ontology-specific signatures. including genes associated with neuronal connectivity and synaptic signaling (Figure 3.10, B and Tables 3.15 and 3.16). Separate analysis of the Psychiatric Genomics Consortium "PGC2" 108 risk loci (Ripke et al., 2014) yielded similar results (Figure 3.12, A and B).

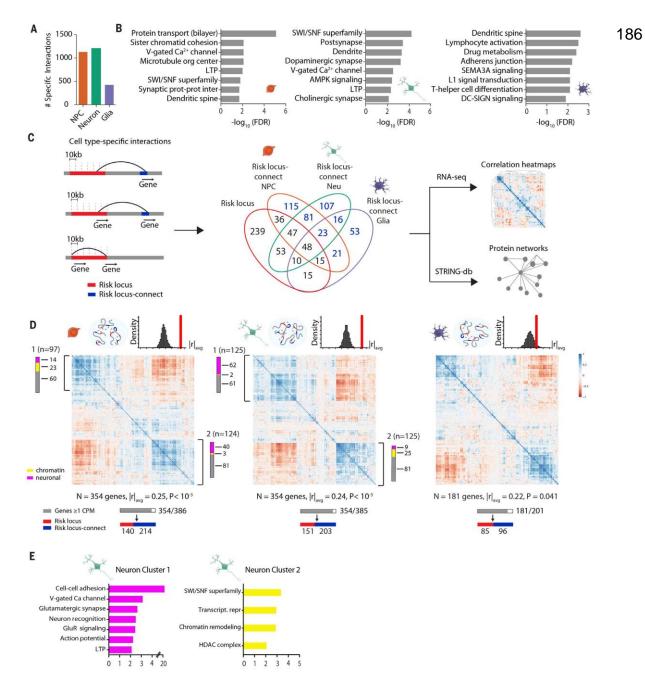


Figure 3.10 | Expanded GWAS risk connectome is associated with gene coregulation.

(A) Counts of highly cell type–specific contacts associated with schizophrenia risk in each of the three hiPSC-derived cell types. (B) GO enrichment of genes located in schizophrenia risk contacts in NPCs (left), neurons (middle), and glia (right). (C) (Left) Schematic workflow of analyses performed with cell type specific contact genes, distinguished as "risk locus" and "risk locus–connect" genes. (Middle) Venn diagram of genes located in the 145 loci and those found in

cell type-specific contacts, with numbers in blue indicating "risk locus-connect" genes. (Right) Schematic workflow of analyses performed with combined set of "risk locus" and "risk locus-connect" genes. (D) RNA Pearson transcriptomic correlation heatmaps consisting of risk locus and risk locus-connect genes derived from the cell type-specific contacts of NPCs (left), neurons (middle), and glia (right). Organization scores (/r /avg) for each heatmap are reported with P values from sampling analysis. Schematics above heatmaps are representations of each cell type's particular connectome (blue oval) and frequency distribution of organization scores from permutation analyses of randomly generated heatmaps (red, observed organization score of heatmap being tested). The gray bar corresponds to *n* genes that have at least 1 count per million in RNA-seq dataset out of the total number of genes and are used to construct the heatmap; red and blue bars indicate how many of the genes in the heatmap are in a risk locus (red) and are risk locus-connect (blue). Fuchsia, neuron connectivity/synaptic function genes; yellow, chromatin remodeling genes as determined from gene ontology analysis in (E). Additional information on coexpression clusters is provided in tables S22 and S23 (Figure 3.11 and 3.12).

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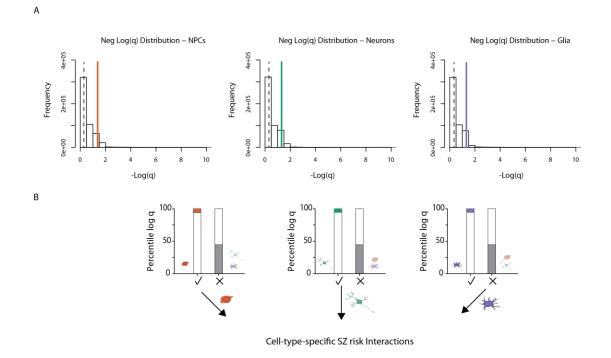


Figure 3.11 | Determining cell type-specific PGC interactions.

(A) Frequency histograms of the distribution of $-\log(q)$ values of binomial interactions for each cell type. Colored lines = 95th percentile of the union set of

all $-\log(q)$ (1.47); grey dashed lines = 50th percentile of the union set of all $-\log(q)$ 188 (0.33). (**B**) Work flow for determining cell type-specific PGC interactions. Interactions that had $-\log(q)$ values \geq 95th percentile in one cell type (color highlight) and $-\log(q)$ values < 50th percentile in the remaining two cell types (grey highlight) were defined as cell type-specific interactions.

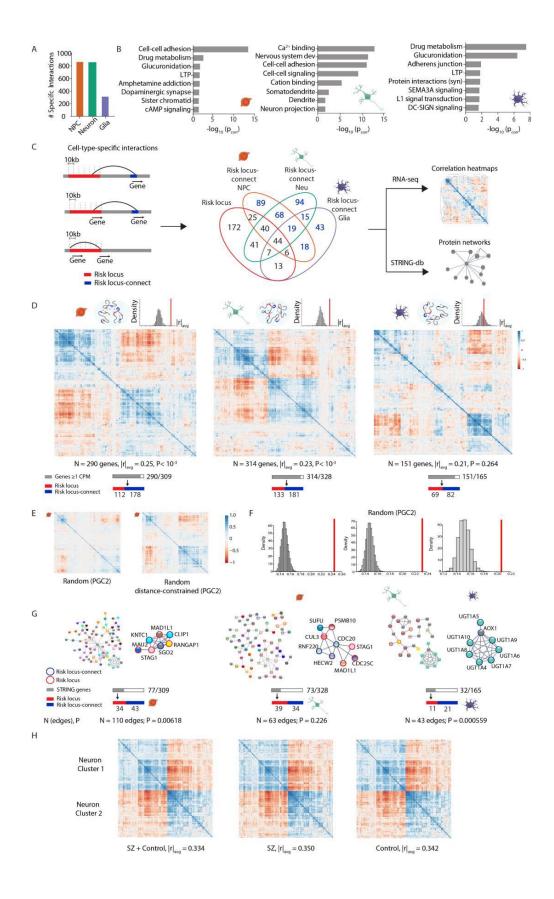


Figure 3.12 | Expanded GWAS risk connectome is associated with significant gene coregulation and protein-protein association networks. (A) Counts of highly cell type-specific significant interactions in each of the 3 hiPSC-derived cell types. (B) GO enrichment of genes located in significant schizophrenia risk- interactions in NPCs (left), neurons (middle), and glia (right). (C) Schematic workflow of analyses performed with cell type-specific interaction genes, distinguished as "risk locus" and "risk locus-connect" genes (left). Venn diagram (middle) of genes located in the 108 loci and those found in cell typespecific interactions, with numbers in blue indicating "risk locus-connect" genes. (D) RNA Pearson correlation heatmaps consisting of risk locus and risk locusconnect genes derived from the cell type-specific interactions of NPCs (left), neurons (middle), and glia (right). Organization scores ($|r|_{avg}$) for each heatmap are reported with P-values from permutation analysis. Schematics above heatmaps are representations of each cell type's unique connectome (blue oval) and frequency distribution of organization scores from permutation analyses of randomly generated heatmaps (red = observed organization score of heatmap being tested); grey bar corresponds to n genes that have at least 1 count per million in RNA-seg dataset out of the total number of genes and are used to construct the heatmap; red and blue bars indicate how many of the genes in the heatmap are in a risk locus (red) and are risk locus-connect (blue). (E) Representative example of a heatmap generated by randomly sampling an identical number of genes as in the heatmap being gueried without (left) and with (right) distance constraints (see Materials and methods for inclusion criteria and further details). (F) Frequency distribution of permuted organization scores for NPCs (left), neurons (middle), and glia (right) for random permutation analyses without distance-constraint in the PGC2-anchored interactions. (G) Overview and representative examples (zoomed in) of protein-protein association networks in NPCs (left), neurons (middle), and glia (right). Numbers of edges connecting the proteins in each network and STRING-computed *P*-values are reported below. Grey bar indicates the subset of these genes whose proteins are involved in the network out of the total number of genes from cell type-specific interactions; red and blue bars indicate how many of the genes in the network are in a risk locus (red) and are risk locus-connect (blue). Risk locus (red circle outline) and risk locus-connect (blue circle outline) are marked in the representative examples. (H) Subset heatmaps of only those genes in the neuronal coexpression clusters 1 and 2 (refer to Figure 3.10 D) from all 94 (hiPSC-derived NPC and neurons; N = 47 libraries from 14 schizophrenia cases and N = 47 libraries from 12 controls) samples (left), schizophrenia cases only (middle), and control only (right).

Because spatial 3DG proximity of genes is an indicator for potential

coregulation (Kustatscher, Grabowski and Rappsilber, 2017), we tested whether

the neural cell type-specific schizophrenia-related chromosomal connectome showed evidence of coordinated transcriptional regulation and proteomic interaction of the participating genes. To this end, we generated lists of genes anchored in the most highly cell type-specific schizophrenia risk associated contacts (Materials and methods) (Figure 3.10, C, Figure 3.11, B, and Table 3.15). Thus, for the NPC-specific contacts, we counted 386 genes, including 146 within the risk loci and another 240 genes positioned elsewhere in the linear genome but connected via an intrachromosomal contact to within-risk-locus sequences. Similarly, for the neuron-specific contacts, we identified 385 genes, including 158 within risk loci and 227 outside of risk loci (Figure 3.10, C). Last, for glia-specific contacts, we identified 201 genes, including 88 within and 113 outside of risk loci. We labeled the intrachromosomal contact genes located outside of schizophrenia risk loci as "risk locus-connect," which we define as a collection of genes identified only through Hi-C interaction data, expandingdepending on cell type—by 50 to 150% the current network of known genes overlapping risk sequences that is informed only by GWAS (Figure 3.10, C).

To examine whether such types of disease associated, cell-type-specific chromosomal connectomes were linked to a coordinated program of gene expression, we analyzed a merged transcriptome dataset (comprised of 47 hiPSC-NPC and 47 hiPSC-forebrain neuron RNA-seq libraries from 22 schizophrenia and control donors not related to those of our Hi-C datasets) (Hoffman *et al.*, 2017). We examined pair-wise correlations of the collective sets

of the 386 NPC, 385 neuron, and 201 glia genes representing "risk locus" and "risk locus-connect" genes (cell-type-specific "risk connectomes"). The risk connectome for each cell type showed extremely strong pair-wise correlations, with two of the largest clusters visualized on the neuron and NPC correlation matrices involving an admixture of 354 "risk locus" and "risk locus connect" genes each, and similarly 181 genes from the glia matrix (Figure 3.10, D and Table 3.17). The averaged gene-by-gene transcript correlation index for each matrix overall, defined here as "organization score" ($|r|_{avg}$), for the NPCs, neurons, and glia were 0.22 to 0.25. Such levels of organized gene expression were robustly significant for NPC and neurons, after controlling for linear genomic distance (1000 random samplings, $|r|_{avg}$, P < 0.001 for NPC and for neuron; P =0.041 for glia) (Figure 3.10, D, Figure 3.12, E, and Table 3.18). There were four large clusters in the correlation matrices of the neuronal and NPC risk connectome: neuronal connectivity and synaptic signaling proteins (neuron cluster 1 and NPC cluster 2) and epigenetic regulators (neuron cluster 2 and NPC cluster 1). For example, within neuron cluster 1 (Figure 3.10, D, middle), 62 of 125 genes encoded neural cell adhesion and synaptic molecules, voltagegated ion channels, and other neuron specific genes (Figure 3.10, E and Tables 3.19 and 3.20). We thus conclude that the chromosomal connectomes associated with schizophrenia risk are cell type specific, with the neuronal risk connectome particularly enriched for genes pertaining to neuronal connectivity, synaptic signaling, and chromatin remodeling (Figure 3.10, D and E). Analyses of

the subset of PGC2 risk loci (108 and 145) provided similar results (Figure 3.12, 193 C to F). Additionally, organization scores for neuron cluster 1 and cluster 2 genes were similar between hiPSC-derived NPCs and forebrain neurons from schizophrenia cases (n = 47) and control (n=47), suggesting that many risk locus–connect and risk locus genes are coregulated across individuals (Figure 3.12, H).

Numerous proteins encoded by risk locus and risk locus-connect genes were associated with synaptic signaling (Table 3.21). The cell type-specific risk locus-connect and risk locus genes show significant protein-protein interaction network effects for NPCs (P = 0.0004) and neurons (P = 0.009) but not glia (Figure 3.13, A, Figures 3.14 to 3.16, and Table 3.21) when examined by using the STRING database v10.5 (Szklarczyk et al., 2015, 2017). We observed many proteomic clusters, including large groups of epigenomic regulators associated with the SWI/SNF (SWItch/Sucrose Non-Fermentable) chromatin remodeling complex and histone lysine methyltransferases and demethylases (Figure 3.13, A and Figures 3.14 and 3.15), many of which were the genes identified in NPC cluster 1 and neuron cluster 2 of the transcriptome analysis (Figure 3.10, D and E). The transcriptomic correlation heatmaps for these protein networks ("STRING" genes), when compared with randomly generated subset heatmaps from the overall ("Full") schizophrenia-related chromosomal connectome (Figure 3.10, D), had higher organization scores in NPCs and neurons (NPC $|r|_{avg}$ = 0.2963, P = 0.007; neuron $|r|_{avg} = 0.2877$, P = 0.008, glia $|r|_{avg} = 0.2225$, P = 0.008

0.595, STRING versus full permutation test) (Materials and methods) (Figure 3.13, B, Figures 3.17 to 3.19, and table S21). Because the transcriptomic correlation heatmap for the schizophrenia-related chromosomal connectome was significantly decreased by the removal specifically of the NPC STRING protein network genes ($P < 10^{-3}$) (Table 3.21), this subset of STRING-interacting proteins may drive the observed orchestrated coregulation. Within these transcriptome- and proteome-based regulatory networks were numerous occasions of coregulated (RNA) and interacting (protein) risk locus and risk locus–connect genes that share the same TAD, including *CDC20*, which regulates dendrite development (Puram *et al.*, 2011; Watanabe, Khodosevich and Monyer, 2014) and is associated at the protein level with *RNF220*, an E3 ubiquitin-ligase and β -catenin stabilizer (Figure 3.13, C) (Ma *et al.*, 2014).

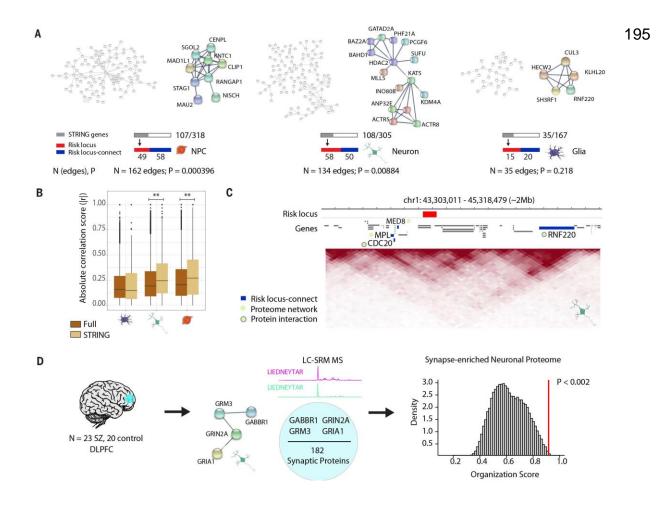
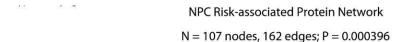


Figure 3.13 | Expanded GWAS risk connectome is linked to protein-protein association networks.

(A) Overview and representative examples (zoomed in) of protein-protein association networks in NPCs (left), neurons (middle), and glia (right). Numbers of edges connecting the proteins in each network and STRING-computed *P* values are reported below. Gray bar indicates the subset of these genes whose proteins are involved in the network out of the total number of genes from cell type–specific interactions; red and blue bars indicate how many of the genes in the network are in a risk locus (red) and are risk locus–connect (blue).

(B) Comparison of organization scores between the full RNA transcriptomic correlation heatmaps (brown) (Figure 3.10, D) and the "STRING" heatmaps (tan) (Figures 3.17 to 3.19), consisting of only those genes in protein networks for each cell type. Permutation test, **P<0.01. (C) Representative neuronal TAD landscape (chr1, ~2 Mb) depicting a schizophrenia risk–associated locus (red) with its risk locus–connect genes (blue), *MED8*, *MPL*, *CDC20*, and *RNF220*, which are members of the neuronal schizophrenia protein network (green circle). *CDC20* and *RNF220* interact at the protein level (green circle with gray border).

(**D**) (Left) Liquid chromotography–selected reaction monitoring (LC-SRM) mass spectrometry (MS) was performed on dorsolateral prefrontal cortex (DLPFC) tissue from 43 adult postmortem brains (23 schizophrenia, 20 control). (Middle) 182 neuronal proteins were reliably quantified, and four of them were observed to have associations in the neuron protein network in (A). (Right) GABBR1, GRM3, GRIN2A, and GRIA1 proteins were found to have significantly more correlated expression than expected by random permutation analysis. Additional information on protein-protein interactions is provided in Figures 3.12 to 3.19.



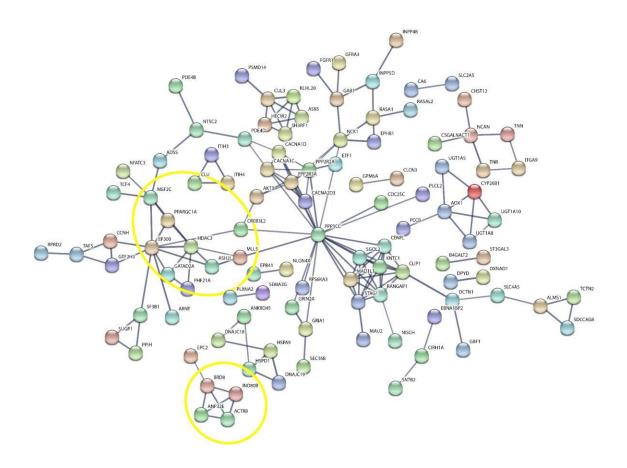


Figure 3.14 | NPC schizophrenia risk-associated protein-protein association network.

Protein-protein network (STRING) derived from genes located in NPC-specific schizophrenia risk interactions. Yellow circles roughly outline groups of genes associated with chromatin remodeling, as highlighted in Figure 3.10, D and E.

Neuron Risk-associated Protein Network

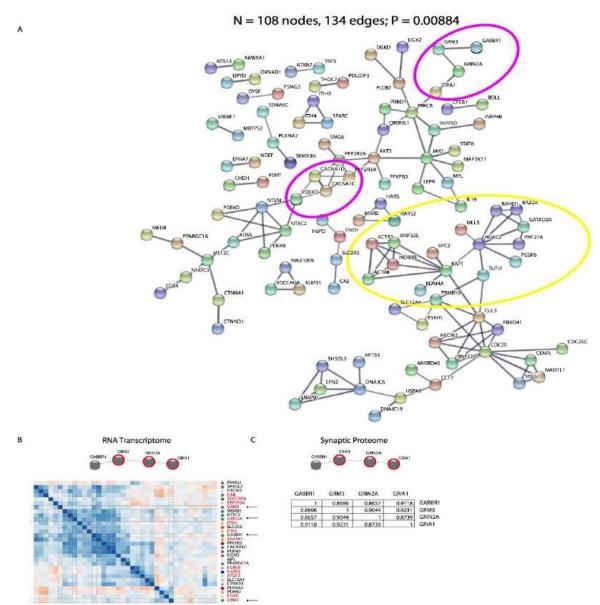


Figure 3.15 | Neuron schizophrenia risk-associated protein-protein association network.

(A) Protein-protein network (STRING) derived from genes located in neuronspecific schizophrenia risk interactions. Yellow and fuchsia circles roughly outline groups of genes associated with chromatin remodeling and neuronal connectivity, respectively, as highlighted in Fig 3.10, D and E. (B) RNA correlation heatmap (PGC2) zoomed in to a cluster of coexpression that contains 4 genes of interest (*GABBR1*, *GRM3*, *GRIN2A*, *GRIA1*) that were identified in the

protein network in panel A. Red = risk locus genes are in red; arrows indicate 4 198 synaptic genes of interest; each color circle represents a different TAD whereby genes co-localized with one or more genes in same TAD or adjacent TADs share the same color. (**C**) Pearson correlation matrix of protein measures for the 4 proteins of interest from LC-SRM MS of adult postmortem DLPFC.

Glia Risk-associated Protein Network

N = 35 nodes, 35 edges; P = 0.218

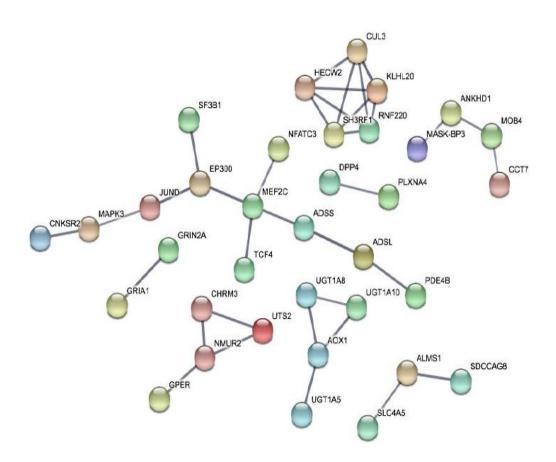
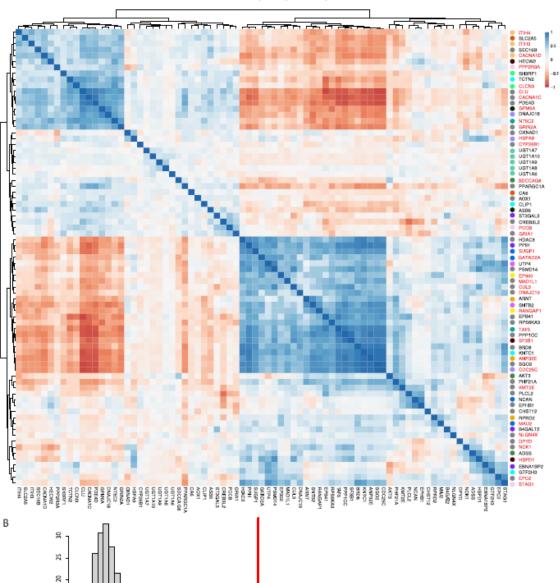


Figure 3.16 | Glia schizophrenia risk-associated protein-protein association network.

Protein-protein network (STRING) derived from genes located in glia-specific schizophrenia risk interactions.



0.30

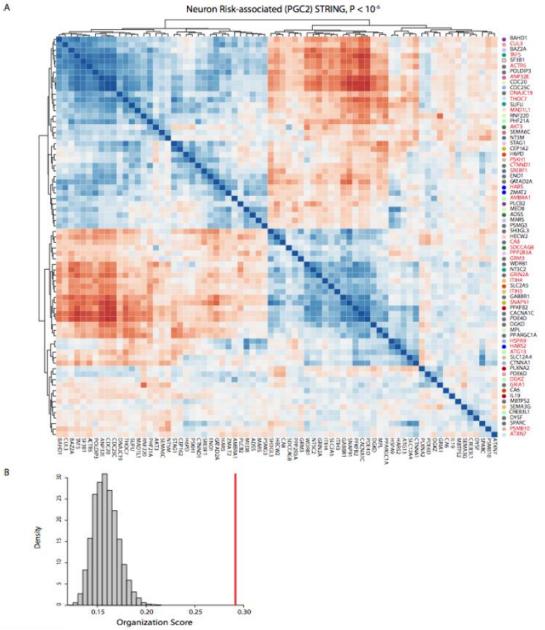
А

0.15

0.20 0.25 Organization Score

Figure 3.17 | NPC schizophrenia risk-associated STRING subset genes 2 show greater transcriptional organization than full risk connectome gene list (PGC2).

(A) Pearson correlation heatmap of genes participating in NPC-specific STRING protein-protein network. Risk locus gene (red), gene present in TAD with no other genes in the network (grey circle), gene co-localized with one or more genes in same TAD or adjacent TADs (colored circles; each color represents a different TAD; see Table S24). (B) Frequency distribution of permuted organization scores, randomly sampling from all genes in NPC risk connectome. Red line = organization score for risk-associated Pearson correlation in panel A.



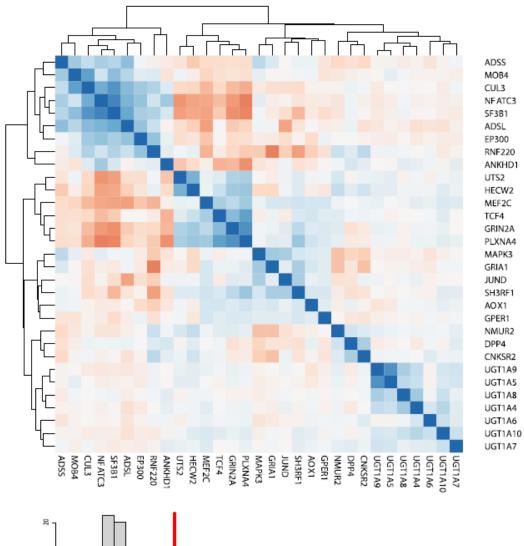
Neuron Risk-associated (PGC2) STRING, P < 10⁻⁶

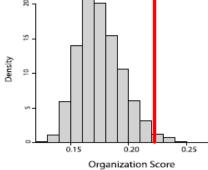
1 0.5 0

-05 -1

Figure 3.18 | Neuron schizophrenia risk-associated STRING subset genes 203 show greater transcriptional organization than full risk connectome gene list (PGC2).

(A) Pearson correlation heatmap of genes participating in neuron-specific STRING protein-protein network. Risk locus gene (red), gene present in TAD with no other genes in the network (grey circle), gene co-localized with one or more genes in same TAD or adjacent TADs (colored circles; each color represents a different TAD; see Table S24). (B) Frequency distribution of permuted organization scores, randomly sampling from all genes in neuron risk connectome. Red line = organization score for risk-associated Pearson correlation in panel A.





А

В

0.5

0

-0.5

-1

Figure 3.19 | Glia schizophrenia risk-associated STRING subset genes do not show greater transcriptional organization than full risk connectome gene list (PGC2).

(A) Pearson correlation heatmap of genes participating in glia-specific STRING protein-protein network. (B) Frequency distribution of permuted organization scores, randomly sampling from all genes in glia risk connectome. Red line = organization score for risk-associated Pearson correlation in panel A.

To examine whether such coregulation could be representative of the prefrontal cortex proteome of the adult brain, we screened a newly generated mass spectrometry-based dataset of 182 neuronal proteins, the majority of which were synaptic, quantified from prefrontal cortex of n = 23 adult schizophrenia and n = 20 control subjects (Table 3.22) (Hahn *et al.*, 2009). Among the 182 proteins, there were four from the risk-associated neuronal protein network (Figure 3.13, D): GABA_B receptor subunit GABBR1 and ionotropic (GRIA1 and GRIN2A) and metabotropic glutamate receptor subunits (GRM3). Protein-protein correlation scores were significantly higher for these four risk-associated proteins than expected from random permutation analysis from the pool of 182 proteins (P < 0.002) across patients and controls. We conclude that the schizophrenia-related chromosomal connectome, tethering other portions of the genome to the sequences associated with schizophrenia heritability, provides a structural foundation for a functional connectome that reflects coordinated regulation of gene expression and interactions within the proteome.

Discussion

Neural progenitor differentiation into neurons and glia is associated with dynamic remodeling of chromosomal conformations, including loss of many NPC-specific chromosomal contacts, with differentiation-induced loop pruning primarily affecting a subset of genes important for neurogenesis (NPC-to-neuron loss) and neuronal function (NPC-to-glia loss). These findings broadly resonate with a recent report linking neural differentiation to multiple scales of 3DG folding, governed by multiple mechanisms, including CTCF-dependent loop alterations, repressive chromatin remodeling, and cell- and lineage-specific transcription factor networks (Bonev et al., 2017). Our results suggest that developmental 3DG remodeling affects a substantial portion of sequences that confer liability for schizophrenia; furthermore, these genes in 3D physical proximity with schizophrenia-risk variants show a surprisingly strong correlation at the level of the transcriptome and proteome. How might the disease-relevant reorganization of the spatial genome (the "chromosomal connectome") provide a structural foundation for coordinated regulation of expression? Recent Hi-C studies in mouse brain showed that chromosomal contacts preferentially occurred between loci targeted by the same transcription factors (Bonev et al., 2017), and likewise, multiple schizophrenia risk loci could converge on intra- and interchromosomal hubs sharing a similar regulatory architecture including specific enhancers as well as transcription and splicing factors (Lomvardas et al., 2006; Khanna, Hu and Belmont, 2014; Quinodoz et al., 2018). Intriguingly, the three major

functional categories associated with the genetic risk architecture of schizophrenia—neuronal connectivity, synaptic signaling, and chromatin remodeling (Gilman et al., 2012; O'dushlaine et al., 2015)—were heavily represented within the cell type-specific chromosomal connectomes of neurons and NPCs described here (Fig. 3.10, B and E) and in whole tissue in vivo (Won et al., 2016; de la Torre-Ubieta et al., 2018). Cell type-specific 3DG reorganization during the course of neural progenitor differentiation, as shown here, could therefore have profound implications for our understanding of the genetic underpinnings of psychiatric disease. For example, inclusion of the cell type-specific risk (sequence) associated chromosomal connectome may lead to refinements of cumulative schizophrenia risk allele burden estimates, including "polygenic risk score" (PRS) or "biologically informed multilocus profile scores" (BIMPS), which currently only explain a small portion of disease risk (Bogdan, Baranger and Agrawal, 2018). Cell type–specific intersection of 3DG and genetic risk maps are of clinical interest beyond psychiatric disorders; for example, risk variants that confer susceptibility to autoimmune disease were embedded in physically interacting chromosomal loci in lymphoblastoid cells (Grubert et al., 2015). Our 3DG maps from neural progenitors and their isogenic neurons and glia are accessible through the PsychENCODE Knowledge Portal (https://synapse.org) and more than double the number of currently available Hi-C datasets from human brain (Dixon et al., 2015; Schmitt et al., 2016; Won et al., 2016), providing investigators with a resource to chart the expanded genome

space associated with cognitive and neuropsychiatric disease in context of cell 208 type–specific remodeling of chromosomal conformations during early development.

Tables

Hyperlink to Excel format of all tables (ChapterIII_Tables)

Hi-C

			-	-											
LIBRARY	TOTAL READS	ALIGNED READS	CHIMERIC READS	Duplicates	FR PAIRS	Valid cis interactions	Subsampled contacts	Loop Calling	5kb (Non- Sub) Loops	10kb (Non- Sub) Loops	5+10kb (Non- Sub) Loops	5kb (Sub) Loops	10kb (Sub) Loops	5+10kb (Sub) Loops	Accession ID
Neuron 1	171,049,419	87%	21%	2%	26%	372,787,143	372,787,143	Reevaluation	2661	5348	8009	1704	5502	7206	
Neuron 1	171,049,419	87%	21%	2%	26%	372,787,143	372,787,143	Standard HiCCUPS					3676		
Neuron 2 (Rep1)	191,616,387	89%	22%	3%	26%										
Neuron 2 (Rep2)	277,560,748	88%	22%	3%	26%										
FB Neuron 1	1,148,145,724	90%	38%	5%	24%	491,798,369	372,787,143	Standard HiCCUPS					5741		
Glia 1	166,079,544	87%	20%	6%	27%	440,950,529	372,787,143	Reevaluation	7289	9153	16442	4531	8453	12984	
Glia 1	166,079,544	87%	20%	6%	27%	440,950,529	372,787,143	Standard HiCCUPS					7103		
Glia 2 (Rep1)	211,708,831	88%	21%	4%	27%										
Glia 2 (Rep2)	312,440,552	86%	21%	3%	27%										
NPC 1	239,221,938	87%	19%	4%	31%	616,826,359	372,787,143	Reevaluation	9070	10941	20011	4584	8429	13013	
NPC 1	239,221,938	87%	19%	4%	31%	616,826,359	372,787,143	Standard HiCCUPS					7378		
NPC 2 (Rep1)	205,102,780	88%	20%	3%	28%										
NPC 2 (Rep 2)	251,568,531	84%	20%	2%	28%										
No Ligase NPC 1	77,814,521	87%	0%	14%	41%										
GM12878	3.587.190.419	ġ	e Rao et al.	(Ref 15)		1,989,154,220	372,787,143	Reevaluation	10281	11268	21549	2769	5309		GSE63525_ GM12878_ insitu_primary
Fetal	2,256,596,541			2016) (Ref. 6))	545,496,617	204,476,188	Standard HiCCUPS	10201	9309	21010	2100	4498		GZ(GSM2054567, GSM2054568, GSM2054569)
Fetal Cortical Plate	2,202,056,563					540,079,810	204,476,188	Standard HiCCUPS		9159			3921		CP (GSM2054564, GSM2054565, GSM2054566)
Mouse ESC	1.035.319.145	See Bo	onev et al. (2	2017) (Ref. 1	7)	471.503.539	471,503,539	Standard HiCCUPS		3496			3496		SRR5339748. .SRR5339753
Mouse NPC	1,284,813,522		"	,	/	619,932,857	471,503,539	Standard HiCCUPS		5896			4756		SRR5339786. SRR5339792
Mouse Cortical Neuron	1,298,855,792		"			615,843,081	471,503,539	Standard HiCCUPS		4774			3807		SRR5339829. .SRR5339835

Hi-C

PC	DOLED
CELL	TOTAL
TYPE	READS
Neurons	640,226,554
Glia	690,228,927
NPCs	695,893,249

ATAC-seq

Sample	Total # Reads	% Uniquely Mapped	non chrM/Duplicates	% Duplicates
NPC 2	134,380,774	99.02	74,493,904	36.33
Neuron 2 Rep 1	96,205,900	99.07	54,130,694	33.5
Neuron 2 Rep 2	81,385,524	99.2	46,418,801	32.46

Table 3.1 | Hi-C and ATAC-Seq sequencing summary and quality controls.

Sequencing and quality control metrics for Hi-C libraries and loops called by cell type. Total reads, number of unfiltered reads; FR pairs, forward-reverse pairs; *Loops are only called from contacts pooled by cell type

**Reevaluation= loops called from union set of initial loop calls (see Methods); Standard HiCCUPS= loop calls with no reevaluation

*** Non-Sub = all cis contacts passing filters; Sub= cis contacts randomly sampled from all cell types

		Term	Term PValue Corrected with Bonferroni				Total genes in	
GOID	GOTerm	PValue	step down	Neg log(P_corr)	% Associated	Nr. Genes	Ťerm	Associated Genes Found
	central nervous system							[ARSB, BASP1, CDH11, CENPF, CEP120, CEP290, CLU, DNER, EN1, EPHA4, FUT10, GARS, GNG12, GRIK1, HOXA2, HOXB2, IGF2BP1, INHBA, IRS2, KDMAA, KIRREL3, LHX8, MACROD2, MAP2, MKKS, MYO1D, NAV2, NCOA3, NR2F2, NRG1, NRP2, PBX3, PDHX, PITX2, POU3F2, PTCH1, PTN, PTPR21, RCAN1, ROBO1, RTN4RL1, SALL1, SEMA3A, SLITRK5, SOX2, SOX4,
GO:0007417	development	78.0E-9	21.0E-6	4.677780705	5.247225025	52	991	SPOCK1, SUDS3, TOP2B, TRA2B, ZBTB16, ZEB1]
GO:0035108	limb morphogenesis	750.0E-9	200.0E-6	3.698970004	10.45751634	16	153	[ALX1, B9D1, BMPR1B, CYP26B1, EN1, FMN1, HOXD12, MAP3K20, PITX2, PTCH1, SALL1, SALL4, SOX4, SP9, TGFB2, ZBTB16]
GO:0072001	renal system development	1.0E-6	270.0E-6	3.568636236	7.590759076	23	303	ADAMTS1, ADAMTS16, AGTR1, BASP1, CENPF, CEP290, COL4A4, DCN, EFNB2, EPHA4, FMN1, GARS, ITGA6, KIRREL3, NFIA, PTCH1, SALL1, SMAD2, SOX4, SULF1, SULF2, TGFB2, ZBTB16]
	cell morphogenesis involved in							[ADGRB3, ALCAM, ANK3, ART4, BMPR1B, BVES, CDH11, EPHA4, FELN1, FEZ1, FLRT3, FMN1, GAP43, GFRA1, HEG1, HOXA2, IRS2, KLF7, LATS1, MAP1B, MAP2, NCOA3, NRG1, NRP2, PARD3, PIK3R1, PLXNB1, POU3F2, PTPRZ1, RCC2, RGMA, ROBO1, S100A10, SDC2, SEMA3A, SLITRK5, STRC,
GO:0000904	differentiation	1.5E-6	410.0E-6	3.387216143	5.52407932	39	706	TGFB2, TOP2B]
GO:0061564	axon development	3.7E-6	970.0E-6	3.013228266	5.988023952	30	501	ALCAM, ANK3, ART4, BMPR1B, CDH11, EPH44, FEZ1, FLRT3, GAP43, GFRA1, HOXA2, IRS2, KLF7, MAP1B, NCAM2, NRG1, NRP2, PARD3, PCDHA4, PIK3R1, PLXNB1, POU3F2, PTPRZ1, RGMA, ROBO1, RTN4RL1, SEMA3A, SLITRK5, TGFB2, TOP2B]
								ADAMTS1, ADAMTS16, AGTR1, BASP1, CENPF, CEP290, COL4A4, DCN,
GO:0001822	kidney development	4.9E-6	1.2E-3	2.920818754	7.368421053	21	285	EFNB2, EPHA4, FMN1, GARS, KIRREL3, PTCH1, SALL1, SMAD2, SOX4, SULF1, SULF2, TGFB2, ZBTB16]
GO:0048667	cell morphogenesis involved in neuron differentiation	7.8E-6	2.0E-3	2.698970004	5.604203152	32	571	ADGRB3, ALCAM, ANK3, ART4, BMPR1B, CDH11, EPHA4, FEZ1, FLRT3, FMM1, GAP43, GFRA1, HOXA2, IRS2, KLF7, MAP1B, MAP2, NRG1, NRP2, PARD3, PIK3R1, PLXNB1, POU3F2, PTPRZ1, RGMA, ROBO1, SDC2, SEMA3A, SLITRK5, STRC, TGFB2, TOP2B]
GO:0030326	embryonic limb morphogenesis	15.0E-6	4.1E-3	2.387216143	9.848484848	13	132	[ALX1, B9D1, CYP26B1, EN1, HOXD12, MAP3K20, PITX2, PTCH1, SALL1, SALL4, SP9, TGFB2, ZBTB16]
GO:0007409	axonogenesis	17.0E-6	4.5E-3	2.346787486	5.844155844	27	462	ALCAM, ANK3, ART4, BMPR1B, CDH11, EPHA4, FEZ1, FLRT3, GAP43, GFRA1, HOXA2, IRS2, KLF7, MAP1B, NRG1, NRP2, PARD3, PIK3R1, PLXNB1, POU3F2, PTPR21, RGMA, ROBO1, SEMA3A, SLITRK5, TGFB2, TOP2BI
GO:0048812	neuron projection morphogenesis	18.0E-6	4.7E-3	2.327902142	5.331179321	33	610	ADGRB3, ALCAM, ANK3, ART4, BMPR1B, CDH11, EPHA4, FEZ1, FLRT3, FMM1, GAP43, GFRA1, HOXA2, IRS2, KIRREL3, KLF7, MAP1B, MAP2, NRG1, NRP2, PARD3, PIK3R1, PLXNB1, POU3F2, PTPR21, RGMA, RIMS2, ROBO1, SDC2, SEMA3A, SLITRK5, TGFB2, TOP2BI
60.0040012	neuron	10.02-0	4.72-3	2.327302142	5.551179521	55	013	
GO:0008038	recognition	27.0E-6	6.9E-3	2.161150909	18.42105263	7	38	EPHA4, GAP43, NCAM2, OPCML, PCDHA4, ROBO1, SEMA3A] CEP120, FUT10, GNG12, IGF2BP1, INHBA, KIRREL3, LHX8, MKKS, MYO1D,
GO:0030900	forebrain development	39.0E-6	10.0E-3	2	5.940594059	24	404	NCOA3, NR2F2, NRG1, NR2, P1742, POU3F2, PTN, ROBO1, RTN4RL1, SALL1, SEMA3A, SLITRK5, SOX2, TOP2B, TRA2B]
GO:0031053	primary miRNA processing	58.0E-6	14.0E-3	1.853871964	40		10	[GARS, HNRNPA2B1, NCBP1, SMAD2]
GO:0007507	heart development	80.0E-6		1.698970004	5.263157895			ADAMTS1, BASP1, BVES, CALCRL, CASP7, DAND5, EFNB2, FLRT3, GARS, HDAC9, HEG1, KCNK2, KDM6A, MKKS, NEBL, NRG1, NRP2, PITX2, PRICKLE1, PTCH1, PTN, ROB01, SALL1, SALL4, SMAD2, SOX4, SUMO1, TGFB2. ZMZ11
	cation channel				0.2001070000			CACNB2, CLU, FAM155A, GRIK1, GRIN2B, HCN4, ITPR2, KCND2, KCNE2, KCNH1, KCNK16, KCNK2, KCNMB1, KCNS1, NALCN, SCN1A, SCN9A,
GO:0005261	activity	94.0E-6	23.0E-3	1.638272164	6.211180124	20	322	TRPM3, TRPM4, TRPM8]
GO:0005272	sodium channel activity	120.0E-6	32.0E-3	1.494850022	14.58333333	7	48	[CLU, GRIK1, HCN4, NALCN, SCN1A, SCN9A, TRPM4]
GO:0005216	ion channel activity	150.0E-6	37.0E-3	1.431798276	5.517241379	24	435	CACNB2, CLCN4, CLU, FAM155A, GABRP, GRIK1, GRIN2B, HCN4, ITPR2, KCND2, KCNE2, KCNH1, KCNK16, KCNK2, KCNMB1, KCNS1, NALCN, SCN1A, SCN9A, SLC2GA2, SLC2GA7, TRPM3, TRPM4, TRPMB]
GO:0048706	embryonic skeletal system development	190.0E-6	48.0E-3	1.318758763	8.8	11	125	[ACVR2A, ALX1, HOXA2, HOXA5, HOXB2, HOXB6, IRX5, SMAD2, SULF1, SULF2, ZEB1]

Table 3.2 | Gene ontology (GO) of Brain-specific loops.

Gene ontology enrichment terms and details for genes in brain-specific loops. GOID, ClueGO term ID; Term PValue, uncorrected p-value; Term PValue Corrected with Bonferroni step down, p-value corrected for multiple comparisons; Neg log(P_corr), negative log-transformed corrected p-values; % Associated, percentage of genes in GO category found in input list of genes; Nr. Genes, number of genes from input list that match the GO category; Total genes in Term, number of genes present in the term according to ClueGo.

GOID	GOTerm	Neg log (P)	Ontology Source	Term PValue		negative log(P)	% Associated Genes		Total genes in term	Associated Genes Found
GOID	embryonic digestive tract		GO_BiologicalProcess-	r value	with Dometroni step down	iog(F)	Genes	Genes	Intern	PCSK5, PDGFRA,
GO:0048566	development		GOA 23.02.2017 10h01	140.0E-6	4.0E-3	2.4	11.43		34	SHH, SHOX2]
00.0010000	dereiepinent		GO BiologicalProcess-	110.02 0	HOE O	2.1				IDMRTA2, GATA2.
GO:0048663	neuron fate commitment		GOA_23.02.2017_10h01	220.0E-6	5.8E-3	2.2	7.04	5	7.	[GBX1, SATB2, SHH]
	positive regulation of neural	1.958607	GO_BiologicalProcess-							[DMRTA2, EGF,
GO:2000179	precursor cell proliferation	315	GOA_23.02.2017_10h01	470.0E-6	11.0E-3	2.0	8.51	4	4	INSM1, SHH]
	negative regulation of		GO_BiologicalProcess-							
GO:0090370	cholesterol efflux		GOA_23.02.2017_10h01	580.0E-6	14.0E-3	1.9	40.00	2		[EGF, SHH]
	modulation by virus of host		GO_BiologicalProcess-							
GO:0019054	process	705	GOA_23.02.2017_10h01	920.0E-6	21.0E-3	1.7	12.00	3	28	[ATG7, KPNA7, VAPA]
	commitment of neuronal cell	4 000050								
GO:0021902	to specific neuron type in forebrain		GO_BiologicalProcess- GOA 23.02.2017 10h01	1.2E-3	25.0E-3	1.6	28.57		-	IGATA2. SATB21
00.0021302	histone methyltransferase		GO MolecularFunction-	1.2L-C	23.02-3	1.0	20.57		· · · · ·	[PRMT8, SETD3,
GO:0042054	activity		GOA 23.02.2017 10h01	1.2E-3	26.0E-3	1.6	6.67	· 4	60	SMYD2, SMYD3
00.00 1200 1	doutily	1.508638			E010E 0		0.01			011102, 011100]
GO:0003030	DNA replication		KEGG 01.03.2017	31.0E-3	31.0E-3	1.5	5.56	2	36	[MCM6, POLA2]
	positive regulation of									
	cerebellar granule cell	1.387216	GO_BiologicalProcess-							
GO:0021940	precursor proliferation		GOA_23.02.2017_10h01	2.0E-3	41.0E-3	1.4	22.22	2	9	EGF, SHH]
	negative regulation of focal		GO_BiologicalProcess-							
GO:0051895	adhesion assembly		GOA_23.02.2017_10h01	10.0E-3	41.0E-3	1.4	10.00	2	20	[BCAS3, RCC2]
	mammary gland alveolus		GO_BiologicalProcess-							
GO:0060749	development		GOA_23.02.2017_10h01	10.0E-3	41.0E-3	1.4	10.00	2	20	[EGF, ERBB4]
00 00 107 17	and the fill of the state of th		GO_BiologicalProcess-	0.05.0	10.05.0					[HDAC9, MYO18B,
GO:0048747	muscle fiber development		GOA_23.02.2017_10h01	9.3E-3	46.0E-3	1.3	5.36	3	56	SHOX2]
00.0045050	positive thymic T cell		GO_BiologicalProcess-	0.55.4	10.05.0		20.00			
GO:0045059	selection	763	GOA_23.02.2017_10h01	2.5E-3	48.0E-3	1.3	20.00	2	10	[DOCK2, SHH]

Table 3.3 | Neuron-specific loops GO.Gene ontology enrichment terms and details for genes in neuron-specific loops(See Table 3.2 for more information)

GOID	COTorm	Neg log(P) Ontology Source	Term PValue	Term PValue Corrected with Bonferroni step down	% Associated Genes		Total genes in
	GOTerm	log(P) Ontology Source 18.397GO_BiologicalProcess-	PValue 260.0E-	·			Term
	neuron differentiation	94GOA_23.02.2017_10h01 17.267GO_BiologicalProcess-	24	400.0E-21		210.00	1333.0
GO:0022008	neurogenesis	606GOA_23.02.2017_10h01 17.040GO_BiologicalProcess-	3.5E-21	5.4E-18		232.00	1562.0
GO:0048699	generation of neurons	959 GOA_23.02.2017_10h01 15.229 GO_BiologicalProcess-	6.0E-21 390.0E-	9.1E-18	15.10	221.00	1464.0
GO:0007417	central nervous system development	148GOA_23.02.2017_10h01 GO_BiologicalProcess-	21	590.0E-18	16.45	163.00	991.0
GO:0048812	neuron projection morphogenesis	13GOA_23.02.2017_10h01 12.853GO_BiologicalProcess-	71.0E-18	100.0E-15	18.26	113.00	619.0
GO:0048666	neuron development	872GOA_23.02.2017_10h01	93.0E-18	140.0E-15	15.51	165.00	1064.0
GO:0031175	neuron projection development	12.537GO_BiologicalProcess- 602GOA_23.02.2017_10h01	190.0E- 18	290.0E-15	16.11	146.00	906.0
GO:0097485	neuron projection guidance	11.958GO_BiologicalProcess- 607GOA_23.02.2017_10h01	730.0E- 18	1.1E-12	24.90	61.00	245.0
GO:0048667	cell morphogenesis involved in neuron differentiation	11.221GO_BiologicalProcess- 849GOA_23.02.2017_10h01	4.0E-15	6.0E-12	18.04	103.00	571.0
GO:0007420	brain development	11.055GO_BiologicalProcess- 517GOA_23.02.2017_10h01	5.8E-15	8.8E-12	16.56	125.00	755.0
GO:0000904	cell morphogenesis involved in differentiation	10.221GO_BiologicalProcess- 849GOA_23.02.2017_10h01	39.0E-15	60.0E-12	16.57	117.00	706.0
	axonogenesis	9.7212GO_BiologicalProcess- 464GOA_23.02.2017_10h01	130.0E- 15	190.0E-12	18.61	86.00	462.0
	axon development	9.6020 GO_BiologicalProcess- 6GOA_23.02.2017_10h01	160.0E-	250.0E-12	18.16		501.0
GO:0006357	regulation of transcription from RNA polymerase II	9.0757GO_BiologicalProcess-	550.0E- 15	840.0E-12		249.00	1999.0
		207GOA_23.02.2017_10h01 9.0268GO_BiologicalProcess-	620.0E				
	transcription from RNA polymerase II promoter	721GOA_23.02.2017_10h01 8.3872GO_BiologicalProcess-	15	940.0E-12		269.00	2205.0
GO:0010628	positive regulation of gene expression	161GOA_23.02.2017_10h01 8.2218GO_BiologicalProcess-	2.7E-12	4.1E-9	12.53	230.00	1836.0
GO:0016055	Wnt signaling pathway cell surface receptor signaling pathway involved in	488GOA_23.02.2017_10h01 8.0861GO_BiologicalProcess-	4.0E-12	6.0E-9	17.36	88.00	507.0
GO:1905114	cell-cell signaling	861 GOA_23.02.2017_10h01 8.0604 GO_BiologicalProcess-	5.4E-12	8.2E-9	16.32	101.00	619.0
GO:0051254	positive regulation of RNA metabolic process positive regulation of transcription from RNA	807GOA_23.02.2017_10h01 GO_BiologicalProcess-	5.8E-12	8.7E-9	12.93	199.00	1539.0
GO:0045944	polymerase II promoter	8GOA_23.02.2017_10h01 7.9208GO_BiologicalProcess-	6.9E-12	10.0E-9	13.85	158.00	1141.0
GO:0045893	positive regulation of transcription, DNA-templated	188GOA_23.02.2017_10h01	8.2E-12	12.0E-9	13.04	192.00	1472.0
GO:0021953	central nervous system neuron differentiation	7.6197GO_BiologicalProcess- 888GOA_23.02.2017_10h01	16.0E-12	24.0E-9	23.71	46.00	194.0
GO:0045664	regulation of neuron differentiation	7.5686GO_BiologicalProcess- 362GOA_23.02.2017_10h01	18.0E-12	27.0E-9	16.30	97.00	595.0
GO:0045935	positive regulation of nucleobase-containing compound metabolic process	7.5086GO_BiologicalProcess- 383GOA_23.02.2017_10h01	20.0E-12	31.0E-9	12.36	224.00	1813.0
GO:0050767	regulation of neurogenesis	7.0043GO_BiologicalProcess- 648GOA_23.02.2017_10h01	66.0E-12	99.0E-9	15.12	111.00	734.0
GO:0051960	regulation of nervous system development	6.9208GO_BiologicalProcess- 188GOA_23.02.2017_10h01	84.0E-12	120.0E-9	14.65	121.00	826.0
	positive regulation of cellular biosynthetic process	6.5686GO_BiologicalProcess- 362GOA_23.02.2017_10h01	180.0E- 12	270.0E-9		229.00	1905.0
	canonical Wnt signaling pathway	6.2218 GO_BiologicalProcess- 488 GOA_23.02.2017_10h01	400.0E-	600.0E-9	19.22		307.0
GO:0010557	positive regulation of macromolecule biosynthetic	6.0915GO_BiologicalProcess-	540.0E- 12	810.0E-9			
		15GOA_23.02.2017_10h01 5.9586GO_BiologicalProcess-	760.0E-			212.00	1751.0
	forebrain development	073GOA_23.02.2017_10h01 5.8860GO_BiologicalProcess-	12 920.0E	1.1E-6	17.33		404.0
	regulation of cell development	566GOA_23.02.2017_10h01 5.7958GO_MolecularFunction-	12	1.3E-6		120.00	851.0
	transcription regulatory region DNA binding	8GOA_23.02.2017_10h01 5.7212GO_BiologicalProcess-	1.1E-9	1.6E-6	14.13	119.00	842.0
GO:0009887	animal organ morphogenesis	464GOA_23.02.2017_10h01 5.7212GO_BiologicalProcess-	1.2E-9	1.9E-6	13.38	140.00	1046.0
GO:0009887	animal organ morphogenesis	464GOA_23.02.2017_10h01 4.8538GO BiologicalProcess-	1.2E-9	1.9E-6	13.38	140.00	1046.0
GO:0030111	regulation of Wnt signaling pathway negative regulation of transcription from RNA	72GOA_23.02.2017_10h01 4.8239GO BiologicalProcess-	9.6E-9	14.0E-6	17.66	59.00	334.0
GO:0000122	polymerase II promoter	087GOA_23.02.2017_10h01	10.0E-9	15.0E-6	13.83	112.00	810.0
GO:0021954	central nervous system neuron development	4.4317GO_BiologicalProcess- 983GOA_23.02.2017_10h01	25.0E-9	37.0E-6	29.49	23.00	78.0
GO:2000113	negative regulation of cellular macromolecule biosynthetic process	4.2924GO_BiologicalProcess- 298GOA_23.02.2017_10h01	35.0E-9	51.0E-6	12.15	168.00	1383.0
GO:0010558		4.1674GO_BiologicalProcess- 911GOA_23.02.2017_10h01	46.0E-9	68.0E-6	11.98	176.00	1469.0
GO:0000976	transcription regulatory region sequence-specific DNA binding	4.1487GO_MolecularFunction- 417GOA_23.02.2017_10h01	48.0E-9	71.0E-6	14.18	96.00	677.0
	sequence-specific double-stranded DNA binding	4.1191 GO_MolecularFunction- 864 GOA_23.02.2017_10h01	51.0E-9	76.0E-6		100.00	717.0
GO:0090287	regulation of cellular response to growth factor	3.9586GO_BiologicalProcess- 073GOA_23.02.2017_10h01	80.0E-9	110.0E-6	18.36		256.0
		3.8860GO_BiologicalProcess-					
30:0045596	negative regulation of cell differentiation	566GOA_23.02.2017_10h01 3.7958GO_BiologicalProcess-	88.0E-9	130.0E-6	14.09	94.00	667.0

		3.7695GO_BiologicalProcess-	<u> </u>				
GO:0045892	negative regulation of transcription, DNA-templated RNA polymerase II regulatory region sequence-	511GOA_23.02.2017_10h01 3.7447GO_MolecularFunction-	110.0E-9	170.0E-6	12.39	145.00	1170.00
GO:0000977	specific DNA binding	275GOA_23.02.2017_10h01 3.7212	120.0E-9	180.0E-6	14.41	85.00	590.00
GO:0004360	Axon guidance	464 KEGG_01.03.2017 3.7212 GO_BiologicalProcess-	130.0E-9	190.0E-6	20.57	36.00	175.00
GO:0035108	limb morphogenesis	464GOA_23.02.2017_10h01 3.6989GO_BiologicalProcess-	130.0E-9	190.0E-6	21.57	33.00	153.00
GO:0010721	negative regulation of cell development	7GOA_23.02.2017_10h01	130.0E-9	200.0E-6	17.33	52.00	300.00
GO:1903507	negative regulation of nucleic acid-templated transcription	3.6989GO_BiologicalProcess- 7GOA_23.02.2017_10h01	140.0E-9	200.0E-6	12.27	149.00	1214.00
GO:0051253	negative regulation of RNA metabolic process	3.6197GO_BiologicalProcess- 888GOA_23.02.2017_10h01	160.0E-9	240.0E-6	12.10	155.00	1281.00
GO:0051961	negative regulation of nervous system development	3.5528GO_BiologicalProcess- 42GOA_23.02.2017_10h01	190.0E-9	280.0E-6	17.50	49.00	280.00
GO:0031327	negative regulation of cellular biosynthetic process	3.3665GO_BiologicalProcess- 315GOA_23.02.2017_10h01	290.0E-9	430.0E-6	11.60	180.00	1552.00
GO:0045597	positive regulation of cell differentiation	3.2839GO_BiologicalProcess- 967GOA 23.02.2017 10h01	350.0E-9	520.0E-6	12.84	117.00	911.0
	regulation of canonical Wnt signaling pathway	3.1191GO_BiologicalProcess- 864GOA_23.02.2017_10h01	520.0E-9	760.0E-6	17.72	45.00	254.0
GO:0033267		2.9586GO_CellularComponent- 073GOA_23.02.2017_10h01	780.0E-9	1.1E-3	18.72	38.00	203.0
		2.9586GO_BiologicalProcess-					
	regulation of neuron projection development negative regulation of nucleobase-containing	073GOA_23.02.2017_10h01 2.8860GO_BiologicalProcess-	800.0E-9	1.1E-3	14.97	66.00	441.0
	compound metabolic process	566GOA_23.02.2017_10h01 2.8538GO_BiologicalProcess-	950.0E-9	1.3E-3	11.59		1441.0
O:0001764	neuron migration	72GOA_23.02.2017_10h01 2.8239GO_BiologicalProcess-	1.0E-6	1.4E-3	20.53	31.00	151.0
O:0030178	negative regulation of Wnt signaling pathway	087GOA_23.02.2017_10h01 2.8239GO BiologicalProcess-	1.0E-6	1.5E-3	18.57	39.00	210.0
O:0010629	negative regulation of gene expression	087GOA_23.02.2017_10h01 2.7212GO CellularComponent-	1.0E-6	1.5E-3	11.23	194.00	1727.0
GO:0044304	main axon	464GOA_23.02.2017_10h01 2.6197GO BiologicalProcess-	1.3E-6	1.9E-3	27.54	19.00	69.0
O:0061035	regulation of cartilage development	888GOA_23.02.2017_10h01	1.6E-6	2.4E-3	27.14	19.00	70.0
O:0048588	developmental cell growth	2.6197GO_BiologicalProcess- 888GOA_23.02.2017_10h01	1.6E-6	2.4E-3	18.45	38.00	206.0
O:2000112	regulation of cellular macromolecule biosynthetic process	2.6197GO_BiologicalProcess- 888GOA_23.02.2017_10h01	1.6E-6	2.4E-3	9.87	411.00	4166.0
O:0015630	microtubule cytoskeleton	2.5086GO_CellularComponent- 383GOA_23.02.2017_10h01	2.1E-6	3.1E-3	11.94	136.00	1139.0
O:0044428	nuclear part	2.4814GO_CellularComponent- 861GOA_23.02.2017_10h01	2.3E-6	3.3E-3	9.77	431.00	4411.0
	negative regulation of neuron differentiation	2.4089GO_BiologicalProcess- 354GOA_23.02.2017_10h01	2.7E-6	3.9E-3	18.23	37.00	203.0
	core promoter proximal region sequence-specific DNA binding	2.3665 GO_MolecularFunction- 315 GOA_23.02.2017_10h01	2.9E-6	4.3E-3	15.20	57.00	375.0
	regulation of RNA metabolic process	2.3565 GO_BiologicalProcess- 473 GOA_23.02.2017_10h01	3.0E-6	4.4E-3		389.00	3933.0
	negative regulation of canonical Wnt signaling	2.2757GO_BiologicalProcess-					
	pathway	241 GOA_23.02.2017_10h01 2.2518 GO_CellularComponent-	3.7E-6	5.3E-3	18.86	33.00	175.0
	nuclear lumen RNA polymerase II core promoter proximal region	12GOA_23.02.2017_10h01 2.1191GO_MolecularFunction-	3.9E-6	5.6E-3		398.00	4045.0
GC:0000978	sequence-specific DNA binding	864GOA_23.02.2017_10h01 2.1079GO_BiologicalProcess-	5.3E-6	7.6E-3	15.15	55.00	363.0
GO:0030901	midbrain development	054 GOA_23.02.2017_10h01 2.0969 GO_BiologicalProcess-	5.4E-6	7.8E-3	21.82	24.00	110.0
GO:0006355	regulation of transcription, DNA-templated	1GOA_23.02.2017_10h01 2.0555GO_BiologicalProcess-	5.5E-6	8.0E-3	9.89	372.00	3763.0
GO:0016070	RNA metabolic process	173GOA_23.02.2017_10h01 2.0555GO_BiologicalProcess-	6.0E-6	8.8E-3	9.61	462.00	4809.0
GO:0016070	RNA metabolic process	173GOA_23.02.2017_10h01	6.0E-6	8.8E-3	9.61	462.00	4809.0
O:1990138	neuron projection extension	2.0222GO_BiologicalProcess- 764GOA_23.02.2017_10h01	6.5E-6	9.5E-3	19.61	30.00	153.0
O:1903506	regulation of nucleic acid-templated transcription	2.0043GO_BiologicalProcess- 648GOA_23.02.2017_10h01	6.9E-6	9.9E-3	9.87	374.00	3791.0
O:0032330	regulation of chondrocyte differentiation	GO_BiologicalProcess- 2GOA_23.02.2017_10h01	6.9E-6	10.0E-3	29.41	15.00	51.0
O:0051962	positive regulation of nervous system development	1.9208GO_BiologicalProcess- 188GOA 23.02.2017 10h01	8.5E-6	12.0E-3	13.80	69.00	500.0
O:0005829	cvtosol	1.8860GO_CellularComponent- 566GOA_23.02.2017_10h01	9.0E-6	13.0E-3		475.00	4982.0
		1.8860GO_BiologicalProcess-	9.1E-6		17.99		189.0
	respiratory tube development	566GOA_23.02.2017_10h01 1.8538GO_CellularComponent- 72COA_23_02_2017_10h01		13.0E-3		34.00	
O:0005634		72GOA_23.02.2017_10h01 1.8538GO_CellularComponent-	10.0E-6	14.0E-3		665.00	7258.0
O:0005634		72GOA_23.02.2017_10h01 1.7695GO_BiologicalProcess-	10.0E-6	14.0E-3		665.00	7258.0
	transcription, DNA-templated	511GOA_23.02.2017_10h01 1.7212GO_BiologicalProcess-	12.0E-6	17.0E-3		384.00	3930.0
O:0021872	forebrain generation of neurons	464GOA_23.02.2017_10h01 1.6989GO_CellularComponent-	13.0E-6	19.0E-3	24.66	18.00	73.0
GO:0070013	intracellular organelle lumen	7GOA_23.02.2017_10h01 1.6777GO BiologicalProcess-	14.0E-6	20.0E-3	9.48	485.00	5118.0
GO:0048568	embryonic organ development	807 GOA_23.02.2017_10h01 1.6777 GO BiologicalProcess-	14.0E-6	21.0E-3	13.97	63.00	451.0
O:0021537	telencephalon development	807GOA_23.02.2017_10h01	15.0E-6	21.0E-3	16.09	42.00	261.0
GO:0045666	positive regulation of neuron differentiation	1.6777GO_BiologicalProcess- 807GOA_23.02.2017_10h01	14.0E-6	21.0E-3	14.90	52.00	349.0
GO:0005856	cytoskeleton	1.6382GO_CellularComponent- 722GOA_23.02.2017_10h01	16.0E-6	23.0E-3	10.51	224.00	2131.0
GO:0051216	cartilage development	1.6382GO_BiologicalProcess- 722GOA_23.02.2017_10h01	16.0E-6	23.0E-3	17.35	34.00	196.0
		1.6020GO_BiologicalProcess- 6GOA_23.02.2017_10h01					

			O_BiologicalProcess-					
GO:0061036	positive regulation of cartilage development		OA_23.02.2017_10h01	23.0E-6	32.0E-3	34.38	11.00	32.00
			O_BiologicalProcess-					
GO:0007423	sensory organ development		OA_23.02.2017_10h01	23.0E-6	33.0E-3	13.20	73.00	553.00
			O_BiologicalProcess-					
	positive regulation of neurogenesis		OA_23.02.2017_10h01	23.0E-6	33.0E-3	13.96	61.00	437.00
	regulation of cell morphogenesis involved in		O_BiologicalProcess-					
GO:0010769	differentiation		OA_23.02.2017_10h01	24.0E-6	35.0E-3	15.69	43.00	274.00
			O_BiologicalProcess-					
GO:0030334	regulation of cell migration		OA_23.02.2017_10h01	28.0E-6	40.0E-3	12.45	91.00	731.00
			O_BiologicalProcess-					
GO:2000027	regulation of organ morphogenesis		OA_23.02.2017_10h01	28.0E-6	40.0E-3	15.52	43.00	277.00
			O_BiologicalProcess-					
GO:2000027	regulation of organ morphogenesis		OA_23.02.2017_10h01	28.0E-6	40.0E-3	15.52	43.00	277.00
		1.3665						
GO:0004310	Wnt signaling pathway		EGG_01.03.2017	30.0E-6	43.0E-3	18.88	27.00	143.00
			O_BiologicalProcess-					
GO:0032774	RNA biosynthetic process	021G0	OA_23.02.2017_10h01	33.0E-6	47.0E-3	9.64	393.00	4077.00

B

				Term PValue						
			Term	Corrected with Benjamini-	negative		Group PValue Corrected			% Associated
	GOTerm single-organism organelle	Ontology Source GO_BiologicalProcess-	PValue	Hochberg	log(FDR)	PValue	with Benjamini-Hochberg	GOLevels	ps	Genes
GO:1902589	organization	GOA_23.02.2017_10h01 GO_BiologicalProcess-	26.0E-12	12.0E-9	7.9E+0	1.7E-9	12.0E-9	[3, 4]	Group11	4.57
	cell development cell morphogenesis	GOA_23.02.2017_10h01 GO_BiologicalProcess-	130.0E-12	21.0E-9	7.7E+0	57.0E-12	790.0E-12	[3, 4, 5]	Group13	4.24
GO:0000904	involved in differentiation cell morphogenesis	GOA_23.02.2017_10h01 GO BiologicalProcess-	110.0E-12	27.0E-9	7.6E+0	2.5E-9	12.0E-9	[4, 5, 6]	Group10	6.23
	involved in differentiation	GOA_23.02.2017_10h01	110.0E-12	27.0E-9	7.6E+0	57.0E-12	790.0E-12		Group13	6.23
GO:0007409	axonogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	13.0E-9	1.2E-6	5.9E+0	2.5E-9	12.0E-9	[6, 7, 8, 9, 10, 11, 12]	Group10	6.71
GO:0048699	generation of neurons	GO_BiologicalProcess- GOA_23.02.2017_10h01	18.0E-9	1.2E-6	5.9E+0	57.0E-12	790.0E-12		Group13	4.37
GO:0007409	axonogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	13.0E-9	1.2E-6	5.9E+0	57.0E-12	790.0E-12	[6, 7, 8, 9, 10, 11, 12]	Group13	6.71
GO:0050770	regulation of axonogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	11.0E-9	1.3E-6	5.9E+0	57.0E-12	790.0E-12	[6, 7, 8, 9, 10, 11, 12, 13]	Group13	10.98
GO:0032989	cellular component morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	18.0E-9	1.4E-6	5.9E+0	57.0E-12	790.0E-12	[3, 4]	Group13	4.83
GO:0045595	regulation of cell differentiation	GO_BiologicalProcess- GOA_23.02.2017_10h01	30.0E-9	1.4E-6	5.9E+0	57.0E-12	790.0E-12	[3, 4, 5]	Group13	4.20
GO:0030182	neuron differentiation	GO_BiologicalProcess- GOA 23.02.2017 10h01	23.0E-9	1.4E-6	5.9E+0	57.0E-12	790.0E-12	[5, 7, 8]	Group13	4.50
	cell morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	29.0E-9	1.5E-6	5.8E+0	57.0E-12	790.0E-12	[4, 5]	Group13	4.97
	regulation of multicellular organismal development	GO_BiologicalProcess- GOA_23.02.2017_10h01	58.0E-9	1.9E-6	5.7E+0	57.0E-12	790.0E-12		Group13	
		GO_BiologicalProcess- GOA 23.02.2017 10h01	52.0E-9	1.9E-6	5.7E+0	57.0E-12	790.0E-12			4.23
	neurogenesis cell morphogenesis involved in neuron differentiation	GO_BiologicalProcess- GOA_23.02.2017_10h01	50.0E-9	2.0E-6	5.7E+0	2.5E-9	12.0E-9	[5, 6, 7, 8,	Group13 Group10	
	cell projection organization	GOA_23.02.2017_10101 GO_BiologicalProcess- GOA_23.02.2017_10h01	47.0E-9	2.0E-6	5.7E+0	57.0E-12	790.0E-12		Group13	4.32
	cell morphogenesis		47.02-3	2.02-0	3.7L+0	57.0L-12	7 50.0L-12		Group 13	4.32
	involved in neuron differentiation	GO_BiologicalProcess- GOA_23.02.2017_10h01	50.0E-9	2.0E-6	5.7E+0	57.0E-12	790.0E-12		Group13	5.95
	neuron projection morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	110.0E-9	3.3E-6	5.5E+0	2.5E-9	12.0E-9	[5, 6, 7, 8, 10, 11]	Group10	5.65
	regulation of neurogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	100.0E-9	3.3E-6	5.5E+0	57.0E-12	790.0E-12	[5, 6, 7, 8]	Group13	5.31
	neuron projection morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	110.0E-9	3.3E-6	5.5E+0	57.0E-12	790.0E-12	[5, 6, 7, 8, 10, 11]	Group13	5.65
GO:0032990	cell part morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	130.0E-9	3.4E-6	5.5E+0	2.5E-9	12.0E-9	[4, 5]	Group10	5.51
GO:0032990	cell part morphogenesis	GO_BiologicalProcess- GOA 23.02.2017 10h01	130.0E-9	3.4E-6	5.5E+0	57.0E-12	790.0E-12	[4, 5]	Group13	5.51
	regulation of neuron	GO_BiologicalProcess- GOA_23.02.2017_10h01	130.0E-9	3.5E-6	5.5E+0	57.0E-12	790.0E-12	[6, 7, 8, 9]	Group13	5.71
	regulation of nervous system development	GO_BiologicalProcess- GOA_23.02.2017_10h01	120.0E-9	3.6E-6	5.4E+0	57.0E-12	790.0E-12		Group13	5.08
	cytoskeleton organization	GO_BiologicalProcess- GOA 23.02.2017 10h01	170.0E-9	4.0E-6	5.4E+0	1.7E-9	12.0E-9	[4, 0, 0]	Group11	4.42
		GO_BiologicalProcess-						[4, 5, 6, 8,		
	neuron development regulation of cell	GO_BiologicalProcess-	210.0E-9	5.0E-6	5.3E+0	57.0E-12	790.0E-12		Group13	4.61
GO:0060284	regulation of cell	GOA_23.02.2017_10h01	260.0E-9	5.6E-6	5.3E+0	57.0E-12	790.0E-12	[4, 5, 6]	Group13	4.94
GO:0010769	morphogenesis involved in differentiation	GO_BiologicalProcess- GOA_23.02.2017_10h01 GO_BiologicalProcess-	360.0E-9	7.5E-6	5.1E+0	57.0E-12	790.0E-12	[5, 6, 7]	Group13	7.66
GO:0090132	epithelium migration neuron projection	GOA_23.02.2017_10h01 GO_BiologicalProcess-	440.0E-9	8.9E-6	5.1E+0	750.0E-9	2.1E-6	[4] [4, 5, 6, 7,	Group12	7.87
GO:0031175		GOA_23.02.2017_10h01 GO BiologicalProcess-	570.0E-9	11.0E-6	5.0E+0	2.8E-9	9.8E-9		Group09	4.75
GO:0031175	development	GOA_23.02.2017_10h01	570.0E-9	11.0E-6	5.0E+0	57.0E-12	790.0E-12	9, 10]	Group13	4.75
	regulation of neuron projection development	GO_BiologicalProcess- GOA_23.02.2017_10h01	730.0E-9	13.0E-6	4.9E+0	57.0E-12	790.0E-12	[5, 6, 7, 8, 9, 10, 11]	Group13	6.12
	regulation of locomotion	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.0E-6	17.0E-6	4.8E+0	750.0E-9	2.1E-6	[2, 3]	Group12	4.77
GO:0030036		GO_BiologicalProcess- GOA_23.02.2017_10h01	1.2E-6	21.0E-6	4.7E+0	1.7E-9	12.0E-9	[4, 5]	Group11	5.27
	regulation of cell morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.4E-6	23.0E-6	4.6E+0	57.0E-12	790.0E-12		Group13	5.91

GO:0016477	cell migration	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.5E-6	24.0E-6	4.6E+0	2.5E-9	12.0E-9	[3, 4, 5]	Group10	4.0
	, , , , , , , , , , , , , , , , , , ,	GO_BiologicalProcess-				750.0E-9				4.0
0:0016477	cell migration regulation of	GOA_23.02.2017_10h01	1.5E-6	24.0E-6	4.6E+0	750.0E-9	2.1E-6	[3, 4, 5]	Group12	4.0
O:1902903	supramolecular fiber organization	GO_BiologicalProcess- GOA_23.02.2017_10h01	3.5E-6	55.0E-6	4.3E+0	1.7E-9	12.0E-9	[4, 5]	Group11	6.
	regulation of anatomical	GO_BiologicalProcess-								
	structure morphogenesis Axon guidance	GOA_23.02.2017_10h01 KEGG 01.03.2017	3.8E-6 4.3E-6	57.0E-6 61.0E-6	4.2E+0 4.2E+0	57.0E-12 2.5E-9	790.0E-12 12.0E-9		Group13 Group10	4.:
O:0030029	actin filament-based	GO_BiologicalProcess- GOA 23.02.2017 10h01	4.2E-6	61.0E-6	4.2E+0	1.7E-9	12.0E-9	101	Croup11	4.
	Axon guidance	KEGG_01.03.2017	4.2E-0 4.3E-6	61.0E-6	4.2E+0 4.2E+0	57.0E-12	790.0E-12		Group11 Group13	4.
O:2000145	regulation of cell motility	GO_BiologicalProcess- GOA 23.02.2017 10h01	4.7E-6	64.0E-6	4.2E+0	750.0E-9	2.1E-6	[3, 4, 5]	Group12	4.
	Proteoglycans in cancer	KEGG_01.03.2017	6.2E-6	83.0E-6	4.1E+0	6.2E-6	14.0E-6		Group03	7.
O:0051270	regulation of cellular component movement	GO_BiologicalProcess- GOA_23.02.2017_10h01	6.8E-6	88.0E-6	4.1E+0	750.0E-9	2.1E-6	[3, 4]	Group12	4.
O:0008092	cytoskeletal protein binding	GO_MolecularFunction- GOA 23.02.2017 10h01	7.0E-6	89.0E-6	4.1E+0	1.7E-9	12.0E-9	[3]	Group11	4.
		GO_CellularComponent-								
0:0043005	neuron projection regulation of epithelial cell	GOA_23.02.2017_10h01 GO_BiologicalProcess-	10.0E-6	120.0E-6	3.9E+0	2.8E-9	9.8E-9	[3, 4] [3, 4, 5, 6,	Group09	4.
O:0010632	migration	GOA_23.02.2017_10h01	9.8E-6	120.0E-6	3.9E+0	750.0E-9	2.1E-6		Group12	8.
O:0043005	neuron projection	GO_CellularComponent- GOA_23.02.2017_10h01	10.0E-6	120.0E-6	3.9E+0	57.0E-12	790.0E-12	[3, 4]	Group13	4.:
0.0003779	actin binding	GO_MolecularFunction- GOA_23.02.2017_10h01	11.0E-6	130.0E-6	3.9E+0	1.7E-9	12.0E-9	[4]	Group11	5.0
	regulation of cell projection	GO_BiologicalProcess-								
O:0031344	organization negative regulation of cell	GOA_23.02.2017_10h01 GO_BiologicalProcess-	12.0E-6	140.0E-6	3.9E+0	57.0E-12	790.0E-12	[4, 5]	Group13	5.0
0:0045596	differentiation immune response to tumor	GOA_23.02.2017_10h01 GO BiologicalProcess-	13.0E-6	140.0E-6	3.9E+0	57.0E-12	790.0E-12	[3, 4, 5, 6]	Group13	4.
O:0002418	cell	GOA_23.02.2017_10h01	14.0E-6	150.0E-6	3.8E+0	14.0E-6	28.0E-6	[3, 4]	Group06	31.3
O:0097435	supramolecular fiber organization	GO_BiologicalProcess- GOA_23.02.2017_10h01	14.0E-6	150.0E-6	3.8E+0	1.7E-9	12.0E-9	[3]	Group11	4.9
0.0044462	cell projection part	GO_CellularComponent- GOA_23.02.2017_10h01	16.0E-6	160.0E-6	3.8E+0	2.8E-9		[2, 3, 4]		4.1
		GO_BiologicalProcess-							Group09	
O:0030334	regulation of cell migration	GOA_23.02.2017_10h01 GO_CellularComponent-	15.0E-6	160.0E-6	3.8E+0	750.0E-9	2.1E-6	[4, 5, 6]	Group12	4.
0:0044463	cell projection part	GOA_23.02.2017_10h01	16.0E-6	160.0E-6	3.8E+0	57.0E-12	790.0E-12	[2, 3, 4]	Group13	4.
O:0051093	negative regulation of developmental process	GO_BiologicalProcess- GOA_23.02.2017_10h01	19.0E-6	180.0E-6	3.7E+0	57.0E-12	790.0E-12	[2, 3, 4]	Group13	4.
0.0005874	microtubule	GO_CellularComponent- GOA 23.02.2017 10h01	20.0E-6	200.0E-6	3.7E+0	1.7E-9	12.0E-9	[5, 6, 7, 8, 9 10]	Group11	5.0
	Role of Abl in Robo-Slit									
O:0001052	signaling positive regulation of	REACTOME_Pathways_01.03.2017	21.0E-6	210.0E-6	3.7E+0	21.0E-6	38.0E-6	[-1]	Group04	44.
O:0070886	calcineurin-NFAT signaling	GO_BiologicalProcess- GOA 23.02.2017 10h01	21.0E-6	210.0E-6	3.7E+0	35.0E-6	55.0E-6	[6, 7, 8, 9,	Group07	44.4
0:0070886	negative regulation of		21.0E-0	210.0E-0	3.7E+0	35.UE-0	55.UE-0	10, 11]	Group07	44.4
O:0051961	nervous system development	GO_BiologicalProcess- GOA 23.02.2017 10h01	27.0E-6	260.0E-6	3.6E+0	57.0E-12	790.0E-12	[3, 4, 5, 6, 7]	Group13	6.4
		GO_BiologicalProcess-						[3, 4, 5, 7,		
0:0001525	angiogenesis ameboidal-type cell	GOA_23.02.2017_10h01 GO_BiologicalProcess-	32.0E-6	290.0E-6	3.5E+0	750.0E-9	2.1E-6	8, 9, 10]	Group12	5.3
0:0001667	migration anatomical structure	GOA_23.02.2017_10h01	38.0E-6	350.0E-6	3.5E+0	750.0E-9	2.1E-6	[4, 5, 6]	Group12	5.
	formation involved in	GO_BiologicalProcess-								
0:0048646	morphogenesis regulation of cytoskeleton	GOA_23.02.2017_10h01 GO BiologicalProcess-	46.0E-6	400.0E-6	3.4E+0	750.0E-9	2.1E-6	[2, 3, 4]	Group12	4.0
O:0051493	organization regulation of plasma	GOA_23.02.2017_10h01 GO BiologicalProcess-	50.0E-6	440.0E-6	3.4E+0	1.7E-9	12.0E-9	[5, 6]	Group11	5.
O:1903729	membrane organization	GOA_23.02.2017_10h01	59.0E-6	490.0E-6	3.3E+0	59.0E-6	83.0E-6	[4, 5, 6]	Group02	10.8
0:0030424	axon	GO_CellularComponent- GOA_23.02.2017_10h01	57.0E-6	490.0E-6	3.3E+0	2.8E-9	9.8E-9	[4, 5]	Group09	5.3
		GO_CellularComponent-								
0:0030424	axon negative regulation of	GOA_23.02.2017_10h01	57.0E-6	490.0E-6	3.3E+0	57.0E-12	790.0E-12	[4, 5]	Group13	5.3
0.0002026	microtubule depolymerization	GO_BiologicalProcess- GOA_23.02.2017_10h01	60.0E-6	500.0E-6	3.3E+0	60.0E-6	77.0E-6	[5, 6, 7, 8, 9 10]	Group00	23.8
	negative regulation of cell	GO_BiologicalProcess-								
0:0010721	development negative regulation of cell	GOA_23.02.2017_10h01 GO_BiologicalProcess-	68.0E-6	550.0E-6	3.3E+0	57.0E-12	790.0E-12	[4, 5, 6, 7]	Group13	6.
O:0060548		GOA_23.02.2017_10h01 GO_BiologicalProcess-	78.0E-6	620.0E-6	3.2E+0	70.0E-6	82.0E-6	[3, 4, 5]	Group08	4.0
O:0031589	cell-substrate adhesion	GOA_23.02.2017_10h01	87.0E-6	660.0E-6	3.2E+0	35.0E-6	55.0E-6	[3]	Group07	5.
O·1901215	negative regulation of neuron death	GO_BiologicalProcess- GOA_23.02.2017_10h01	86.0E-6	670.0E-6	3.2E+0	70.0E-6	82.0E-6	[4 5 6]	Group08	7.
	blood vessel	GO_BiologicalProcess-						[3, 4, 6, 7,		
0:0048514	morphogenesis	GOA_23.02.2017_10h01 GO_BiologicalProcess-	85.0E-6	670.0E-6	3.2E+0	750.0E-9	2.1E-6	8, 9]	Group12	4.
O:0070997	neuron death regulation of cell-matrix	GOA_23.02.2017_10h01 GO_BiologicalProcess-	94.0E-6	700.0E-6	3.2E+0	70.0E-6	82.0E-6	[4]	Group08	5.
O:0001952	adhesion	GOA_23.02.2017_10h01	98.0E-6	720.0E-6	3.1E+0	35.0E-6	55.0E-6	[4, 5]	Group07	9.
0:0036477	somatodendritic compartment	GO_CellularComponent- GOA_23.02.2017_10h01	100.0E-6	740.0E-6	3.1E+0	2.8E-9	9.8E-9	[3, 4]	Group09	4.
		GO_MolecularFunction-								
	tubulin binding	GOA_23.02.2017_10h01 GO_BiologicalProcess-	100.0E-6	760.0E-6	3.1E+0	1.7E-9	12.0E-9		Group11	5.
O:0006935	chemotaxis	GOA_23.02.2017_10h01	110.0E-6	780.0E-6	3.1E+0	2.5E-9	12.0E-9	[3, 4]	Group10	4.
O:0006935	chemotaxis	GO_BiologicalProcess- GOA_23.02.2017_10h01	110.0E-6	780.0E-6	3.1E+0	750.0E-9	2.1E-6	[3, 4]	Group12	4.
0:0006935	chemotaxis	GO_BiologicalProcess- GOA_23.02.2017_10h01	110.0E-6	780.0E-6	3.1E+0	57.0E-12	790.0E-12	[3, 4]	Group13	4.
		GO_BiologicalProcess-								
U:0050919	negative chemotaxis	GOA_23.02.2017_10h01 GO_MolecularFunction-	120.0E-6	830.0E-6	3.1E+0	120.0E-6	120.0E-6	[4, 5]	Group05	20.
O:0008017	microtubule binding	GOA_23.02.2017_10h01	120.0E-6	850.0E-6	3.1E+0	1.7E-9	12.0E-9	[5]	Group11	6.
		GO_BiologicalProcess-								

GO:0002040 sprouting angiogenesis GOA_23.02.2017_10h01 140.0E-6 940.0E-6 3.0E+0 750.0E-9 2.1E-69, 10, 11] Group12 10.96 Z	GO:0002040 sprouting angiogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	140.0E-6	940.0E-6	3.0E+0	750.0E-9	[4, 5, 6, 8, 2.1E-69, 10, 11] Group12	10.96	217
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Ŭ							Group PValue			%	
			Term	Term PValue Corrected with	Neg log	Group	Corrected with Benjamini-	GOLev	GOGroup		Nr.
GOID	GOTerm	Ontology Source GO BiologicalProcess-	PValue	Benjamini-Hochberg	(FDR)	PValue	Hochberg	els	s	Genes	Genes
GO:0007399	nervous system development	GOA_23.02.2017_10h01	78.0E-9	45.0E-6	4.3E+0	13.0E-9	880.0E-9	[4, 5]	Group69	4.61	109.00
GO:0009653	anatomical structure morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	200.0E-9	58.0E-6	4.2E+0	200.0E-9	4.4E-6		Group39	4.47	114.00
GO:0098590	plasma membrane region	GO_CellularComponent- GOA_23.02.2017_10h01	900.0E-9	170.0E-6	3.8E+0	1.4E-6	18.0E-6	[2, 3, 4, 5, 6]	Group52	5.50	57.00
GO:0006928	movement of cell or subcellular component	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.6E-6	230.0E-6	3.6E+0	2.9E-6	32.0E-6	[3]	Group62	4.56	93.00
	Neurotransmitter Receptor Binding And Downstream Transmission In The	REACTOME_Pathways_0									
GO:000053	Postsynaptic Cell	1.03.2017 REACTOME_Pathways_0	3.8E-6	440.0E-6	3.4E+0	6.1E-6	58.0E-6	5[-1]	Group63	7.62	26.00
GO:000054	Transmission across Chemical Synapses	1.03.2017	3.8E-6	440.0E-6	3.4E+0	6.1E-6	58.0E-6	[-1]	Group63	7.62	26.00
GO:0000055	Neuronal System	REACTOME_Pathways_0 1.03.2017	3.8E-6	440.0E-6	3.4E+0	6.1E-6	58.0E-6	[-1]	Group63	7.62	26.00
GO:0001844	GABA A (rho) receptor activation	REACTOME_Pathways_0 1.03.2017	3.8E-6	440.0E-6	3.4E+0	6.1E-6	58.0E-6	[-1]	Group63	7.62	26.00
GO:0001845	GABA receptor activation	REACTOME_Pathways_0 1.03.2017	3.8E-6	440.0E-6	3.4E+0	6.1E-6	58.0E-6	[-1]	Group63	7.62	26.00
GO:0048699	generation of neurons	GO_BiologicalProcess- GOA_23.02.2017_10h01	15.0E-6	1.1E-3	3.0E+0	13.0E-9	880.0E-9	6. 71	Group69	4.71	69.00
	tube morphogenesis	GO_BiologicalProcess- GOA_23.02.2017_10h01	20.0E-6						Group51	6.93	26.00
		GO_CellularComponent-									
	cell projection	GOA_23.02.2017_10h01 GO_BiologicalProcess-	14.0E-6				260.0E-6		Group58	4.41	88.00
GO:0048468	cell development	GOA_23.02.2017_10h01 GO BiologicalProcess-	12.0E-6	1.2E-3	2.9E+0	13.0E-9	880.0E-9	3, 4, 5]	Group69	4.39	89.00
GO:0030182	neuron differentiation	GOA_23.02.2017_10h01 GO BiologicalProcess-	20.0E-6	1.2E-3	2.9E+0	13.0E-9	880.0E-9	[5, 7, 8]	Group69	4.80	64.00
GO:0022008	neurogenesis	GOA_23.02.2017_10h01 GO BiologicalProcess-	26.0E-6	1.3E-3	2.9E+0	13.0E-9	880.0E-9	[5, 6]	Group69	4.61	72.00
GO:0035295	tube development	GOA_23.02.2017_10h01	55.0E-6	2.1E-3	2.7E+0	260.0E-6	1.2E-3	[3, 4]	Group51	5.75	36.00
GO:0048870	cell motility	GO_BiologicalProcess- GOA_23.02.2017_10h01	53.0E-6	2.2E-3	2.7E+0	2.9E-6	32.0E-6	[2, 3, 4]	Group62	4.52	70.00
GO:1902495	transmembrane transporter complex	GO_CellularComponent- GOA 23.02.2017 10h01	51.0E-6	2.3E-3	2.6E+0	1.1E-3	3.5E-3	[2, 3, 4, 5]	Group68	7.01	23.00
GO:0098794	nostsvnanse	GO_CellularComponent- GOA 23.02.2017 10h01	50.0E-6	2.4E-3	2.6E+0	1.4E-6	18.0E-6	[2 3]	Group52	6.36	28.00
	leukocvte differentiation	GO_BiologicalProcess- GOA 23.02.2017 10h01	71.0E-6			1.2E-3	3.7E-3	[5, 6, 7,	Group56	6.07	30.00
		GO_BiologicalProcess-						[7, 8, 9,			
	monocyte differentiation	GOA_23.02.2017_10h01 GO_CellularComponent-	74.0E-6						Group67	18.42	7.00
GO:0043005	neuron projection	GOA_23.02.2017_10h01 GO_BiologicalProcess-	93.0E-6	3.0E-3	2.5E+0	32.0E-6	260.0E-6	[3, 4]	Group58	4.89	51.00
	cell projection organization Nicotine addiction	GOA_23.02.2017_10h01 KEGG 01.03.2017	110.0E-6	3.2E-3 3.2E-3	2.5E+0	13.0E-9 150.0E-9	880.0E-9 5.0E-6		Group69 Group70	4.53	65.00 7.00
	GABAergic synapse	KEGG_01.03.2017	160.0E-6	4.3E-3	2.4E+0	150.0E-9	5.0E-6	[-1]	Group70	11.36	10.00
GO:0048666	neuron development	GO_BiologicalProcess- GOA_23.02.2017_10h01	160.0E-6	4.4E-3	2.4E+0	13.0E-9	880.0E-9	[4, 5, 6, 8, 9]	Group69	4.79	51.00
GO:0044463	cell projection part	GO_CellularComponent- GOA_23.02.2017_10h01	190.0E-6	4.5E-3	2.3E+0	32.0E-6	260.0E-6	[2, 3, 4]	Group58	4.75	52.00
GO:0002573	myeloid leukocyte differentiation	GO_BiologicalProcess- GOA 23.02.2017 10h01	180.0E-6	4.5E-3	2.3E+0	650.0E-6	2.3E-3	[6, 7, 8, 9]	Group67	7.88	16.00
	Morphine addiction	KEGG_01.03.2017 GO_BiologicalProcess-	220.0E-6	4.5E-3	2.3E+0	150.0E-9	5.0E-6	[-1]	Group70	10.99	10.00
GO:0007267	cell-cell signaling	GOA_23.02.2017_10h01	240.0E-6	4.6E-3	2.3E+0	240.0E-6	1.3E-3	[3]	Group42	4.30	72.00
GO:0044224	juxtaparanode region of axon	GO_CellularComponent- GOA_23.02.2017_10h01	240.0E-6	4.6E-3	2.3E+0	600.0E-6	2.3E-3	[4, 5, 6, 87, 8]	Group57	33.33	4.00
GO:0007163	establishment or maintenance of cell polarity	GO_BiologicalProcess- GOA_23.02.2017_10h01	210.0E-6	4.7E-3	2.3E+0	210.0E-6	1.3E-3	3[3]	Group19	8.11	15.00
GO:1990778	protein localization to cell periphery	GO_BiologicalProcess- GOA_23.02.2017_10h01	230.0E-6	4.7E-3	2.3E+0	25.0E-3	25.0E-3	5]	Group53	7.20	18.00
	establishment of protein localization to plasma membrane	GO_BiologicalProcess- GOA_23.02.2017_10h01	260.0E-6		2.3E+0	25.0E-3			Group53	8.78	13.00
		GO_CellularComponent-						[3, 4, 5,		6.92	
	ion channel complex	GOA_23.02.2017_10h01 GO_BiologicalProcess-	180.0E-6		2.3E+0		3.5E-3		Group68		20.00
00.0001411	central nervous system development	GOA_23.02.2017_10h01 GO_BiologicalProcess-	210.0E-6	4.72 0	2.3E+0	10.0E 3	000.02 0	, o, o	Group69	4.84	48.00
GO:0060562	epithelial tube morphogenesis	GOA_23.02.2017_10h01 GO_CellularComponent-	280.0E-6	4.9E-3	2.3E+0	110.0E-6	820.0E-6	[4, 5, 6]	Group66	6.51	22.00
GO:0097458	neuron part	GOA_23.02.2017_10h01 GO_CellularComponent-	290.0E-6	5.0E-3	2.3E+0	32.0E-6	260.0E-6	[<u>2, 3]</u> [3, 4, 5,	Group58	4.42	63.00
	postsynaptic membrane	GOA_23.02.2017_10h01	400.0E-6		2.2E+0 2.2E+0	1.4E-6 400.0E-6	18.0E-6	6, 7, 8]		7.11	17.00 14.00
	Axon guidance	KEGG_01.03.2017 GO_BiologicalProcess-		6.1E-3					Group13		
	cell migration	GOA_23.02.2017_10h01 GO_BiologicalProcess-	370.0E-6		2.2E+0	2.9E-6		[4, 5, 6,	Group62	4.41	62.00
GO:0072073	kidney epithelium development	GOA_23.02.2017_10h01 GO_BiologicalProcess-	380.0E-6	6.2E-3	2.2E+0	110.0E-6	820.0E-6		Group66	8.44	13.00
GO:0007044	cell-substrate junction assembly	GOA_23.02.2017_10h01 GO_MolecularFunction-	400.0E-6	6.3E-3	2.2E+0	140.0E-6	980.0E-6	[5]	Group60	10.20	10.00
GO:0003779	actin binding	GOA_23.02.2017_10h01	460.0E-6	6.5E-3	2.2E+0	2.7E-3	6.6E-3	[4]	Group59	5.88	25.00

00 0004050	and the Provider	GO_CellularComponent-	450.05.0	0.05.0	0.05.0	110.05.0	000.05	10.01	0	0.40	04.00	
	cell leading edge	GOA_23.02.2017_10h01	450.0E-6	6.6E-3	2.2E+0	140.0E-6	980.0E-6	[2, 3]	Group60	6.12	24.00	
	regulation of mesenchymal cell	GO_BiologicalProcess-										1
GO:0010464	proliferation	GOA_23.02.2017_10h01	500.0E-6	7.0E-3	2.2E+0	500.0E-6	2.0E-3	[4, 5]	Group25	16.22	6.00	1
	regulation of multicellular organismal	GO_BiologicalProcess-										1
GO:2000026	development	GOA_23.02.2017_10h01	530.0E-6	7.2E-3	2.1E+0	13.0E-9	880.0E-9	[3, 4]	Group69	4.13	75.00	1
		GO_BiologicalProcess-										1
GO:0048729	tissue morphogenesis	GOA_23.02.2017_10h01	590.0E-6	7.9E-3	2.1E+0	450.0E-9	7.4E-6	[3, 4]	Group55	5.19	34.00	1
		GO_CellularComponent-						[2, 3, 4	,			1
GO:0097060	synaptic membrane	GOA_23.02.2017_10h01	690.0E-6	8.9E-3	2.1E+0	1.4E-6	18.0E-6	5, 6, 7]	Group52	6.35	20.00	1
	Interactions of neurexins and neuroligins a	tREACTOME_Pathways_0										1
GO:0001464	synapses	1.03.2017	780.0E-6	9.6E-3	2.0E+0	150.0E-9	5.0E-6	[-1]	Group70	11.27	8.00	1
		REACTOME_Pathways_0										1
GO:0001465	Protein-protein interactions at synapses	1.03.2017	780.0E-6	9.6E-3	2.0E+0	150.0E-9	5.0E-6	[-1]	Group70	11.27	8.00	1
		GO_BiologicalProcess-										1
GO:0032989	cellular component morphogenesis	GOA_23.02.2017_10h01	770.0E-6	9.7E-3	2.0E+0	13.0E-9	880.0E-9	[3, 4]	Group69	4.55	49.00	1

Table 3.4 | NPC-specific loops GO.

(A) Gene ontology enrichment terms and details for genes in NPC-specific loops.
(B) Gene ontology enrichment terms and details for genes in significant NPC loops that are lost in neurons. Negative log(FDR) from Benjamini-Hochberg correction. (C) Gene ontology enrichment terms and details for genes in significant NPC loops that are lost in glia. Negative log(FDR) from Benjamini-Hochberg correction. (See Table 3.2 for more information).

GOID	GOTerm	Neg	Ontology Source		Term PValue Corrected with Bonferroni step down		% Associated Genes		Total genes in term
	anatomical structure morphogenesis	20.602	Ontology Source GO_BiologicalProcess- GOA_23.02.2017_10h01	1.4E-24	2.5E-21	20.6	8.36	213	
	neuron differentiation	14.769	GO_BiologicalProcess- GOA_23.02.2017_10h01	980.0E-21	1.7E-15	14.8	9.53	127	
	cell development	14.552	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.5E-18	2.8E-15	14.6	8.29	168	
	nervous system development	13.958	GO_BiologicalProcess- GOA_23.02.2017_10h01	6.3E-18	11.0E-15	14.0	7.83	185	
	circulatory system development	12.886	GO_BiologicalProcess- GOA_23.02.2017_10h01	76.0E-18	130.0E-15	12.9	10.09	102	
	neurogenesis	11.638	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.3E-15	2.3E-12	11.6	8.51	133	
GO:0007167	enzyme linked receptor protein signaling	11.455	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.9E-15	3.5E-12	11.5	9.67	101	
	system development	11.346	GO_BiologicalProcess- GOA_23.02.2017_10h01	2.5E-15	4.5E-12	11.3	6.28	296	4711
	cell morphogenesis involved in differentiation	11.214	GO_BiologicalProcess- GOA_23.02.2017_10h01	3.5E-15	6.1E-12	11.2	11.05	78	706
GO:0030154	cell differentiation	11	GO_BiologicalProcess- GOA_23.02.2017_10h01	6.0E-15	10.0E-12	11.0	6.50	261	4013
GO:0007169	transmembrane receptor protein tyrosine kinase signaling pathway	879	GO_BiologicalProcess- GOA_23.02.2017_10h01	17.0E-15	30.0E-12	10.5	10.73	78	727
GO:0000902	cell morphogenesis	486	GO_BiologicalProcess- GOA_23.02.2017_10h01	18.0E-15	33.0E-12	10.5	9.64	95	985
GO:0007275	multicellular organism development	43		29.0E-15	51.0E-12	10.3	6.03	319	5288
GO:0048869	cellular developmental process	608	GO_BiologicalProcess- GOA_23.02.2017_10h01	35.0E-15	62.0E-12	10.2	6.36	267	4199
GO:0048667	cell morphogenesis involved in neuron differentiation	073	GO_BiologicalProcess- GOA_23.02.2017_10h01	63.0E-15	110.0E-12	10.0	11.56	66	571
GO:0032989	cellular component morphogenesis	087	GO_BiologicalProcess- GOA_23.02.2017_10h01	89.0E-15	150.0E-12	9.8	9.20	99	1076
GO:0048646	anatomical structure formation involved in morphogenesis	383	GO_BiologicalProcess- GOA_23.02.2017_10h01	170.0E-15	310.0E-12	9.5	9.26	96	1037
GO:0007507	heart development	509	GO_BiologicalProcess- GOA_23.02.2017_10h01	390.0E-15	690.0E-12	9.2	11.43	63	551
GO:0048666	neuron development	566	GO_BiologicalProcess- GOA_23.02.2017_10h01	750.0E-15	1.3E-9	8.9	9.02	96	1064
GO:0006928	movement of cell or subcellular component	72	GO_BiologicalProcess- GOA_23.02.2017_10h01	840.0E-15	1.4E-9	8.9	7.45	152	2041
GO:0051270	regulation of cellular component movement	315	GO_BiologicalProcess- GOA_23.02.2017_10h01	2.4E-12	4.3E-9	8.4	9.56	82	858
GO:0009887	animal organ morphogenesis	8.0506	GO_BiologicalProcess- GOA_23.02.2017_10h01	5.1E-12	8.9E-9	8.1	8.89	93	1046
GO:0009888	tissue development	8	GO_BiologicalProcess- GOA_23.02.2017_10h01	6.0E-12	10.0E-9	8.0	7.36	146	1983
GO:0072358	cardiovascular system development	8	GO_BiologicalProcess- GOA_23.02.2017_10h01	5.9E-12	10.0E-9	8.0	10.32	68	659
GO:0048812	neuron projection morphogenesis	511	GO_BiologicalProcess- GOA_23.02.2017_10h01	9.9E-12	17.0E-9	7.8	10.50	65	619
GO:0001568	blood vessel development	722	GO_BiologicalProcess- GOA_23.02.2017_10h01	13.0E-12	23.0E-9	7.6	10.40	65	625
GO:0070848	response to growth factor	267	GO_BiologicalProcess- GOA_23.02.2017_10h01	14.0E-12	26.0E-9	7.6	9.97	70	702
GO:0009790	embryo development	5	GO_BiologicalProcess- GOA_23.02.2017_10h01	18.0E-12	32.0E-9	7.5	8.80	90	1023
GO:0061061	muscle structure development	861	GO_BiologicalProcess- GOA_23.02.2017_10h01	19.0E-12	33.0E-9	7.5	10.41	64	615
GO:0061061	muscle structure development	861	GO_BiologicalProcess- GOA_23.02.2017_10h01	19.0E-12	33.0E-9	7.5	10.41	64	615
GO:0048513	animal organ development	161	GO_BiologicalProcess- GOA_23.02.2017_10h01	23.0E-12	41.0E-9	7.4	6.35	220	3464
GO:0048513	animal organ development	161	GO_BiologicalProcess- GOA_23.02.2017_10h01	23.0E-12	41.0E-9	7.4	6.35	220	3464
GO:0009966	regulation of signal transduction	021	GO_BiologicalProcess- GOA_23.02.2017_10h01	27.0E-12	47.0E-9	7.3	6.60	193	2924
GO:0009966	regulation of signal transduction	7.3279 021	GO_BiologicalProcess- GOA_23.02.2017_10h01	27.0E-12	47.0E-9	7.3	6.60	193	2924
GO:0009966	regulation of signal transduction	021	GO_BiologicalProcess- GOA_23.02.2017_10h01	27.0E-12	47.0E-9	7.3	6.60	193	2924
GO:0031175	neuron projection development		GO_BiologicalProcess- GOA_23.02.2017_10h01	38.0E-12	66.0E-9	7.2	9.05	82	906
GO:0040012	regulation of locomotion		GO_BiologicalProcess- GOA_23.02.2017_10h01	39.0E-12	68.0E-9	7.2	9.20	79	859
GO:2000145	regulation of cell motility	7	GO_BiologicalProcess- GOA_23.02.2017_10h01	58.0E-12	100.0E-9	7.0	9.43	74	785
GO:0071363	cellular response to growth factor stimulus	275	GO_BiologicalProcess- GOA_23.02.2017_10h01	100.0E-12	180.0E-9	6.7	9.82	66	672
GO:0048518	positive regulation of biological process	6.4814 861	GO_BiologicalProcess- GOA_23.02.2017_10h01	190.0E-12	330.0E-9	6.5	5.65	319	5645
GO:0048518	positive regulation of biological process	861	GO_BiologicalProcess- GOA_23.02.2017_10h01	190.0E-12	330.0E-9	6.5	5.65	319	5645
GO:0048518	positive regulation of biological process	6.4814	GO_BiologicalProcess- GOA_23.02.2017_10h01	190.0E-12	330.0E-9	6.5	5.65	319	5645
GO:0048514	blood vessel morphogenesis	6.4814 861	GO_BiologicalProcess- GOA_23.02.2017_10h01	190.0E-12	330.0E-9	6.5	10.58	57	539
	positive regulation of cellular process	6.4436	GO_BiologicalProcess- GOA_23.02.2017_10h01	200.0E-12	360.0E-9	6.4	5.78	291	5037
	positive regulation of cellular process	6.4436	GO_BiologicalProcess- GOA_23.02.2017_10h01	200.0E-12	360.0E-9	6.4	5.78	291	5037

		6.4436	GO_BiologicalProcess-	1		1			
GO:0048522	positive regulation of cellular process	975		200.0E-12	360.0E-9	6.4	5.78	291	503
GO:0007409	axonogenesis	875		260.0E-12	450.0E-9	6.3	11.04	51	462
GO:0030334	regulation of cell migration	062		310.0E-12	540.0E-9	6.3	9.44	69	73
GO:0007166	cell surface receptor signaling pathway	595		360.0E-12	630.0E-9	6.2	6.46	188	290
GO:0007166	cell surface receptor signaling pathway	595	GOA_23.02.2017_10h01	360.0E-12	630.0E-9	6.2	6.46	188	290
GO:0010646	regulation of cell communication	219	GO_BiologicalProcess- GOA_23.02.2017_10h01	480.0E-12	830.0E-9	6.1	6.28	204	324
GO:0010646	regulation of cell communication	219	GO_BiologicalProcess- GOA_23.02.2017_10h01	480.0E-12	830.0E-9	6.1	6.28	204	324
GO:0010646	regulation of cell communication		GO_BiologicalProcess- GOA_23.02.2017_10h01	480.0E-12	830.0E-9	6.1	6.28	204	324
GO:0051094	positive regulation of developmental process		GO_BiologicalProcess- GOA 23.02.2017 10h01	550.0E-12	950.0E-9	6.0	7.94	100	126
	positive regulation of developmental process	6.0222	GO_BiologicalProcess- GOA_23.02.2017_10h01	550.0E-12	950.0E-9	6.0	7.94	100	
	heart morphogenesis	6.0043	GO_BiologicalProcess- GOA_23.02.2017_10h01	570.0E-12	990.0E-9	6.0	14.05	34	
		5.8538	GO_BiologicalProcess-						
	regulation of signaling		GO_BiologicalProcess-	820.0E-12	1.4E-6	5.9	6.24	206	
GO:0023051	regulation of signaling		GOA_23.02.2017_10h01 GO_BiologicalProcess-	820.0E-12	1.4E-6	5.9	6.24	206	330
GO:0023051	regulation of signaling		GOA_23.02.2017_10h01 GO BiologicalProcess-	820.0E-12	1.4E-6	5.9	6.24	206	330
GO:0030029	actin filament-based process	511	GOA_23.02.2017_10h01 GO BiologicalProcess-	980.0E-12	1.7E-6	5.8	9.27	67	72
GO:0016477	cell migration	464		1.1E-9	1.9E-6	5.7	7.61	107	140
GO:0005737	cytoplasm	7	GOA_23.02.2017_10h01	1.1E-9	2.0E-6	5.7	4.92	558	1134
GO:0048598	embryonic morphogenesis	807	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.2E-9	2.1E-6	5.7	9.71	60	61
GO:0035556	intracellular signal transduction	5.6382 722	GO_BiologicalProcess- GOA_23.02.2017_10h01	1.3E-9	2.3E-6	5.6	6.41	183	285
GO:0010604	positive regulation of macromolecule metabolic process	5.4089 354	GO_BiologicalProcess- GOA_23.02.2017_10h01	2.2E-9	3.9E-6	5.4	6.27	192	306
GO:0048870		5.3372 422	GO_BiologicalProcess- GOA_23.02.2017_10h01	2.6E-9	4.6E-6	5.3	7.35	114	155
	positive regulation of multicellular organismal	5.3279	GO_BiologicalProcess-						
GO:0051240	process positive regulation of multicellular organismal	5.3279	GOA_23.02.2017_10h01 GO_BiologicalProcess-	2.7E-9	4.7E-6	5.3	7.35	114	155
	process		GOA_23.02.2017_10h01 GO_BiologicalProcess-	2.7E-9	4.7E-6	5.3	7.35	114	155
GC:0009893	positive regulation of metabolic process	3 5.2757	GOA_23.02.2017_10h01 GO BiologicalProcess-	2.9E-9	5.0E-6	5.3	6.16	204	331
GO:0050793	regulation of developmental process	241	GOA_23.02.2017_10h01 GO BiologicalProcess-	3.0E-9	5.3E-6	5.3	6.60	158	239
GO:0050793	regulation of developmental process	241	GOA_23.02.2017_10h01 GO BiologicalProcess-	3.0E-9	5.3E-6	5.3	6.60	158	239
GO:0050793	regulation of developmental process	241	GOA_23.02.2017_10h01	3.0E-9	5.3E-6	5.3	6.60	158	239
GO:0001525	angiogenesis	373	GO_BiologicalProcess- GOA_23.02.2017_10h01	3.2E-9	5.5E-6	5.3	10.62	48	45
GO:0007423	sensory organ development		GO_BiologicalProcess- GOA_23.02.2017_10h01	3.4E-9	5.9E-6	5.2	9.95	55	55
GO:0045595	regulation of cell differentiation	5.1870 866	GO_BiologicalProcess- GOA_23.02.2017_10h01	3.8E-9	6.5E-6	5.2	7.23	117	161
GO:0045595	regulation of cell differentiation	5.1870 866	GO_BiologicalProcess- GOA_23.02.2017_10h01	3.8E-9	6.5E-6	5.2	7.23	117	161
	positive regulation of macromolecule biosynthetic process		GO_BiologicalProcess- GOA 23.02.2017 10h01	4.2E-9	7.3E-6	5.1	7.08	124	175
		5.1191	GO_BiologicalProcess-						
	ameboidal-type cell migration		GOA_23.02.2017_10h01 GO_CellularComponent-	4.4E-9	7.6E-6	5.1	11.70	40	
GO:0005622		739	GO_BiologicalProcess-	5.7E-9	9.8E-6	5.0	4.66	676	
GO:0048729	tissue morphogenesis	5	GOA_23.02.2017_10h01 GO_BiologicalProcess-	6.2E-9	10.0E-6	5.0	9.31	61	65
GO:0048729	tissue morphogenesis	4.9586	GOA_23.02.2017_10h01 GO BiologicalProcess-	6.2E-9	10.0E-6	5.0	9.31	61	65
GO:0007417	central nervous system development	073		6.9E-9	11.0E-6	5.0	8.17	81	99
GO:0007417	central nervous system development	073	GOA_23.02.2017_10h01	6.9E-9	11.0E-6	5.0	8.17	81	99
GO:0051960	regulation of nervous system development	188	GO_BiologicalProcess- GOA_23.02.2017_10h01	7.4E-9	12.0E-6	4.9	8.60	71	82
GO:0051960	regulation of nervous system development	188	GO_BiologicalProcess- GOA_23.02.2017_10h01	7.4E-9	12.0E-6	4.9	8.60	71	82
GO:1902531	regulation of intracellular signal transduction	275	GO_BiologicalProcess- GOA_23.02.2017_10h01	10.0E-9	18.0E-6	4.7	6.91	128	185
	signal transduction	4.6989	GO_BiologicalProcess- GOA_23.02.2017_10h01	11.0E-9	20.0E-6	4.7	5.41	334	617
	signal transduction	4.6989	GO_BiologicalProcess- GOA_23.02.2017_10h01	11.0E-9	20.0E-6	4.7	5.41	334	
	regulation of anatomical structure	4.6382	GO_BiologicalProcess-						
	morphogenesis	4.6382	GOA_23.02.2017_10h01 GO_BiologicalProcess-	14.0E-9	23.0E-6	4.6	8.07	81	
	epithelium development	4.6197	GOA_23.02.2017_10h01 GO_BiologicalProcess-	13.0E-9	23.0E-6	4.6	7.53	97	
GO:0009891	positive regulation of biosynthetic process positive regulation of nitrogen compound	888	GOA_23.02.2017_10h01 GO_BiologicalProcess-	14.0E-9	24.0E-6	4.6	6.82	132	193
GO:0051173	metabolic process	888	GOA_23.02.2017_10h01 GO_BiologicalProcess-	14.0E-9	24.0E-6	4.6	6.83	132	193
GO:0030030	cell projection organization	888	GOA_23.02.2017_10h01	14.0E-9	24.0E-6	4.6	7.31	105	143
GO:0098590	plasma membrane region	6	GO_CellularComponent- GOA_23.02.2017_10h01	14.0E-9	25.0E-6	4.6	8.00	83	103
	actin cytoskeleton organization		GO_BiologicalProcess- GOA_23.02.2017_10h01	18.0E-9	31.0E-6	4.5	9.27	58	62

		4.5086GO_BiologicalProcess-						
GO:0035295	tube development	383GOA_23.02.2017_10h01 4.4948GO_BiologicalProcess-	18.0E-9	31.0E-6	4.5	9.27	58	62
GO:0007010	cytoskeleton organization	5GOA_23.02.2017_10h01 4.4948GO_BiologicalProcess-	19.0E-9	32.0E-6	4.5	7.55	94	124
GO:0048583	regulation of response to stimulus	5GOA_23.02.2017_10h01	18.0E-9	32.0E-6	4.5	5.84	231	395
O:0048583	regulation of response to stimulus	4.4948GO_BiologicalProcess- 5GOA_23.02.2017_10h01	18.0E-9	32.0E-6	4.5	5.84	231	395
O:0048583	regulation of response to stimulus	4.4948GO_BiologicalProcess- 5GOA_23.02.2017_10h01	18.0E-9	32.0E-6	4.5	5.84	231	395
O:0010628	positive regulation of gene expression	4.4814GO_BiologicalProcess- 861GOA_23.02.2017_10h01	19.0E-9	33.0E-6	4.5	6.86	126	183
	epithelial cell migration	4.4685GO_BiologicalProcess- 211GOA_23.02.2017_10h01	20.0E-9	34.0E-6	4.5	12.75	32	25
	positive regulation of cell differentiation	4.4559 GO_BiologicalProcess- 32 GOA 23.02.2017 10h01	21.0E-9	35.0E-6	4.5	8.23	75	9
		4.4559GO_BiologicalProcess-						
	positive regulation of cell differentiation	32GOA_23.02.2017_10h01 4.4436GO_BiologicalProcess-	21.0E-9	35.0E-6	4.5	8.23	75	9
GC:0034330	cell junction organization	975GOA_23.02.2017_10h01 4.2839GO_BiologicalProcess-	21.0E-9	36.0E-6	4.4	12.23	34	2
GO:0071495	cellular response to endogenous stimulus	967GOA_23.02.2017_10h01 4.2441GO BiologicalProcess-	30.0E-9	52.0E-6	4.3	7.40	98	13
GO:0045664	regulation of neuron differentiation	251 GOA_23.02.2017_10h01 4.0861 GO_BiologicalProcess-	34.0E-9	57.0E-6	4.2	9.24	55	5
O:0045216	cell-cell junction organization	861GOA_23.02.2017_10h01	48.0E-9	82.0E-6	4.1	12.55	31	24
O:0031328	positive regulation of cellular biosynthetic process	4.0809GO_BiologicalProcess- 219GOA_23.02.2017_10h01	49.0E-9	83.0E-6	4.1	6.72	128	19
O:0031325	positive regulation of cellular metabolic process	4.0757GO_BiologicalProcess- 207GOA_23.02.2017_10h01	50.0E-9	84.0E-6	4.1	6.06	188	31
O:0044424	intracellular part	4.0315GO_CellularComponent- 171GOA 23.02.2017 10h01	54.0E-9	93.0E-6	4.0	4.65	660	1419
	regulation of cellular component organization	4.0315GO_BiologicalProcess- 171GOA_23.02.2017_10h01	55.0E-9	93.0E-6	4.0	6.36	155	24
		4.0043GO_BiologicalProcess-					70	
	regulation of cell development	648GOA_23.02.2017_10h01 4.0043GO_BiologicalProcess-	59.0E-9	99.0E-6	4.0	8.23		8
	regulation of cell development	648GOA_23.02.2017_10h01 3.9586GO_BiologicalProcess-	59.0E-9	99.0E-6	4.0	8.23	70	8
O:0031589	cell-substrate adhesion	073 GOA_23.02.2017_10h01 3.9586 GO_BiologicalProcess-	70.0E-9	110.0E-6	4.0	11.08	37	3
O:0048568	embryonic organ development	073GOA_23.02.2017_10h01 3.9586GO BiologicalProcess-	68.0E-9	110.0E-6	4.0	9.98	45	4
O:0003205	cardiac chamber development	073GOA_23.02.2017_10h01	69.0E-9	110.0E-6	4.0	14.81	24	1
O:0043009	chordate embryonic development	3.9586GO_BiologicalProcess- 073GOA_23.02.2017_10h01	70.0E-9	110.0E-6	4.0	9.09	55	6
O:0010632	regulation of epithelial cell migration	3.9208GO_BiologicalProcess- 188GOA_23.02.2017_10h01	75.0E-9	120.0E-6	3.9	13.90	26	1
O:0060537	muscle tissue development	3.8538GO_BiologicalProcess- 72GOA_23.02.2017_10h01	84.0E-9	140.0E-6	3.9	10.53	40	3
	regulation of localization	3.8538GO_BiologicalProcess- 72GOA_23.02.2017_10h01	86.0E-9	140.0E-6	3.9	6.23	165	264
	muscle tissue development	3.8538GO_BiologicalProcess- 72GOA_23.02.2017_10h01	84.0E-9	140.0E-6	3.9	10.53	40	3
	positive regulation of nervous system	3.8538GO_BiologicalProcess-						
	development	72GOA_23.02.2017_10h01 3.7447GO_BiologicalProcess-	87.0E-9	140.0E-6	3.9	9.60	48	5
O:0034329	cell junction assembly regulation of cellular response to growth factor	275GOA_23.02.2017_10h01 3.7447GO_BiologicalProcess-	100.0E-9	180.0E-6	3.7	12.96	28	2
O:0090287	stimulus	275 GOA_23.02.2017_10h01 3.7212 GO_BiologicalProcess-	110.0E-9	180.0E-6	3.7	12.11	31	2
O:0051093	negative regulation of developmental process	464GOA_23.02.2017_10h01 3.7212GO_BiologicalProcess-	110.0E-9	190.0E-6	3.7	8.05	72	8
O:0051093	negative regulation of developmental process	464GOA_23.02.2017_10h01	110.0E-9	190.0E-6	3.7	8.05	72	8
O:0051093	negative regulation of developmental process	3.7212GO_BiologicalProcess- 464GOA_23.02.2017_10h01	110.0E-9	190.0E-6	3.7	8.05	72	8
O:0003279	cardiac septum development	3.6777GO_BiologicalProcess- 807GOA_23.02.2017_10h01	130.0E-9	210.0E-6	3.7	18.18	18	
	regulation of multicellular organismal process	3.6575GO_BiologicalProcess- 773GOA 23.02.2017 10h01	130.0E-9	220.0E-6	3.7	6.13	172	28
	regulation of multicellular organismal process	3.6575GO_BiologicalProcess- 773GOA_23.02.2017_10h01	130.0E-9	220.0E-6		6.13	172	28
	regulation of multicellular organismal process	3.6575GO BiologicalProcess-						
		773GOA_23.02.2017_10h01 3.6575GO_BiologicalProcess-	130.0E-9	220.0E-6	3.7	6.13	172	28
0:0051239	regulation of multicellular organismal process	773GOA_23.02.2017_10h01 3.5228GO_BiologicalProcess-	130.0E-9	220.0E-6	3.7	6.13	172	28
O:0019222	regulation of metabolic process	787 GOA_23.02.2017_10h01 3.4948 GO_Biological Process-	180.0E-9	300.0E-6	3.5	5.24	348	66
O:0060255	regulation of macromolecule metabolic process	5GOA_23.02.2017_10h01 3.4814GO_BiologicalProcess-	190.0E-9	320.0E-6	3.5	5.31	326	61
O:0014031	mesenchymal cell development	861GOA_23.02.2017_10h01	200.0E-9	330.0E-6	3.5	22.58	14	
0:0007420	brain development	3.4685GO_BiologicalProcess- 211GOA_23.02.2017_10h01	200.0E-9	340.0E-6	3.5	8.34	63	7
O:0007420	brain development	3.4685GO_BiologicalProcess- 211GOA_23.02.2017_10h01	200.0E-9	340.0E-6	3.5	8.34	63	7
	cardiac chamber morphogenesis	3.4685GO_BiologicalProcess- 211GOA_23.02.2017_10h01	200.0E-9	340.0E-6	3.5	16.13	20	1:
O:0038084	vascular endothelial growth factor signaling	3.4436GO_BiologicalProcess-			3.4	33.33	10	
		975GOA_23.02.2017_10h01 3.4089GO_BiologicalProcess-	210.0E-9	360.0E-6				
	head development	354GOA_23.02.2017_10h01 3.4089GO_BiologicalProcess-	230.0E-9	390.0E-6	3.4	8.23	65	7
O:0060322	head development	354 GOA_23.02.2017_10h01 3.3767 GO CellularComponent-	230.0E-9	390.0E-6	3.4	8.23	65	79
O:0099568	cytoplasmic region	507GOA_23.02.2017_10h01 3.3372GO_BiologicalProcess-	250.0E-9	420.0E-6	3.4	10.87	35	3
GO:0060562	epithelial tube morphogenesis	422GOA_23.02.2017_10h01	280.0E-9	460.0E-6	3.3	10.65	36	3
	mesenchyme development	3.3098GO_BiologicalProcess- 039GOA_23.02.2017_10h01	290.0E-9	490.0E-6	3.3	12.08	29	2

		3.3010GO_BiologicalProcess-						
GO:0048519 ne	gative regulation of biological process	3GOA_23.02.2017_10h01	300.0E-9	500.0E-6	3.3	5.47	275	5031
		3.3010GO_BiologicalProcess-						
GO:0048519 ne	gative regulation of biological process	3GOA_23.02.2017_10h01	300.0E-9	500.0E-6	3.3	5.47	275	5031
		3.3010GO_BiologicalProcess-						
GO:0048519 ne	gative regulation of biological process	3GOA_23.02.2017_10h01	300.0E-9	500.0E-6	3.3	5.47	275	5031
		3.2291 GO_BiologicalProcess-						
50:0042692 mi	uscle cell differentiation	48GOA_23.02.2017_10h01	350.0E-9	590.0E-6	3.2	10.18	39	383
0.0074040	0. 1	3.2218GO_BiologicalProcess- 487GOA 23.02.2017 10h01	360.0E-9	000.05.0	3.2	0.04	450	0.400
	Ilular response to organic substance	487GOA_23.02.2017_10h01 3.2146GO BiologicalProcess-	360.0E-9	600.0E-6	3.2	6.24	150	2403
GO:2000026 de	gulation of multicellular organismal	702GOA 23.02.2017 10h01	370.0E-9	610.0E-6	3.2	6.62	120	1814
	gulation of multicellular organismal	3.2146GO BiologicalProcess-	370.0E-9	810.0E-8	3.2	0.02	120	1014
GO:2000026 de		702GOA 23.02.2017 10h01	370.0E-9	610.0E-6	3.2	6.62	120	1814
	gulation of multicellular organismal	3.2146GO BiologicalProcess-	370.02-3	010.02-0	J.Z	0.02	120	1014
GO:2000026 de		702GOA 23.02.2017 10h01	370.0E-9	610.0E-6	3.2	6.62	120	1814
	sitive regulation of transcription from RNA	3.2076GO BiologicalProcess-	070.0L 3	010.0E 0	0.2	0.02	120	1014
	lymerase II promoter	083GOA 23.02.2017 10h01	370.0E-9	620.0E-6	3.2	7.36	84	1141
50.00 100 11 po		3.1804GO_BiologicalProcess-	010.02 0	020.02 0	0.2	7.00	0.	
O:0050767 red	gulation of neurogenesis	561GOA 23.02.2017 10h01	390.0E-9	660.0E-6	3.2	8.31	61	734
		3.1804GO BiologicalProcess-			0			
GO:0050767 red	gulation of neurogenesis	561GOA 23.02.2017 10h01	390.0E-9	660.0E-6	3.2	8.31	61	734
		3.1549GO CellularComponent-						
GO:0014069 po	stsynaptic density	02GOA_23.02.2017_10h01	420.0E-9	700.0E-6	3.2	12.44	27	217
	NA polymerase II transcription factor activity,	3.1549GO_MolecularFunction-						
GO:0000981 se	quence-specific DNA binding	02GOA_23.02.2017_10h01	420.0E-9	700.0E-6	3.2	8.28	61	737
		3.1249GO_BiologicalProcess-						
GO:0030900 for	rebrain development	387GOA_23.02.2017_10h01	450.0E-9	750.0E-6	3.1	9.90	40	404
		3.1249GO_BiologicalProcess-						
GO:0048523 ne	gative regulation of cellular process	387GOA_23.02.2017_10h01	450.0E-9	750.0E-6	3.1	5.54	252	4552
		3.1249GO_BiologicalProcess-						
GO:0048523 ne	gative regulation of cellular process	387GOA_23.02.2017_10h01	450.0E-9	750.0E-6	3.1	5.54	252	4552
		3.1191GO_BiologicalProcess-						
30:0001501 ske	eletal system development	864GOA_23.02.2017_10h01	460.0E-9	760.0E-6	3.1	9.18	47	512
00 00 40705	database a seconda a seconda	3.1191 GO_BiologicalProcess-	100.05.0	700.05.0		10.00	07	
50:0048705 SK	eletal system morphogenesis	864GOA_23.02.2017_10h01	460.0E-9	760.0E-6	3.1	12.39	27	218
0.0007405	uron projection guidance	3.1191GO_BiologicalProcess- 864GOA 23.02.2017 10h01	460.0E-9	760.0E-6	3.1	11.84	29	24
50:0097485 ne	uron projection guidance	3.0861GO MolecularFunction-	460.0E-9	760.0E-6	3.1	11.64	29	243
GO:0043168 an	ion binding	861GOA 23.02.2017 10h01	490.0E-9	820.0E-6	3.1	6.01	172	2860
00.0043100 dii		3.0315GO BiologicalProcess-	430.0E-9	820.0E-0	3.1	0.01	1/2	2000
	be morphogenesis	171GOA 23.02.2017 10h01	560.0E-9	930.0E-6	3.0	10.13	38	375
50.0000208 lui		3.0268GO BiologicalProcess-	000.0L-3	530.0E-0	5.0	10.15	50	575
30.0048562 or	nbrvonic organ morphogenesis	721GOA 23.02.2017 10h01	560.0E-9	940.0E-6	3.0	10.86	33	304
50.00-0002 EII	ioryonic organ morphogenesis	12100/120.02.2017_10101	500.0L-3	540.0L-0	5.0	10.00	55	

Table 3.5 | Glia-specific loops GO.Gene ontology enrichment terms and details for genes in glia-specific loops (See
Table 3.2 for more information).

GOID	GOTerm	Neg log(P) Ontology Source	Term PValue	Term PValue Corrected with Bonferroni step down	% Associated Genes	Nr. Genes	Total genes in term
GO:0044444	cytoplasmic part	GO_CellularComponent- 11.7GOA_23.02.2017_10h01	1.1E-15	2.1E-12	17.24	1624	9421
GO:0005829	cytosol	GO_CellularComponent- 8.6GOA_23.02.2017_10h01	1.3E-12	2.5E-9	18.27	910	4982
GO:0043547	positive regulation of GTPase activity	GO_BiologicalProcess- 5.4GOA_23.02.2017_10h01	2.0E-9		23.62	163	690
GO:0006464	cellular protein modification process	GO_BiologicalProcess- 5.2GOA 23.02.2017 10h01	3.1E-9		18.12	740	4085
GO:0043231	intracellular membrane-bounded organelle	GO_CellularComponent- 4.3GOA 23.02.2017 10h01	25.0E-9		16.39	1762	10749
		GO_BiologicalProcess-					
GO:0015031	protein transport	4.3GOA_23.02.2017_10h01 GO_BiologicalProcess-	26.0E-9		19.33	405	2095
GO:0018193	peptidyl-amino acid modification	4.2GOA_23.02.2017_10h01 GO_BiologicalProcess-	35.0E-9		20.66	264	1278
GO:0015833	peptide transport	4.1GOA_23.02.2017_10h01 GO CellularComponent-	40.0E-9	75.0E-6	19.25	410	2130
GO:0044446	intracellular organelle part	4.0GOA_23.02.2017_10h01 GO_MolecularFunction-	51.0E-9	95.0E-6	16.64	1481	8901
GO:0032555	purine ribonucleotide binding	3.4GOA_23.02.2017_10h01 GO BiologicalProcess-	220.0E-9	410.0E-6	19.19	379	1975
GO:0051223	regulation of protein transport	3.3GOA_23.02.2017_10h01	250.0E-9	460.0E-6	21.73	178	819
GO:0005654	nucleoplasm	GO_CellularComponent- 3.2GOA_23.02.2017_10h01	310.0E-9	580.0E-6	17.99	619	3441
GO:0032559	adenyl ribonucleotide binding	GO_MolecularFunction- 3.2GOA_23.02.2017_10h01	360.0E-9	670.0E-6	19.58	316	1614
GO:0030554	adenyl nucleotide binding	GO_MolecularFunction- 3.1GOA_23.02.2017_10h01	390.0E-9	730.0E-6	19.56	318	1626
GO:0090087	regulation of peptide transport	GO_BiologicalProcess- 3.0GOA 23.02.2017 10h01	510.0E-9	970.0E-6	21.44	182	849
GO:0006468	protein phosphorylation	GO_BiologicalProcess- 3.0GOA 23.02.2017 10h01	540.0E-9	1.0E-3	19.04	376	1975
GO:1902531	regulation of intracellular signal transduction	GO_BiologicalProcess- 3.0GOA 23.02.2017 10h01	620.0E-9		19.16	355	1853
GO:0044428	nuclear part	GO_CellularComponent- 2.7GOA_23.02.2017_10h01	970.0E-9		17.46	770	4411
		GO_BiologicalProcess-					
GO:0030097	hemopoiesis hematopoietic or lymphoid organ	GO_BiologicalProcess-	1.4E-6		21.52	164	762
GO:0048534	development negative regulation of signal	2.4GOA_23.02.2017_10h01 GO_BiologicalProcess-	2.1E-6		21.24	171	805
GO:0009968	transduction	2.4GOA_23.02.2017_10h01 GO_MolecularFunction-	2.3E-6	4.3E-3	20.02	239	1194
GO:0005524	ATP binding	2.3 GOA_23.02.2017_10h01 GO BiologicalProcess-	2.5E-6	4.7E-3	19.29	304	1576
GO:0050708	regulation of protein secretion	2.3GOA_23.02.2017_10h01 GO_BiologicalProcess-	2.5E-6	4.7E-3	23.62	103	436
GO:0002791	regulation of peptide secretion	2.0GOA_23.02.2017_10h01	5.1E-6	9.6E-3	23.06	107	464
GO:0051247	positive regulation of protein metabolic process	GO_BiologicalProcess- 2.0GOA_23.02.2017_10h01	5.3E-6		19.07	312	1636
GO:0004015	Rap1 signaling pathway	2.0KEGG_01.03.2017 GO_CellularComponent-	6.2E-6		27.14	57	210
GO:0031981	nuclear lumen	2.0GOA_23.02.2017_10h01 GO_BiologicalProcess-	6.4E-6		17.40	704	4045
GO:0019932	second-messenger-mediated signaling transmembrane receptor protein	1.9GOA_23.02.2017_10h01 GO BiologicalProcess-	6.9E-6	12.0E-3	25.77	67	260
GO:0007169	tyrosine kinase signaling pathway 3',5'-cyclic-AMP phosphodiesterase	1.8 GOA_23.02.2017_10h01 GO MolecularFunction-	8.5E-6	15.0E-3	21.18	154	727
GO:0004115	activity positive regulation of cellular protein	1.8GOA_23.02.2017_10h01 GO_BiologicalProcess-	8.7E-6	16.0E-3	66.67	10	15
GO:0032270	metabolic process	1.7GOA_23.02.2017_10h01	11.0E-6	20.0E-3	19.08	292	1530
GO:0031399	regulation of protein modification process	GO_BiologicalProcess- 1.7GOA_23.02.2017_10h01	11.0E-6	21.0E-3	18.75	333	1776
GO:0009306	protein secretion	GO_BiologicalProcess- 1.7GOA_23.02.2017_10h01	11.0E-6	21.0E-3	22.06	120	544
GO:0048666	neuron development	GO_BiologicalProcess- 1.5GOA_23.02.2017_10h01	18.0E-6	34.0E-3	19.83	211	1064
GO:0002790	peptide secretion	GO_BiologicalProcess- 1.5GOA_23.02.2017_10h01	19.0E-6	35.0E-3	21.70	125	576
GO:0007167	enzyme linked receptor protein signaling pathway		23.0E-6		19.83	207	1044

Table 3.6 | GM12878 lymphoblastoid-specific loops GO.Gene ontology enrichment terms and details for genes in GM12878-specificloops (See Table 3.2 for more).

			No. Overlap Glia TADs	ahr (Clia Cana)	v1 (Clia Cono)	v2 (Clia Cono)	Cono ID (ENSC)	Gene	Stron	Gono longth (br
chr2	139600000	143750000		chr2	139654893		ENSG00000229104	Symbol YY1P2	+	dGene length (bp 185
chr2	139600000	143750000		chr2	141750111		ENSG0000221892	Y RNA	+	100
chr2	139600000	143750000		chr2	141924825		ENSG00000252015	U6	ŀ	10
chr2	139600000	143750000		chr2	143635194	143799885	ENSG00000115919	KYNU	+	11480
chr2	139600000	143750000		chr2	140988995		ENSG00000168702	LRP1B	-	190027
chr2	140425000	143750000		chr2	141750111		ENSG00000201892	Y_RNA	+	11
chr2	140425000	143750000	11	chr2	141924825	141924926		U6	t	10
chr2	140425000	143750000	11	chr2	143635194	143799885		KYNU L DD4D	+	11480
chr2	140425000 139600000	143750000	11	chr2	140988995		ENSG00000168702 ENSG00000229104	LRP1B YY1P2	Ē.	190027
chr2 chr2	139600000	142950000 142950000	10		139654893 141750111		ENSG00000229104	Y_RNA	+	185 11
chr2	139600000	142950000		chr2	141924825		ENSG00000252015	U6	Ľ	10
chr2	139600000	142950000		chr2	140988995		ENSG00000168702	LRP1B	-	190027
chr18	25175000	28550000		chr18	25373549	25373648		Mir 384	n/a	9
chr18	25175000	28550000		chr18	25613650	25613747		Mir 340	n/a	9
chr18	25175000	28550000	9	chr18	27878875	27878926		MIR302F	n/a	5
chr18	25175000	28550000	9	chr18	25530929		ENSG00000170558	CDH2	-	22651
chr18	25175000	28550000	9	chr18	24916342	25175128	ENSG00000264151	AK127888	-	12
chr9	80925000	85725000	9	chr9	82478897	82478939	n/a	DQ575560	n/a	4
chr9	80925000	85725000	9	chr9	82479022	82479053	n/a	DQ583878	n/a	3
chr9	80925000	85725000	9	chr9	82479147	82479179		DQ589820	n/a	3
chr9	80925000	85725000	9	chr9	82479196	82479300	n/a	DQ580124	n/a	10
chr9	80925000	85725000	9	chr9	82479444	82479479		DQ595182	n/a	3
chr9 chr9	80925000 80925000	85725000 85725000	g	chr9 chr9	82479496 82479564	82479529		DQ596925 DQ597713	n/a	3
chr9 chr9	80925000	85725000	9	chr9 chr9	82479564 82479638	82479595 82479675		DQ597713 DQ586158	n/a n/a	3
chr9 chr9	80925000	85725000		chr9 chr9	82479636	82479675		DQ586158 DQ588659	n/a n/a	3
chr9	80925000	85725000	9	chr9	82479911	82479966		DQ599976	n/a	5
chr9	80925000	85725000	9	chr9	82480085	82480149		DQ574305	n/a	6
chr9	80925000	85725000	9	chr9	82480265	82480311		DQ600106	n/a	4
chr9	80925000	85725000	9	chr9	82480444	82480513	3n/a	DQ574306	n/a	6
chr9	80925000	85725000	9	chr9	82480639	82480689	n/a	DQ599155	n/a	5
chr9	80925000	85725000	9	chr9	82480842	82480885	in/a	DQ574330	n/a	4
chr9	80925000	85725000	9	chr9	82481004	82481059	n/a	DQ579956	n/a	5
chr9	80925000	85725000	9	chr9	82481263	82481303		DQ594965	n/a	4
chr9	80925000	85725000		chr9	82481394	82481432		DQ596802	n/a	3
chr9	80925000	85725000		chr9	82481509	82481544		DQ574826	n/a	3
chr9	80925000	85725000		chr9	82481703	82481771		DQ592136	n/a	6
chr9	80925000	85725000 85725000	9	chr9	82482216 82482361		n/a	DQ577348	n/a	3
chr9 chr0	80925000 80925000	85725000	9	chr9	82482630	82482391	n/a Sn/a	DQ593172 DQ592375	n/a	3
chr9 chr9	80925000	85725000	9	chr9 chr9	82483105	82482673		DQ592375 DQ570523	n/a n/a	3
chr9	80925000	85725000	9	chr9	82483941	82483980	n/a	DQ573195	n/a	3
chr9	80925000	85725000	9	chr9	82973117	82973151		DQ591664	n/a	3
								SPATA31D5		
chr9	80925000	85725000	g	chr9	84528351	84534842	ENSG00000240632	Р	+	649
chr9	80925000	85725000	9	chr9	84531745			DQ592725	n/a	2
chr9	80925000	85725000	g	chr9	84532087	84532118		DQ577940	n/a	3
chr9	80925000	85725000	y	chr9	84533136			DQ578305	n/a	6
chr9	80925000	85725000	g	6110	84534683	84534718		DQ582032	n/a	3
chr9 chr9	80925000 80925000	85725000 85725000	9	chr9 chr9	84535666 84543342	84535704	n/a ENSG00000189357	DQ584769 SPATA31D4	n/a	3
chr9	80925000	85725000		chr9	84547133	84547164		DQ577940	+ n/a	3
chr9	80925000	85725000	q	chr9	84548208	84548275		DQ578305	n/a	6
chr9	80925000	85725000	9	chr9	84549755	84549790		DQ582032	n/a	3
chr9	80925000	85725000		chr9	84550738	84550776		DQ584769	n/a	3
chr9	80925000	85725000	9	chr9	84558414	84565009	ENSG00000186788	SPATA31D3	+	659
chr9	80925000	85725000	9	chr9	84562225	84562256	in/a	DQ577940	n/a	3
chr9	80925000	85725000	9	chr9	84563300	84563367		DQ578305	n/a	6
chr9	80925000	85725000	9	chr9	84891466	84891571		Mir_544	n/a	10
chr9	80925000	85725000	9	0.110	85045810			DQ588544	n/a	2
chr9	80925000	85725000		chr9	85202301	85202353		Mir_1302	n/a	5
chr9	80925000	85725000		chr9	80912058		ENSG00000135069	PSAT1	<u>†</u>	2000
chr9 chr9	80925000 80925000	85725000 85725000		chr9 chr9	84304627 84545140		ENSG00000233926 ENSG00000267559	BC036431 AK097447	t -	8718 4686
chr9 chr9	80925000	85725000	9	chr9	84545140		ENSG00000287559 ENSG00000214929	SPATA31D1	Ĺ	4000
chr9 chr9	80925000	85725000	9	chr9	85594499		ENSG00000214929 ENSG00000165105	RASEF	Ľ	8354
chr9	80925000	85725000	9	chr9	82186877		ENSG00000106829	TLE4	+	15477
chr9	80925000	85725000	9	chr9	84198597	84303596	ENSG00000196781	TLE1	F	10499
chr10	60100000	62425000	8	chr10	60474774		ENSG00000183055	FAM133CP	+	251
chr10	60100000	62425000		chr10	61019597			Mir_584	n/a	12
								Metazoa_SR		
chr10	60100000	62425000		chr10	61821742			Р	n/a	30
chr10	60100000	62425000		chr10	61841498		ENSG00000202190	Y_RNA	+	11
chr10	60100000	62425000		chr10	60094738		ENSG0000072401	UBE2D1	+	3051
chr10	60100000	62425000		chr10	60144902		ENSG00000108064	TFAM	+	1408
chr10	60100000	62425000		chr10	61410521		ENSG00000165449	SLC16A9	ţ	5912
chr10	60100000	62425000		chr10	61496747		ENSG00000227877	LINC00948	F	1645
chr10	60100000	62425000		chr10	61715187 60272903		ENSG00000235931	C10orf40	<u>[</u>	548
chr10	60100000	62425000		chr10 chr10			ENSG00000122870 ENSG00000165443	BICC1	+	31594
chr10 chr10	60100000 60100000	62425000 62425000		chr10 chr10	60936347 61005888		ENSG00000165443 ENSG00000148541	PHYHIPL FAM13C	£	7118
chr10	60100000	62425000		chr10 chr10	61548505		ENSG00000148541	CCDC6	£	11640
chr10	60100000	62425000		chr10	61786055		ENSG00000151150	ANK3	f	36357
chr3	58525000	62425000		chr3	59956575			NPCR	- n/a	240
	58525000	62275000		chr3	60842060		ENSG00000212211	U3	-	240
chr3	00020000	62275000		chr3	61068514			5S_rRNA	n/a	21

chr3 chr3										
	58525000	62275000		chr3	58549844		ENSG00000168309	FAM107A	-	13647
chra	58525000	62275000	80	chr3	58619669	58652561	ENSG00000198643	FAM3D	-	32892
chr3	58525000	62275000	80	chr3	58727736	59035715	ENSG00000163689	C3orf67	-	307979
chr3	58525000	62275000	80	chr3	58810196	59004819	ENSG00000242428	AK090895	+	194623
chr3	58525000	62275000		chr3	62247493	62304622	ENSG00000241472	PTPRG-AS1	-	27507
chr3	58525000	62275000		shr3	59735035		ENSG00000189283	FHIT		1502098
									-	
chr3	58525000	62275000		chr3	61547242	62280573	ENSG00000144724	PTPRG	+	727758
chr4	175250000	177250000	80	chr4	175344945		ENSG00000265846	MIR4276	+	70
chr4	175250000	177250000	80	chr4	175474212	175474248	n/a	HH834010	n/a	36
chr4	175250000	177250000	80	chr4	175848510	175848545	n/a	HH834010	n/a	35
chr4	175250000	177250000	80	chr4	177019313	177019436	ENSG00000201516	SNORA51	+	123
chr4	175250000	177250000		chr4	175411327		ENSG00000164120	HPGD	<u> </u>	32722
									<u> </u>	÷=·==
chr4	175250000	177250000		chr4	176711457		ENSG00000249106	BC038536	+	21883
chr4	175250000	177250000	80	chr4	177105724	177116822	ENSG00000150628	SPATA4	-	11098
chr4	175250000	177250000	80	chr4	177134825	177190373	ENSG00000164122	ASB5		55548
chr4	175250000	177250000		chr4	177241089	177253396	ENSG00000129128	SPCS3	+	8911
chr4	175250000	177250000		chr4	175205054		ENSG00000164118	CEP44		4531
									-	
chr4	175250000	177250000		chr4	175563197		ENSG00000145451	GLRA3	-	187268
chr4	175250000	177250000	80	chr4	175750818		ENSG00000168594	AK093264	+	41120
chr4	175250000	177250000	80	chr4	175839508	175899331	ENSG00000168594	ADAM29	+	59823
chr4	175250000	177250000	80	chr4	176554087	176923842	ENSG00000150625	GPM6A		369755
chr4	175250000	177250000		chr4	176986984	177103979	ENSG00000150627	WDR17	+	116995
chr5	162950000	167650000		shr5	165036445	165036684	ENSG00000252794	7SK	+	239
					167460095				Ĺ	
chr5	162950000	167650000		chr5			ENSG00000253065	SNORA40	<u> </u>	115
chr5	162950000	167650000		chr5	163781145	164029423	ENSG00000241956	BC011998	+	248278
chr5	162950000	167650000	80	chr5	166711842	167691162	ENSG00000145934	TENM2	+	938158
chr1	215800000	218375000	70	chr1	216825011	216825068	n/a	Mir_598	n/a	57
chr1	215800000	218375000		chr1	218066241		ENSG00000231814	LINC00210	+	27905
chr1	215800000	218375000		shr1	216676587		ENSG00000196482	ESRRG	t –	220227
chr1	215800000	218375000		chr1	217603833		ENSG0000092978	GPATCH2	r	200576
chr1	215800000	218375000		chr1	217804694		ENSG00000162814	SPATA17	+	235790
chr1	215800000	218375000	70	chr1	215796235	216596738	ENSG00000042781	USH2A	<u>-</u>	796738
chr1	71550000	75175000		chr1	72259914		ENSG00000228853	NEGR1-IT1	-	42781
chr1	71550000	75175000		shr1	75043113	75091782	ENSG00000234497	CR627203	+	48669
chr1	71550000	75175000			75171171	75199092	ENSG00000116791	CRYZ	Ľ	3829
UII I	1000000	101/5000	<u>۴</u>	chr1	75171171	10199092	L113G0000110/91		F	3829
1		76/770					ENO000000000000000000000000000000000000	ZRANB2-		
chr1	71550000	75175000		chr1	71547006		ENSG00000229956	AS2	+	153406
chr1	71550000	75175000	70	chr1	73771852	73804560	ENSG00000233973	BC041341	+	32708
chr1	71550000	75175000		shr1	74491701		ENSG00000162620	LRRIQ3	-	172170
0.11.1	11000000	10110000			11101101	1 100001 1	2110000000102020	FPGT-		
chr1	71550000	75175000		chr1	74663895	75040440	ENSG00000116783	TNNI3K	L.	346221
									+	
chr1	71550000	75175000		chr1	75033794	75139422	ENSG00000178965	C1orf173	-	105628
chr1	71550000	75175000	70	chr1	71868624		ENSG00000172260	NEGR1	-	879781
chr10	65600000	69550000	70	chr10	66585284	66586634	ENSG00000216740	ANXA2P3	+	1350
chr10	65600000	69550000		chr10	66585343	66585369	n/a	DJ439558	n/a	26
chr10	65600000	69550000		chr10	66586101	66586126		DJ439576	n/a	25
chr10	65600000	69550000		chr10	66586203	66586227		DJ439561	n/a	23
									II/d	
chr10	65600000	69550000		chr10	68254456		ENSG00000252203	SnoU40	+	101
chr10	65600000	69550000	7c	chr10	69524260	69524407	n/a	HI650153	n/a	147
chr10	65600000	69550000	70	chr10	68685791	68860867	ENSG00000198739	LRRTM3	+	175076
chr10	65600000	69550000	70	chr10	67679724	69455949	ENSG00000183230	CTNNA3	-	1776225
								TRNA Pseu		
chr12	96925000	98800000	70	chr12	97490690	97490764	n/a	do	n/a	74
chr12	96925000	98800000		chr12	97506929	97507042		Mir_584	n/a	113
chr12	96925000	98800000		chr12	97866326	97868402		AK129935	n/a	2076
chr12	96925000	98800000	7c	chr12	97885686	97885756	ENSG00000221479	MIR1251	+	70
								SnoMe28S_		
chr12	96925000	98800000	70	chr12	97945914	97946006	ENSG00000251844	Am2634	-	92
chr12	96925000	98800000	70	chr12	97957589		ENSG00000207586	MIR135A2	+	100
chr12	96925000	98800000		shr12	98389160	98389226	ENSG00000263890	MIR4303		66
									-	
chr12	96925000	98800000		chr12	98752079		n/a	Mir_548	n/a	84
chr12	96925000	98800000		chr12	96883352		ENSG00000188596	C12orf55	+	9765
chr12	96925000	98800000		chr12	97301000		ENSG00000139350	NEDD1	+	46469
chr12	96925000	98800000	70	chr12	98123825	98150295	ENSG00000257501	LOC643711	-	26470
chr12	96925000	98800000		chr12	96933914		ENSG00000188596	AX747187	+	90515
chr12	96925000	98800000		shr12	97041752	97159032	ENSG00000188596	C12orf63		117280
chr12	96925000	98800000		chr12	97858798	01100002			+	
-	63375000			chr16		97027544	ENSG00000255704		+	
chr16	0.3375000					97927544	ENSG00000255794	RMST	+ +	68746
		66400000			65420321	65420381	n/a	RMST JB153694	+ + n/a	60
chr16	63375000	66400000	7c	chr16	65420321 66335711	65420381 66335843	n/a ENSG00000201999	RMST JB153694 5S_rRNA	+ + n/a +	60 132
chr16	63375000 63375000	66400000 66400000	7c 7c	chr16 chr16	65420321 66335711 65318401	65420381 66335843 65610203	n/a ENSG00000201999 ENSG00000261742	RMST JB153694 5S_rRNA LINC00922	+ + n/a +	60 132 291802
	63375000	66400000	7c 7c	chr16	65420321 66335711	65420381 66335843 65610203	n/a ENSG00000201999	RMST JB153694 5S_rRNA	+ + n/a + -	60 132
chr16	63375000 63375000	66400000 66400000	7c 7c 7c	chr16 chr16	65420321 66335711 65318401	65420381 66335843 65610203 65155919	n/a ENSG00000201999 ENSG00000261742	RMST JB153694 5S_rRNA LINC00922 CDH11	+ + n/a + - -	60 132 291802
chr16 chr16 chr2	63375000 63375000 63375000 140500000	66400000 66400000 66400000 142900000	7c 7c 7c 7c 7c	chr16 chr16 chr16 chr16 chr2	65420321 66335711 65318401 64980682 141750111	65420381 66335843 65610203 65155919 141750228	n/a ENSG00000201999 ENSG00000261742 ENSG00000140937 ENSG00000201892	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA	+ n/a + - -	60 132 291802 175237 117
chr16 chr16 chr2 chr2	63375000 63375000 63375000 140500000 140500000	66400000 66400000 66400000 142900000 142900000	7c 7c 7c 7c 7c 7c	chr16 chr16 chr16 chr2 chr2 chr2	65420321 66335711 65318401 64980682 141750111 141924825	65420381 66335843 65610203 65155919 141750228 141924926	n/a ENSG0000201999 ENSG0000261742 ENSG0000140937 ENSG0000201892 ENSG0000252015	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6	+ n/a + - - +	60 132 291802 175237 117 101
chr16 chr16 chr2 chr2 chr2 chr2	63375000 63375000 63375000 140500000 140500000 140500000	66400000 66400000 142900000 142900000 142900000	7c 7c 7c 7c 7c 7c 7c 7c	chr16 chr16 chr16 chr2 chr2 chr2 chr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995	65420381 66335843 65610203 65155919 141750228 141924926 142889270	n/a ENSG0000201999 ENSG0000261742 ENSG0000140937 ENSG0000201892 ENSG00000252015 ENSG00000168702	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B	+ n/a + - - -	60 132 291802 175237 117 101 1900275
chr16 chr16 chr2 chr2 chr2 chr2 chr2	63375000 63375000 63375000 140500000 140500000 140500000 33875000	66400000 66400000 142900000 142900000 142900000 37175000	70 70 70 70 70 70 70 70 70 70 70	shr16 shr16 shr16 shr2 shr2 shr2 shr2 shr2 shr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127	65420381 66335843 65610203 65155919 141750228 141924926 142889270 33953284	n/a ENSG0000201999 ENSG0000261742 ENSG00002140937 ENSG0000252015 ENSG0000252015 ENSG00000188702 ENSG00000239649	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML	+ + n/a + - - - -	60 132 291802 175237 117 101 101 1900275 2157
chr16 chr16 chr2 chr2 chr2 chr2	63375000 63375000 63375000 140500000 140500000 140500000	66400000 66400000 142900000 142900000 142900000	70 70 70 70 70 70 70 70 70 70 70	chr16 chr16 chr16 chr2 chr2 chr2 chr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995	65420381 66335843 65610203 65155919 141750228 141924926 142889270 33953284	n/a ENSG0000201999 ENSG0000261742 ENSG0000140937 ENSG0000201892 ENSG00000252015 ENSG00000168702	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548	+ n/a + - - - - -	60 132 291802 175237 117 101 1900275
chr16 chr16 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 140500000 140500000 33875000 33875000	66400000 66400000 142900000 142900000 142900000 37175000 37175000	7c 7c 7c 7c 7c 7c 7c 7c 7c	chr16 chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127 34628738	65420381 66335843 65610028 141750228 141924926 142889270 33953284 34628822	n/a ENSG00000201999 ENSG00000261742 ENSG00000201892 ENSG00000201892 ENSG00000168702 ENSG00000168702 ENSG00000168702 ENSG00000239649 ENSG00000212025	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML	+ n/a + - - - - -	60 132 291802 175237 117 101 1900275 2157 84
chr16 chr16 chr2 chr2 chr2 chr2 chr2	63375000 63375000 63375000 140500000 140500000 140500000 33875000	66400000 66400000 142900000 142900000 142900000 37175000	7c 7c 7c 7c 7c 7c 7c 7c 7c	shr16 shr16 shr16 shr2 shr2 shr2 shr2 shr2 shr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127	65420381 66335843 65610203 65155919 141750228 141924926 142889270 33953284	n/a ENSG00000201999 ENSG00000261742 ENSG00000201892 ENSG00000201892 ENSG00000168702 ENSG00000168702 ENSG00000168702 ENSG00000239649 ENSG00000212025	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548	+ n/a + - - - - - -	60 132 291802 175237 117 101 101 1900275 2157
chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 140500000 33875000 33875000 33875000	66400000 66400000 142900000 142900000 142900000 37175000 37175000 37175000	7 c 7 c 7 c 7 c 7 c 7 c 7 c 7 c 7 c 7 c	chr16 chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr3	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127 34628738 36581891	65420381 66335843 65610028 65155919 141750228 141924926 142889270 33953284 34628822 36582713	n/a ENSG0000201999 ENSG0000261742 ENSG00000261742 ENSG00000252015 ENSG00000252015 ENSG00000252015 ENSG00000239649 ENSG00000239649 ENSG00000212025	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548 LOC1002889 11	+	60 132 291802 175237 117 100275 2157 84 822
chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 140500000 33875000 33875000 33875000 33875000	66400000 66400000 142900000 142900000 142900000 37175000 37175000 37175000 37175000	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	shr16 shr16 shr2	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127 34628738 36581891 36779403	65420381 66335843 65610203 65155919 141750228 141924926 142889270 33953284 34628822 36582713 36825332	n/a ENSG0000201999 ENSG0000261742 ENSG0000261742 ENSG000002501892 ENSG00000252015 ENSG00000239649 ENSG00000212025 n/a ENSG0000171055	RMST JB153694 5S_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548 LOC1002889 11 FEZ2	+	60 132 291802 175237 117 101 1900275 2157 84 822 45929
chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 140500000 33875000 33875000 33875000 33875000 33875000	66400000 66400000 142900000 142900000 37175000 37175000 37175000 37175000 37175000 37175000	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	hr16 hr16 hr16 hr2 hr2 hr2 hr2 hr2 hr2 hr2 hr2	65420321 66335711 65318401 64980682 141750111 141924825 140968995 33951127 34628738 36581891 36759403 36923832	65420381 66335843 65155919 141750228 141924926 14289270 33953284 34628822 36582713 36825332 37041937	n'a ENSG0000201999 ENSG0000261742 ENSG000020149337 ENSG0000201892 ENSG00000252015 ENSG00000239649 ENSG00000239649 ENSG00000212025 n'a ENSG00000171055 ENSG00000205221	RMST JB153694 JS_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548 LOC1002889 11 FEZ2 VIT	+	60 1332 291802 175237 117 101 1900275 2157 848 822 45929 118105
chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 140500000 33875000 33875000 33875000 33875000 33875000	66400000 66400000 142900000 37175000 37175000 37175000 37175000 37175000 37175000 37175000	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	hr16 hr16 hr17 hr2 hr2 hr2 hr2 hr2 hr2 hr2 hr2	65420321 66335711 65318401 64980682 141750111 141924825 33951127 34628738 36581891 36779403 36923832 37064840	65420381 66336843 65610203 65155919 141750228 141924926 33953284 34628822 36582713 36825332 37041937 37193615	n/a ENSG0000201999 ENSG0000261742 ENSG0000261742 ENSG00000252015 ENSG0000252015 ENSG0000252015 ENSG0000239649 ENSG0000212025 n/a ENSG0000171055 ENSG00000171055 ENSG00000115808	RMST JB153694 SS_rRNA LINC00922 CDH11 Y_RNA U6 LRP1B MYADML Mir_548 LOC1002889 11 FEZ2 VIT STRN	+	60 132 291802 17537 117 101 1900275 2157 84 84 822 45929 118105 110160
chr16 chr16 chr2 chr2 chr2 chr2 chr2 chr2 chr2 chr2	63375000 63375000 140500000 140500000 33875000 33875000 33875000 33875000 33875000 33875000 33875000 33875000	66400000 66400000 142900000 142900000 37175000 37175000 37175000 37175000 37175000 37175000 37175000 37175000	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	hr16 hr16 hr12 hr2 hr2 hr2 hr2 hr2 hr2 hr2 hr	65420321 66335711 65318401 64980682 141750111 141924825 140988995 33951127 34628738 36581891 36779403 36923832 37064840 36583369	66420381 66335843 65610203 65155919 141750228 141924926 142899270 33953284 34628822 36582713 36825332 37041937 37193615 36778278	n/a ENSG0000201999 ENSG0000261742 ENSG000020140937 ENSG0000201892 ENSG00000252015 ENSG00000239649 ENSG00000212025 n/a ENSG00000171055 ENSG00000171055 ENSG000001715808 ENSG0000159038	RMST JB153694 SS_rRNA LINC00922 CDH11 Y_RNA U6 LRP18 MYADML Mir_548 LOC1002889 11 FEZ2 VIT STRN CRIM1	+	60 132 291802 175237 117 101 1900275 2157 84 822 45929 118105 110160 194909
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chr13	30025000	31700000	6chr13	30510667	30524625ENSG00000122043	LINC00544	+	13958
chr13	30025000	31700000	6chr13	31480311	31499709ENSG00000102802	MEDAG	+	19398
chr13	30025000	31700000	6chr13	31506833	31549153ENSG00000175664	TEX26	+	42320
chr13	30025000	31700000	6chr13	29598747	30080084ENSG00000132938	MTUS2	+	55084
chr13	30025000	31700000	6chr13	30083550	30169825ENSG00000139514	SLC7A1	-	86275
chr13	30025000	31700000	6chr13	30776766	30881191ENSG00000102781	KATNAL1	-	104425
chr13	30025000	31700000	6chr13	30914406	30948036ENSG00000238121	LINC00426	-	33630
chr13	30025000	31700000	6chr13	31191829	31233686ENSG00000132952	USPL1	+	41857
chr13	30025000	31700000	6chr13	31287614	31338565ENSG00000132965	ALOX5AP	+	50951
chr13	30025000	31700000	6chr13	30338544	30424820ENSG00000122042	UBL3	-	86276
chr13	30025000	31700000	6chr13	31456971	31506745ENSG00000224743	TEX26-AS1	-	49774

Table 3.7 | TADs expanded in neurons compared to glia.

TAD calls in neurons that encompass multiple TADs in glia (Table 3.7) and NPCs (Table 3.8). Chr(Neu TAD), x1 (Neu TAD), x2 (Neu TAD) – coordinates for TADs in neurons; No. Overlap Glia TADs, number of TADs in glia that overlap with the TAD called in neurons (columns A-C). Chr(Glia/NPC gene), x1 (Glia/NPC gene), x2 (Glia/NPC gene) – coordinates for genes located in TADs in column D in glia (Table 3.7) and NPCs (Table 3.8), respectively. Gene ID (ENSG), ENSEMBL gene IDs.

chr10 chr6 chr9 chr10	(Neu TAD) 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 6010000 20525000 6775	x2(Neu TAD) 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 9925000 9025000	TADs 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 8 8 8	gene) chr10 chr110 chr111 chr111 chr111 chr111 chr111 chr111 chr111 chr111 chr111 chr111 </th <th>Gene) 60474774 61019597 61821742 61019597 61821742 61821742 61841498 60094738 60144902 61410521 61496747 61715187 60036347 6105888 61548505 61786055 20567407 22134830 21563971 22146832 24171982 20534687 21666674 6834701 7167235 9442059 7796490 6757640 83134</th> <th>Gene) 60477293 61019717 61822047 61841612 60130513 60158990 61469649 61513203 61720671 60588845 61007534 61122352 61666414 62149634 20567491 224570750 24358512 24147757 22194616 24358820 24358512 24358251 2435851 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24355551 24355551 24355551 243555555555555555555555555555555555555</th> <th>Gene ID (ENSG) ENSG0000183055 n/a n/a ENSG0000022190 ENSG0000072401 ENSG00000186449 ENSG00000238731 ENSG00000238931 ENSG00000238931 ENSG0000012870 ENSG00000186443 ENSG00000186443 ENSG0000018981 ENSG0000018981 ENSG0000018091 ENSG00000181510 n/a ENSG00000124766 ENSG00000124766 ENSG00000124768 ENSG00000124768 ENSG00000124768 ENSG00000124768 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000025065 ENSG0000025065 <t< th=""><th>Gene Symbol FAM133CP Mir_584 Metazoa_SRP Y_RNA UBE2D1 TFAM SLC16649 LINC00948 C10orf40 BICC1 PHYHIPL FAM13C CCDC6 ANK3 Mir_548 LOC729177 HDGFL1 KAAG1 SOX4 PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DGS0140 Mir_584 AK094342 SNORDZ7 Metazoa_SRP</th><th>Strand + r\/a r\/a + - - - + - + + + + + + + + + +</th><th>(bp) 2519 2519 120 3051 114 30513 14088 59128 16456 5484 315942 71187 728 288</th></t<></th>	Gene) 60474774 61019597 61821742 61019597 61821742 61821742 61841498 60094738 60144902 61410521 61496747 61715187 60036347 6105888 61548505 61786055 20567407 22134830 21563971 22146832 24171982 20534687 21666674 6834701 7167235 9442059 7796490 6757640 83134	Gene) 60477293 61019717 61822047 61841612 60130513 60158990 61469649 61513203 61720671 60588845 61007534 61122352 61666414 62149634 20567491 224570750 24358512 24147757 22194616 24358820 24358512 24358251 2435851 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24358551 24355551 24355551 24355551 243555555555555555555555555555555555555	Gene ID (ENSG) ENSG0000183055 n/a n/a ENSG0000022190 ENSG0000072401 ENSG00000186449 ENSG00000238731 ENSG00000238931 ENSG00000238931 ENSG0000012870 ENSG00000186443 ENSG00000186443 ENSG0000018981 ENSG0000018981 ENSG0000018091 ENSG00000181510 n/a ENSG00000124766 ENSG00000124766 ENSG00000124768 ENSG00000124768 ENSG00000124768 ENSG00000124768 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG00000272188 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000022506 ENSG0000025065 ENSG0000025065 <t< th=""><th>Gene Symbol FAM133CP Mir_584 Metazoa_SRP Y_RNA UBE2D1 TFAM SLC16649 LINC00948 C10orf40 BICC1 PHYHIPL FAM13C CCDC6 ANK3 Mir_548 LOC729177 HDGFL1 KAAG1 SOX4 PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DGS0140 Mir_584 AK094342 SNORDZ7 Metazoa_SRP</th><th>Strand + r\/a r\/a + - - - + - + + + + + + + + + +</th><th>(bp) 2519 2519 120 3051 114 30513 14088 59128 16456 5484 315942 71187 728 288</th></t<>	Gene Symbol FAM133CP Mir_584 Metazoa_SRP Y_RNA UBE2D1 TFAM SLC16649 LINC00948 C10orf40 BICC1 PHYHIPL FAM13C CCDC6 ANK3 Mir_548 LOC729177 HDGFL1 KAAG1 SOX4 PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DGS0140 Mir_584 AK094342 SNORDZ7 Metazoa_SRP	Strand + r\/a r\/a + - - - + - + + + + + + + + + +	(bp) 2519 2519 120 3051 114 30513 14088 59128 16456 5484 315942 71187 728 288
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Metazoa_SRP</td><td>+ +</td><td>30513 14088 59128 16456 5484 315942 771187 71187 116464 117909 333579 84 12592 1073 1382 4878 1362 10732 1382 4878 1361 1362 1072 21344 47734 186298 196498 196298 196598 196598 196598 196598 196598 196598 196598</td></td>	62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 62425000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9225000 9225000 9225000 9225000 9225000 9225000 <td>9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9</td> <td>chr10 chr10 chr6 chr9 chr9</td> <td>60094738 60144902 61410521 61470521 61715187 60936347 61005888 61548505 20567407 22134830 22569677 22134830 22569677 24357130 22569677 24357130 21593971 222847472 24126413 22146882 24171982 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796409 6757640</td> <td>60130513 60158990 61469649 61513203 61720671 60588845 61007534 61122352 61666414 20567491 22147422 22570750 24358512 24358512 24358549 2230082 24147451 22198849 2230082 24147757 22194616 835011 7167357 8861724 8861724 8888207 9442347</td> <td>ENSG0000072401 ENSG0000018064 ENSG00000180649 ENSG00000238931 ENSG00000238931 ENSG00000185449 ENSG00000184541 ENSG00000164541 ENSG00000184561 ENSG00000181150 In/a ENSG00000181150 ENSG00000181273 ENSG00000124766 ENSG00000124766 ENSG00000124768 ENSG00000124768 ENSG00000127168 ENSG00000125676</td> <td>UBE2D1 TFAM SLC16A9 LINC00948 C10orf40 BICC1 PHYHIPL FAM13C CCDC6 ANK3 Mir_548 LOC729177 HDGFL1 KAAG1 SOX4 PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP</td> <td>+ +</td> <td>30513 14088 59128 16456 5484 315942 771187 71187 116464 117909 333579 84 12592 1073 1382 4878 1362 10732 1382 4878 1361 1362 1072 21344 47734 186298 196498 196298 196598 196598 196598 196598 196598 196598 196598</td>	9 9 9 9 9 9 9 9 9 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chr6 chr6 chr6 chr6 chr6 chr6 chr6 chr6 chr9 chr10 chr10 chr10 chr10 chr10 chr10 chr10 chr10 chr10 chr4 chr4 chr4 chr4 chr4 chr4 <td>20525000 20525000 20525000 20525000 20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000</td> <td>24375000 24375000 24375000 24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000</td> <td>8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8</td> <td>chr6 chr6 chr6 chr6 chr6 chr6 chr9 chr9</td> <td>22287472 24126413 22146882 24171982 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640</td> <td>22303082 24147757 22194616 24358280 21232634 22194616 6835011 7167357 8861724 8888207 9442347</td> <td>ENSG0000124766 ENSG0000172179 ENSG00000152954 ENSG00000152954 ENSG00000145096 ENSG00000145096 ENSG00000230581 r/a ENSG00000230581 ENSG00000225766 ENSG00000225766</td> <td>PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP</td> <td>+ - + + + + + + + + + + + + + +</td> <td>4878 15610 21344 47734 186298 697947 527942 310 122 3591 72</td>	20525000 20525000 20525000 20525000 20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000	24375000 24375000 24375000 24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000	8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8	chr6 chr6 chr6 chr6 chr6 chr6 chr9	22287472 24126413 22146882 24171982 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640	22303082 24147757 22194616 24358280 21232634 22194616 6835011 7167357 8861724 8888207 9442347	ENSG0000124766 ENSG0000172179 ENSG00000152954 ENSG00000152954 ENSG00000145096 ENSG00000145096 ENSG00000230581 r/a ENSG00000230581 ENSG00000225766 ENSG00000225766	PRL NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	+ - + + + + + + + + + + + + + +	4878 15610 21344 47734 186298 697947 527942 310 122 3591 72
chr6	20525000 20525000 20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000	24375000 24375000 24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000	8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 7 7	chr6 chr6 chr6 chr6 chr6 chr9	24126413 22146882 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640	24147757 22194616 24358280 21232634 22194616 6835011 7167357 8861724 8888207 9442347	ENSG0000152954 ENSG0000272168 ENSG00000146038 ENSG00000145996 ENSG00000272168 ENSG00000230581 n/a ENSG00000225706 ENSG00000225706	NRSN1 LINC00340 DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	+ + + + + + + n/a + +	21344 47734 186298 697947 527942 310 122 3591 72
chr6	20525000 20525000 20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000	24375000 24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000	8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 7 7 7	chr6 chr6 chr6 chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr9	22146882 24171982 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640	22194616 24358280 21232634 22194616 6835011 7167357 8861724 8888207 9442347	ENSG0000272168 ENSG0000146038 ENSG0000145996 ENSG00000272168 ENSG00000230581 n/a ENSG00000225706 ENSG00000251699	LINC00340 DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	+ - + + + + n/a + +	47734 186298 697947 527942 310 122 3591 72
chr6	20525000 20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000	24375000 24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000	8 8 8 8 8 8 8 8 8 8 8 8 8 7 7 7	chr6 chr6 chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr9	24171982 20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640	24358280 21232634 22194616 6835011 7167357 8861724 8888207 9442347	ENSG0000146038 ENSG00000145996 ENSG00000272168 ENSG00000230581 n/a ENSG00000225706 ENSG00000251699	DCDC2 CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	- + + + + n/a + +	186298 697947 527942 310 122 3591 72
cht6	20525000 20525000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000	24375000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000	8 8 8 8 8 8 8 8 8 8 8 8 7 7 7	chr6 chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr9	20534687 21666674 6834701 7167235 8858133 8888135 9442059 7796490 6757640	21232634 22194616 6835011 7167357 8861724 8888207 9442347	ENSG00000145996 ENSG00000272168 ENSG00000230581 n/a ENSG00000225706 ENSG00000251699	CDKAL1 LINC00340 DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	+ + n/a + +	697947 527942 310 122 3591 72
chr9 chr10 chr4	6775000 6775000 6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000 60100000 60100000	9225000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000	8 8 8 8 8 8 8 8 7 7 7	chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr9	6834701 7167235 8858133 8888135 9442059 7796490 6757640	6835011 7167357 8861724 8888207 9442347	ENSG00000230581 n/a ENSG00000225706 ENSG00000251699	DQ580140 Mir_584 AK094342 SNORD27 Metazoa_SRP	+ n/a + +	310 122 3591 72
chi9 chi9 chi9 chi9 chi9 chi9 chi9 chi9 chi9 chi10 chi10 chi4 chi4 chi4 chi4 chi4 chi4 chi4 chi4 chi4	6775000 6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000 60100000 60100000	9925000 9925000 9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000	8 8 8 8 8 8 8 7 7 7	chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr9	7167235 8858133 8888135 9442059 7796490 6757640	7167357 8861724 8888207 9442347	n/a ENSG00000225706 ENSG00000251699	Mir_584 AK094342 SNORD27 Metazoa_SRP	n/a + +	122 3591 72
chr9 chr9 chr9 chr9 chr9 chr9 chr9 chr10 chr10 chr14 chr4 chr4	6775000 6775000 6775000 6775000 6775000 60100000 60100000 60100000 60100000 60100000	9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000 61800000	8 8 8 8 8 8 7 7 7	chr9 chr9 chr9 chr9 chr9 chr9 chr9	8858133 8888135 9442059 7796490 6757640	8861724 8888207 9442347	ENSG00000225706 ENSG00000251699	AK094342 SNORD27 Metazoa_SRP	++++	3591 72
chr9 chr9 chr9 chr9 chr9 chr10 chr110 chr14 chr4	6775000 6775000 6775000 6775000 60100000 60100000 60100000 60100000 60100000	9925000 9925000 9925000 9925000 9925000 61800000 61800000 61800000 61800000	8 8 8 8 7 7 7	chr9 chr9 chr9 chr9 chr9 chr9	8888135 9442059 7796490 6757640	8888207 9442347	ENSG00000251699	SNORD27 Metazoa_SRP	+	72
chr9 chr9 chr9 chr10 chr10 chr14 chr4 chr4	6775000 6775000 60100000 60100000 60100000 60100000 60100000	9925000 9925000 9925000 61800000 61800000 61800000 61800000	8 8 7 7	chr9 chr9 chr9	7796490 6757640		ENSG00000265735		+	200
chr9 chr9 chr10 chr4	6775000 6775000 60100000 60100000 60100000 60100000 60100000	9925000 9925000 61800000 61800000 61800000 61800000	8 8 7 7	chr9 chr9	6757640	7799799				
chr10 chr4	6775000 60100000 60100000 60100000 60100000 60100000	9925000 61800000 61800000 61800000 61800000 61800000	8 7 7	chr9			ENSG00000137038	C9orf123	-	3309 400648
chr10 chr4	60100000 60100000 60100000 60100000 60100000	61800000 61800000 61800000 61800000	7			7175648 10612723	ENSG00000107077 ENSG00000153707	KDM4C PTPRD	+	400648
chr10 chr4	60100000 60100000 60100000	61800000 61800000 61800000			60474774	60477293	ENSG00000183055	FAM133CP	+	2519
chr10 chr4	60100000 60100000	61800000		chr10	61019597	61019717	n/a	Mir_584	n/a	120
chr10 chr4	60100000		7	chr10	60094738	60130513	ENSG0000072401	UBE2D1	+	30513
chr10 chr10 chr10 chr10 chr10 chr10 chr4			7	chr10 chr10	60144902 61410521	60158990 61469649	ENSG00000108064 ENSG00000165449	TFAM SLC16A9	+	14088 59128
chr10 chr10 chr10 chr10 chr10 chr10 chr4		61800000	7	chr10	61496747	61513203	ENSG00000227877	LINC00948	-	16456
chr10 chr10 chr10 chr10 chr4	60100000	61800000	7	chr10	61715187	61720671	ENSG00000235931	C10orf40	-	5484
chr10 chr10 chr4	60100000	61800000	7	chr10	60272903	60588845	ENSG00000122870	BICC1	+	315942
chr10 chr10 chr4	60100000 60100000	61800000 61800000	7	chr10 chr10	60936347 61005888	61007534 61122352	ENSG00000165443 ENSG00000148541	PHYHIPL FAM13C	+	71187 116464
chr10 chr4 chr4 chr4 chr4 chr4 chr4 chr4 chr4	60100000	61800000	7	chr10	61548505	61666414	ENSG00000108091	CCDC6	-	117909
chr4 chr4 chr4 chr4 chr4 chr4 chr4 chr4	60100000	61800000	7	chr10	61786055	62149634	ENSG00000151150	ANK3	-	13945
chr4 chr4 chr4 chr4 chr4 chr4 chr4 chr4	52675000	55125000	7	chr4	53578620	53580305	ENSG00000226950	DANCR	+	1685
chr4 chr4 chr4 chr4 chr4 chr4	52675000 52675000	55125000 55125000	7	chr4 chr4	53578848 53579415	53578914 53579537	ENSG00000264585 ENSG00000212588	MIR4449 SNORA26	+ +	66 122
chr4 chr4 chr4 chr4 chr4	52675000	55125000	7	chr4	53609683	53617807	ENSG00000212588 ENSG00000226887	ERVMER34-1	-	8124
chr4 chr4	52675000	55125000	7	chr4	53728494	53733002	ENSG00000128045	RASL11B	+	4508
chr4	52675000	55125000	7	chr4	54851665	54853449	ENSG00000229585	RPL21P44	-	1784
	52675000 52675000	55125000 55125000	7	chr4 chr4	54966247 52709275	54968122 52783003	ENSG00000180613 ENSG00000109184	GSX2 DCUN1D4	+	1875 73728
chr4	52675000	55125000	7	chr4	52859865	52883786	ENSG00000188993	LRRC66	-	23921
chr4	52675000	55125000	7	chr4	52886860	52904485	ENSG00000163069	SGCB	-	17625
chr4	52675000	55125000	7	chr4	53656160	53681631	ENSG00000250302	LOC152578	+	25471
chr4	52675000	55125000	7	chr4	53811820	53824254	ENSG00000248115	AK055055 FIP1L1	+	12434
chr4 chr4	52675000 52675000	55125000 55125000	7	chr4 chr4	54294193 54366243	54326103 54389443	ENSG00000145216 ENSG00000250930	LNX1-AS1	+	31910 23200
chr4	52675000	55125000	7	chr4	54459122	54471548	ENSG00000248494	LNX1-AS2	+	12426
chr4	52675000	55125000	7	chr4	55095263	55164412	ENSG00000134853	PDGFRA	+	29737
chr4	52675000	55125000	7	chr4	52917592	52963458	ENSG00000163071	SPATA18	+	45866
chr4 chr4	52675000 52675000	55125000 55125000	7	chr4 chr4	53739150 54326436	54232242 54457753	ENSG00000184178 ENSG00000072201	SCFD2 LNX1	-	493092 131317
chr4	52675000	55125000	7	chr4	54875957	54930815	ENSG00000109220	CHIC2	-	54858
chr4	52675000	55125000	7	chr4	53457126	53525502	ENSG00000109189	USP46	-	68376
chr5	162950000	167650000	7	chr5	165036445	165036684	ENSG00000252794	7SK	+	239
chr5 chr5	162950000 162950000	167650000 167650000	7	chr5 chr5	167460095 163781145	167460210 164029423	ENSG00000253065 ENSG00000241956	SNORA40 BC011998	- +	115 248278
chr5	162950000	167650000	7	chr5	166711842	167691162	ENSG00000241956	TENM2	+	938158
chr9	97300000	99100000	7	chr9	97572243	97572339	ENSG00000252153	MIR2278	+	96
chr9	97300000	99100000	7	chr9	97781172	97784562	ENSG00000148120	AL137535	+	3390
chr9	97300000	99100000	7	chr9	97847489	97848370	ENSG00000207563	MIR23B	+	881
chr9 chr9	97300000 97300000	99100000 99100000	7	chr9 chr9	97848295 97884155	97848376 97886342	ENSG00000207617 n/a	MIR3074 BC041030	- n/a	81 2187
chr9	97300000	99100000	7	chr9	98225890	98232301	n/a	LOC100507346	n/a	6411
chr9	97300000	99100000	7	chr9	98680445	98680471	ENSG00000237631	DQ588075	+	26
chr9	97300000	99100000	7	chr9	98782013	98784037	ENSG00000225194	LINC00092	-	2024
chr9 chr9	97300000	99100000 99100000	7	chr9 chr9	97317422 97321002	97330409 97356075	ENSG00000231806 ENSG00000130957	BC080653 FBP2	+	12987 35073
chr9		99100000	7	chr9	98205263	98270831	ENSG00000185920	PTCH1	-	65568
chr9	97300000 97300000	99100000	7	chr9	98568369	98638259	ENSG00000175611	LINC00476	-	69890
chr9	97300000 97300000 97300000		7	chr9	98868942	98878693	n/a	LOC158434	n/a	9751
chr9 chr9	97300000 97300000	99100000 99100000	7	chr9 chr9	98997588 97365420	99064434 97401923	ENSG00000130948 ENSG00000165140	HSD17B3 FBP1	-	66846 36503

chr9	97300000	99100000	7	chr9	97521930	97849500	ENSG00000148120	C9orf3	+	327570
chr9	97300000	99100000	7	chr9	97861335	98079991	ENSG00000158169	FANCC	-	218656
chr9	97300000	99100000	7	chr9	98637899	98731122	ENSG00000182150	ERCC6L2	+	93223
chr9	97300000	99100000	7	chr9	98828120	98864194	ENSG00000237212	LOC158435	+	36074
chr9	97300000	99100000	7	chr9	99082987	99145992	ENSG00000130958	SLC35D2	-	17013
chr1	71550000	75175000	6	chr1	72259914	72302695	ENSG00000228853	NEGR1-IT1	-	42781
chr1	71550000	75175000	6	chr1	75043113	75091782	ENSG00000234497	CR627203	+	48669
chr1	71550000	75175000	6	chr1	75171171	75199092	ENSG00000116791	CRYZ	-	3829
chr1	71550000	75175000	6	chr1	71547006	71703406	ENSG00000229956	ZRANB2-AS2	+	153406
chr1	71550000	75175000	6	chr1	73771852	73804560	ENSG00000233973	BC041341	+	32708
chr1	71550000	75175000	6	chr1	74491701	74663871	ENSG00000162620	LRRIQ3	-	172170
chr1	71550000	75175000	6	chr1	74663895	75010116	ENSG00000116783	FPGT-TNNI3K	+	346221
chr1	71550000	75175000	6	chr1	75033794	75139422	ENSG00000178965	C1orf173	-	105628
chr1	71550000	75175000	6	chr1	71868624	72748405	ENSG00000172260	NEGR1	-	879781
chr1	95650000	98950000	6	chr1	95699710	95712781	ENSG00000122481	RWDD3	+	13071
chr1	95650000	98950000	6	chr1	95970531	95970615	ENSG00000216037	Mir 548	-	84
chr1	95650000	98950000	6	chr1	97161411	97161723	ENSG00000223229	7SK	-	312
chr1	95650000	98950000	6	chr1	98510798	98510907	ENSG00000225206	MIR2682	-	109
chr1	95650000	98950000	6	chr1	95582893	95663161	ENSG00000152078	TMEM56	+	13161
chr1	95650000	98950000	6	chr1	95975895	95981020	ENSG00000228971	BC067883	+	5125
chr1	95650000	98950000	6	chr1	98453555	98515249	ENSG00000225206	MIR137HG		61694
chr1	95650000	98950000	6	chr1	95628774	95699538	ENSG00000226026	AK090700	-	49538
chr1	95650000	98950000	6	chr1	95940292	95944912	ENSG0000233907	FLJ31662	+	4620
chr1	95650000	98950000	6	chr1	97187174	97280605	ENSG00000117569	PTBP2	+	93431
chr1	95650000	98950000	6	chr1	97543299	98386615	ENSG00000188641	DPYD	-	843316
chr1	95650000	98950000	6	chr1	97561478	97788511	ENSG00000232878	DPYD-AS1	+	227033
chr1	95650000	98950000	6	chr1	98676266	98738214	ENSG00000226053	LOC729987	+	61948
chr10	60225000	61375000	6	chr10	60474774	60477293	ENSG00000220035	FAM133CP	+	2519
chr10	60225000	61375000	6	chr10	61019597	61019717	n/a	Mir 584	n/a	120
chr10	60225000	61375000	6	chr10	60272903	60588845	ENSG00000122870	BICC1	1//a +	315942
chr10 chr10	60225000	61375000	6	chr10 chr10	60936347	61007534	ENSG00000122870	PHYHIPL	+ +	71187
chr10 chr10	60225000	61375000	6	chr10	61005888	61122352	ENSG00000165443 ENSG00000148541	FAM13C	+	116464
chr10 chr10	60475000	61375000	6	chr10 chr10	60474774	60477293	ENSG00000148541 ENSG00000183055	FAM13C FAM133CP	+	2293
chr10 chr10	60475000	61375000	6	chr10 chr10	61019597	61019717	n/a ENSC00000122870	Mir_584 BICC1	n/a +	120
chr10 chr10	60475000	61375000	6	chr10	60272903 60936347	60588845	ENSG00000122870	BICC1		113845
chr10	60475000	61375000 61375000	6	chr10		61007534	ENSG00000165443	PHYHIPL FAM12C	+	71187
chr10	60475000 69725000	72125000	6	chr10	61005888	61122352	ENSG00000148541 ENSG00000264405	FAM13C	-	116464
chr12			6	chr12	69978501	69978603		MIR3913-1	-	102
chr12	69725000 69725000	72125000 72125000	6	chr12	69978502	69978602	n/a	MIR3913-2	n/a	100
chr12			6	chr12	70002344	70004942	ENSG00000198812	LRRC10	-	2598
chr12	69725000	72125000	6	chr12	70195707		n/a	Mir_548	n/a	84
chr12	69725000	72125000	6	chr12	71300696	71300798	ENSG00000207387	Y_RNA	+	102
chr12	69725000	72125000	6	chr12	71869656	71869910	n/a	7SK	n/a	254
chr12	69725000	72125000	6	chr12	69742133	69748013	ENSG0000090382	LYZ	+	5880
chr12	69725000	72125000	6	chr12	69753531	69784576	ENSG00000127337	YEATS4	+	31045
chr12	69725000	72125000	6	chr12	69864128	69973562	ENSG00000166225	FRS2	+	109434
chr12	69725000	72125000	6	chr12	70047388	70093196	ENSG00000127325	BEST3	-	45808
chr12	69725000	72125000	6	chr12	70132630	70216984	ENSG00000127328	RAB3IP	+	84354
chr12	69725000	72125000	6	chr12	70320436	70352503	ENSG00000166268	C12orf28	+	32067
chr12	69725000	72125000	6	chr12	70910631	71031220	ENSG00000127329	PTPRB	-	120589
chr12	69725000	72125000	6	chr12	71518876	71551779	ENSG00000127324	TSPAN8	-	32903
chr12	69725000	72125000	6	chr12	72003378	72057749	ENSG00000133858	ZFC3H1	-	54371
chr12	69725000	72125000	6	chr12	72057676	72074428	ENSG00000173451	THAP2	+	16752
chr12	69725000	72125000	6	chr12	69979207	69995357	ENSG00000166226	CCT2	+	16150
chr12	69725000	72125000	6	chr12	70107414	70132348	ENSG00000247131	BC042465	-	24934
chr12	69725000	72125000	6	chr12	70636773	70748773	ENSG00000111596	CNOT2	+	112000
chr12	69725000	72125000	6	chr12	70760061	70828072	ENSG00000135643	KCNMB4	+	68011
chr12	69725000	72125000	6	chr12	70861864	70932592	ENSG00000258168	BC031864	+	70728
chr12	69725000	72125000	6	chr12	71833549	71980088	ENSG00000139292	LGR5	+	146539
chr12	69725000	72125000	6	chr12	72079877	72097839	ENSG00000139291	TMEM19	+	17962
chr12	69725000	72125000	6	chr12			EN3G00000139291		т	
chr12	96925000		0		71031852	71314584	ENSG00000153233	PTPRR	-	282732
chr12		98800000	6	chr12	97490690	97490764	ENSG00000153233 n/a	TRNA_Pseudo	- n/a	74
chr12	96925000	98800000 98800000		chr12 chr12		97490764 97507042	ENSG00000153233	TRNA_Pseudo Mir_584	-	
	96925000 96925000		6		97490690	97490764	ENSG00000153233 n/a n/a n/a	TRNA_Pseudo	- n/a	74
chr12		98800000	6 6	chr12	97490690 97506929	97490764 97507042	ENSG00000153233 n/a n/a	TRNA_Pseudo Mir_584	- n/a n/a	74 113
	96925000 96925000	98800000 98800000 98800000	6 6 6	chr12 chr12 chr12	97490690 97506929 97866326 97885686	97490764 97507042 97868402 97885756	ENSG0000153233 n/a n/a n/a ENSG00000221479	TRNA_Pseudo Mir_584 AK129935	- n/a n/a n/a	74 113 2076 70
chr12	96925000 96925000 96925000	98800000 98800000 98800000 98800000	6 6 6 6	chr12 chr12 chr12 chr12	97490690 97506929 97866326 97885686 97945914	97490764 97507042 97868402 97885756 97946006	ENSG00000153233 n/a n/a eNSG00000221479 ENSG00000251844	TRNA_Pseudo Mir_584 AK129935 MIR1251 SnoMe28S_Am263 4	- n/a n/a + -	74 113 2076 70 92
chr12 chr12	96925000 96925000 96925000 96925000 96925000	98800000 98800000 98800000 98800000 98800000 98800000	6 6 6 6 6	chr12 chr12 chr12 chr12 chr12 chr12	97490690 97506929 97866326 97885686 97945914 97957589	97490764 97507042 97868402 97885756 97946006 97957689	ENSG00000153233 n/a n/a ENSG00000221479 ENSG00000251844 ENSG00000207586	TRNA_Pseudo Mir_584 AK129935 MIR1251 SnoMe28S_Am263 4 MIR135A2	- n/a n/a n/a	74 113 2076 70 92 100
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chr12 chr12 chr12 chr12	96925000 96925000 96925000 96925000 96925000 96925000	98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000	6 6 6 6 6	chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12	97490690 97506929 97866326 97885686 97945914 97957589 98389160 98752079	97490764 97507042 97868402 97885756 97946006 97957689 98389226 98752163	ENSG0000153233 n/a n/a ENSG00000221479 ENSG00000251844 ENSG0000025186 ENSG00000263890 n/a	TRNA_Pseudo Mir_584 AK129935 MIR1251 SnoMe28S_Am263 4 MIR135A2 MIR1303 Mir_548	- n/a n/a + -	74 113 2076 70 92 100
chr12 chr12 chr12 chr12 chr12 chr12	96925000 96925000 96925000 96925000 96925000 96925000 96925000	98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000	6 6 6 6 6 6 6 6	chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12	97490690 97506929 97866326 97885686 97945914 97957589 98389160 98752079 96883352	97490764 97507042 97868402 97885756 97946006 97957689 98389226 98752163 96934765	ENSG0000153233 n/a n/a eNSG0000221479 ENSG0000251844 ENSG0000207586 ENSG0000263890 n/a ENSG0000188596	TRNA_Pseudo Mir_584 AK129935 MIR1251 SnoMe28S_Am263 4 MIR135A2 MIR4303 Mir_548 C12ort55	- n/a n/a + - + - n/a +	74 113 2076 70 92 100 66 84 9765
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chr12 chr14 chr14 chr14 chr14 chr16 chr18 chr18 chr18 chr18 chr18 chr18 chr18 chr18 chr18 chr2 chr2	96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 25850000 25850000 25850000 25850000 25850000 25850000 25850000 49100000 49100000 49100000 49350000 103425000 103425000	98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 29100000 29100000 29100000 29100000 29100000 29100000 29100000 29100000 51725000000 5172500000000000000000000000000000000000	$\begin{array}{c} 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ $	chr12 chr14 chr14 chr14 chr14 chr16 chr18 chr18 chr18 chr18 chr12 chr14	97490690 97506929 97866326 97885686 97885686 97885686 97857589 98389160 98752079 96883352 97301000 98123825 97301000 98123825 97301000 98123825 97301000 98123825 97858798 976477847 296833914 97041752 97858798 26641372 220815088 220915088 22091794 28085032 51677970 49866541 51105992 51677970 49866541 51105992 51677970 49866541 51105992 51677970	97490764 97507042 97868402 97885756 97946006 97957689 98383226 98752163 99834765 97347469 98150295 97024429 97150032 97927544 97927544 297927544 29792754 27065960 28142425 79634622 779634622 779634622 779634622 779634622 779634622 7107426 5110745 511075 511075	ENSG0000153233 n/a n/a n/a n/a ENSG0000221479 ENSG0000221479 ENSG00002251844 ENSG00002251844 ENSG0000257801 ENSG0000188596 ENSG00000188596 ENSG0000018596 ENSG0000018596 ENSG0000025754 ENSG0000025754 ENSG0000025794 ENSG0000025794 ENSG0000025794 ENSG00000258748 ENSG00000258748 ENSG00000258748 ENSG00000258748 ENSG00000258748 ENSG00000258748 ENSG00000258748 ENSG00000189302 ENSG000001878273 ENSG00000187323 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG00000187233 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG0000187234 ENSG000028744 ENSG000028744 ENSG0000287444 ENSG000028744 ENSG0000287444 ENSG00000287444 ENSG0000087444 ENSG0000087444 ENSG000087444 ENSG000087444 EN	TRNA_Pseudo TRNA_Pseudo Mir_584 Akt129935 MIR1251 SnoMe28S_Am263 4 MIR135A2 MIR5303 Mir_548 C12orf55 NEDD1 LOC643711 AX747187 C12orf63 RMST Mir_548 DD413662 LINC00645 NOVA1 BC148262 MAF WWOX BC034434 MBD2 DCC DCC DD413674 POU3F3 AK095498	- n/a n/a n/a +	74 113 2076 70 92 100 66 84 9765 46469 26470 90515 117280 68746 84 84 84 84 22 27049 151872 351220 6878 111328 11328 11328 11328 1135732 1434 47030 1195732 20 503 6886
chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr12 chr14 chr14 chr14 chr14 chr14 chr14 chr14 chr16 chr18 chr12 chr2	96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 96925000 25850000 25850000 25850000 25850000 25850000 25850000 78125000 78125000 78125000 49100000 49350000 49350000 103425000 103425000 103425000	98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 98800000 29100000 29100000 29100000 29100000 29100000 29100000 29100000 29100000 517250000 5172500000000000000000000000000000000000	$\begin{array}{c} 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ 6\\ $	chr12 chr14 chr14 chr14 chr14 chr16 chr18 chr18 chr18 chr12 chr18 chr12 chr14	97490690 97506929 97866326 97886326 97885686 97885686 97885686 98389160 98752079 98683352 97301000 98123825 96633914 97041752 27377847 226641372 27377847 22063026 28081793 26915088 27791205 79627744 7813326 51105992 51677970 49866541 51105992 51677970 49866541 105471968	97490764 97507042 977668402 9785756 97957689 98389226 98389226 98752163 98389226 98752163 97347469 98150295 97024429 971759032 97027544 26641456 27377931 29663048 28142425 776634622 27066960 28142425 7151158 51062273 51107426 51751158 51062273 51107426	ENSG0000153233 n/a n/a n/a ENSG0000221479 ENSG0000221479 ENSG0000251844 ENSG0000251844 ENSG0000257804 ENSG0000257504 ENSG0000257501 ENSG00000257504 ENSG00000257594 ENSG00000255794 ENSG00000255794 ENSG00000255794 ENSG00000255794 ENSG00000255794 ENSG0000025615 ENSG00000256153 ENSG00000178573 ENSG00000178573 ENSG00000178733 ENSG00000178733 ENSG00000178733 ENSG00000134046 ENSG00000134046 ENSG00000134046 ENSG00000134046 ENSG00000134046 ENSG00000134046 ENSG00000134046	TRNA_Pseudo Mir_584 AK129935 MIR1251 SnoMe28S_Am263 4 MIR135A2 MIR135A2 MIR135A2 MIR135A2 MIR5303 Mir_548 C12orl65 NEDD1 LOC643711 AX747187 C12orl63 MIR4307 DD413682 LINC00645 DV413682 MAF WWOX BC034434 MBD2 DCC BC034434 MBD2 DCC DD413674 POU3F3 AK095498 LOC00287010	- n/a n/a n/a + + - - - + + + + + + + + - - + + - - + + - - + + - - - + - - - + - - - - + -	74 113 2076 70 92 100 66 84 9765 46469 26470 90515 117280 68746 84 227049 27049 27049 151872 351220 6878 1113238 1434 47030 1195732 1434 47030 1195732 20 1503 6886
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chr2	103425000	105550000	6	chr2	105050804	105129215	ENSG00000235597	LOC150568	+	78411
chr2	188350000	190325000	6	chr2	189162218	189162315	ENSG00000207951	MIR561	+	97
chr2	188350000	190325000	6	chr2	189842817	189842886	ENSG00000221502	MIR1245A	+	69
chr2	188350000	190325000	6	chr2	189842819	189842887	n/a	MIR1245B	n/a	68
chr2	188350000	190325000	6	chr2	189860355	189860418	n/a	MIR3606	n/a	63
chr2	188350000	190325000	6	chr2	189997761	189997837	ENSG00000264725	MIR3129	-	76
chr2	188350000	190325000	6	chr2	189598464	189654831	ENSG00000174325	DIRC1	-	56367
chr2	188350000	190325000	6	chr2	189839098	189877472	ENSG00000168542	COL3A1	+	38374
chr2	188350000	190325000	6	chr2	188328957	188419219	ENSG0000003436	TFPI	-	69219
chr2	188350000	190325000	6	chr2	189156395	189460652	ENSG00000144366	GULP1	+	304257
chr2	188350000	190325000	6	chr2	189896640	190044605	ENSG00000204262	COL5A2	-	147965
chr2	188350000	190325000	6	chr2	190306158	190340264	ENSG00000115368	WDR75	+	18842
chr2	33875000	37175000	6	chr2	33951127	33953284	ENSG00000239649	MYADML	-	2157
chr2	33875000	37175000	6	chr2	34628738	34628822	ENSG00000212025	Mir 548	-	84
chr2	33875000	37175000	6	chr2	36581891	36582713	n/a	LOC100288911	n/a	822
chr2	33875000	37175000	6	chr2	36779403	36825332	ENSG00000171055	FEZ2	-	45929
chr2	33875000	37175000	6	chr2	36923832	37041937	ENSG00000205221	VIT	+	118105
chr2	33875000	37175000	6	chr2	37064840	37193615	ENSG00000115808	STRN	-	110160
chr2	33875000	37175000	6	chr2	36583369	36778278	ENSG00000150938	CRIM1	+	194909
chr2	39550000	42025000	6	chr2	39620662	39620759	ENSG00000252239	U6	-	97
chr2	39550000	42025000	6	chr2	41559792	41559915	n/a	Mir_584	n/a	123
chr2	39550000	42025000	6	chr2	39741286	39828484	ENSG00000231312	LOC728730	+	87198
chr2	39550000	42025000	6	chr2	39893034	39945104	ENSG00000152154	TMEM178A	+	52070
chr2	39550000	42025000	6	chr2	39476406	39664453	ENSG0000011566	MAP4K3	-	114453
chr2	39550000	42025000	6	chr2	39963199	40006416	ENSG00000138050	THUMPD2	-	43217
chr2	39550000	42025000	6	chr2	40144773	40482349	ENSG00000227028	SLC8A1-AS1	+	337576
chr2	39550000	42025000	6	chr2	40339285	40657444	ENSG00000183023	SLC8A1	-	318159
chr2	62125000	64150000	6	chr2	62432960	62433052	ENSG00000266097	MIR5192	+	92
chr2	62125000	64150000	6	chr2	62489521	62489806	ENSG0000239958	Metazoa_SRP	-	285
chr2	62125000	64150000	6	chr2	62727355	62733604	ENSG00000239958	TMEM17	-	6249
chr2 chr2	62125000	64150000	6	chr2 chr2	62953770	62953875	ENSG00000186889	Y RNA	-	105
chr2 chr2	62125000	64150000	6	chr2 chr2	63271099	63274846	ENSG00000252436 ENSG00000231609	LOC100132215	-	3747
							ENSG00000231809	OTX1	+	7030
chr2	62125000	64150000 64150000	6	chr2	63277936	63284966			+	
chr2	62125000		6	chr2	63344985	63346677	ENSG00000242412	DBIL5P2 BC071802	1	1692
chr2	62125000	64150000	6	chr2	62296587	62374016	ENSG00000229839		1.	77429
chr2	62125000	64150000	6	chr2	62423261	62451866	ENSG00000170340	B3GNT2	+	28605
chr2	62125000	64150000	6	chr2	62839006	62889763	ENSG00000226622	BC038779 COMMD1	1.	50757
chr2	62125000	64150000	6	chr2	62132802	62363205	ENSG00000173163		+	230403
chr2	62125000	64150000	6	chr2	62933000	63273621	ENSG00000115504	EHBP1	+	340621
chr2	62125000	64150000	6	chr2	63348534	63815867	ENSG00000143951	WDPCP	-	467333
chr2	62125000	64150000	6	chr2	63816284	63834330	ENSG0000014641	MDH1	+	18046
chr2	62125000	64150000	6	chr2	64069013	64118696	ENSG00000169764	UGP2	+	49683
chr2	62125000	64150000	6	chr2	64119666	64246214	ENSG00000143952	VPS54	-	30334
chr21	30400000	32450000	6	chr21	30552745	30552960	ENSG00000212479	U3	-	215
chr21	30400000	32450000	6	chr21	31538240	31538971	ENSG00000156282	CLDN17	-	731
chr21	30400000	32450000	6	chr21	31581468	31584101	ENSG00000227342	LINC00307	-	2633
chr21	30400000	32450000	6	chr21	31661462	31661832	ENSG00000232263	KRTAP25-1	-	370
chr21	30400000	32450000	6	chr21	31691449	31692607	ENSG00000197683	KRTAP26-1	-	1158
chr21	30400000	32450000	6	chr21	31709330	31710012	ENSG00000206107	KRTAP27-1	-	682
chr21	30400000	32450000	6	chr21	31720716	31720924	ENSG00000186980	KRTAP23-1	-	208
chr21	30400000	32450000	6	chr21	31743708	31744557	ENSG00000182816	KRTAP13-2	-	849
chr21	30400000	32450000	6	chr21	31747611	31747696	ENSG00000265007	MIR4327	-	85
chr21	30400000	32450000	6	chr21	31797710	31798230	ENSG00000240432	KRTAP13-3	-	520
chr21	30400000	32450000	6	chr21	31802593	31803076	ENSG00000186971	KRTAP13-4	+	483
chr21	30400000	32450000	6	chr21	31812645	31813098	ENSG00000186970	KRTAP15-1	+	453
chr21	30400000	32450000	6	chr21	31852363	31852636	ENSG00000184351	KRTAP19-1	-	273
chr21	30400000	32450000	6	chr21	31859508	31859667	ENSG00000186965	KRTAP19-2	-	159
chr21	30400000	32450000	6	chr21	31863781	31864275	ENSG00000244025	KRTAP19-3	-	494
chr21	30400000	32450000	6	chr21	31869173	31869428	ENSG00000186967	KRTAP19-4	-	255
chr21	30400000	32450000	6	chr21	31874189	31874408	ENSG00000186977	KRTAP19-5	-	219
chr21	30400000	32450000	6	chr21	31913853	31914181	ENSG00000186925	KRTAP19-6	-	328
chr21	30400000	32450000	6	chr21	31933416	31933608	ENSG00000244362	KRTAP19-7	-	192
chr21	30400000	32450000	6	chr21	31962423	31962716	ENSG00000206106	KRTAP22-2	-	293
chr21	30400000	32450000	6	chr21	31971004	31971193	ENSG00000186930	KRTAP6-2	-	189
chr21	30400000	32450000	6	chr21	31973439	31973586	ENSG00000186924	KRTAP22-1	+	147
chr21	30400000	32450000	6	chr21	31986004	31986223	ENSG00000184724	KRTAP6-1	-	219
chr21	30400000	32450000	6	chr21	31988773	31988944	ENSG00000244624	KRTAP20-1	+	171
chr21	30400000	32450000	6	chr21	31992945	31993169	ENSG00000206105	KRTAP20-4	+	224
chr21	30400000	32450000	6	chr21	32007582	32007780	ENSG00000184032	KRTAP20-2	+	198
chr21	30400000	32450000	6	chr21	32015182	32015455	ENSG00000206104	KRTAP20-3	+	273
chr21	30400000	32450000	6	chr21	32090842	32091095	ENSG0000231068	KRTAP21-3	-	253
chr21	30400000	32450000	6	chr21	32119268	32119520	ENSG00000187026	KRTAP21-2	-	252
chr21	30400000	32450000	6	chr21	32127456	32127696	ENSG00000187005	KRTAP21-1	1 -	240
chr21	30400000	32450000	6	chr21	32185014	32185570	ENSG00000183640	KRTAP8-1	-	556
chr21	30400000	32450000	6	chr21	32201357	32202051	ENSG00000256386	KRTAP7-1	-	694
chr21	30400000	32450000	6	chr21	32252963	32253874	ENSG00000230380	KRTAP11-1	-	911
chr21	30400000	32450000	6	chr21	32410477	32410795	ENSG00000182391	KRTAP19-8	1.	318
chr21	30400000	32450000	6	chr21	30428647	30446010	ENSG00000156261	CCT8	-	17363
chr21	30400000	32450000	6	chr21	30565814	30660526	ENSG00000130201 ENSG00000215533	LINC00189	+	94712
chr21	30400000	32450000	6	chr21	30671219	30718469	ENSG000002155555 ENSG00000156273	BACH1	+	47250
chr21	30400000	32450000	6	chr21	30968359	31003067	ENSG00000136273	GRIK1-AS2	+	34708
chr21 chr21	30400000	32450000	6	chr21 chr21	31120493	31136325	ENSG00000174680	GRIK1-AS2	+	15832
chr21 chr21	30400000	32450000	6	chr21 chr21	31120493 31653626	31136325	ENSG00000174680 ENSG00000188694	KRTAP24-1	-	15832
	30400000	32450000						KRTAP24-1 KRTAP13-1	-	747
chr21			6	chr21	31768391	31769138	ENSG00000198390		+	
chr21	30400000	32450000	6	chr21	31964758	31965374	ENSG00000212938	KRTAP6-3	+	616
chr21	30400000	32450000	6	chr21	30452872	30548202	ENSG00000156265	MAP3K7CL	+	95330
chr21	30400000	32450000	6	chr21	30925865	31312282	ENSG00000171189	GRIK1	-	386417
chr21	30400000	32450000	6	chr21	31586323	31588469	ENSG00000156284	CLDN8	-	2146
chr21	30400000	32450000	6	chr21	30396937	30426807	ENSG00000156256	USP16	+	26807
	180425000	182400000	6	chr3	180586548	180588578	ENSG00000114416	BC034416	+	2030
chr3	180425000	182400000	6	chr3	180701497	180707562	ENSG00000205981	DNAJC19	-	6065
chr3 chr3		182400000	6	chr3	180949524	180949631	ENSG00000206932	U6	+	107
chr3	180425000									
chr3 chr3 chr3 chr3	180425000	182400000	6	chr3	181413636	181413690	n/a	JA611300	n/a	54
chr3 chr3 chr3 chr3 chr3 chr3	180425000 180425000	182400000 182400000	6 6	chr3 chr3	181429711	181432223	ENSG00000181449	SOX2	+	2512
chr3 chr3 chr3 chr3 chr3 chr3 chr3	180425000 180425000 180425000	182400000 182400000 182400000	6 6 6	chr3 chr3	181429711 181686246	181432223 181686469	ENSG00000181449 ENSG00000252257	SOX2 7SK	+ +	2512 223
chr3 chr3 chr3 chr3 chr3 chr3	180425000 180425000	182400000 182400000	6 6	chr3	181429711	181432223	ENSG00000181449	SOX2	+	2512

chr3	180425000	182400000	6	chr3	181670166	181721828	ENSG00000242512	BC036236	+	51662
chr3	180425000	182400000	6	chr3	180425376	180587966	ENSG00000145075	DKFZp434A128	-	162590
chr3	180425000	182400000	6	chr3	182164757	182204150	ENSG00000241098	FLJ46066	-	39393
chr3	180425000	182400000	6	chr3	180774467	181460013	ENSG00000242808	SOX2-OT	+	685546
chr3	58525000	62275000	6	chr3	59956575	59958982	n/a	NPCR	n/a	2407
chr3	58525000	62275000	6	chr3	60842060	60842277	ENSG00000212211	U3	-	217
chr3	58525000	62275000	6	chr3	61068514	61068637	n/a	5S_rRNA	n/a	123
chr3	58525000	62275000	6	chr3	58549844	58563491	ENSG00000168309	FAM107A	-	13647
chr3	58525000	62275000	6	chr3	58619669	58652561	ENSG00000198643	FAM3D	-	32892
chr3	58525000	62275000	6	chr3	58727736	59035715	ENSG00000163689	C3orf67	-	307979
chr3	58525000	62275000	6	chr3	58810196	59004819	ENSG00000242428	AK090895	+	194623
chr3	58525000	62275000	6	chr3	62247493	62304622	ENSG00000241472	PTPRG-AS1	-	27507
chr3	58525000	62275000	6	chr3	59735035	61237133	ENSG00000189283	FHIT	-	1502098
chr3	58525000	62275000	6	chr3	61547242	62280573	ENSG00000144724	PTPRG	+	727758

Table 3.8 | TADs expanded in neurons compared to NPC.

TAD calls in neurons that encompass multiple TADs in glia (Table 3.7) and NPCs (Table 3.8). Chr(Neu TAD), x1 (Neu TAD), x2 (Neu TAD) – coordinates for TADs in neurons; No. Overlap NPC TADs, number of TADs in NPC that overlap with the TAD called in neurons (columns A-C). Chr(Glia/NPC gene), x1 (Glia/NPC gene), x2 (Glia/NPC gene) – coordinates for genes located in TADs in column D in glia (Table 3.7) and NPCs (Table 3.8), respectively. Gene ID (ENSG), ENSEMBL gene IDs.

NEURONS												
Riskchr			Risk overlap			x2	chr2	y1	y2			NPCfdrDonu
chr10 chr10	104570118 104570118	105059896 105059896	5000 5000	chr10 chr10	104590000 104590000	104595000 104595000		104660000 104660000	104665000 104665000	0.4254983	0.06027781	0.14398974
chr10 chr18	52747689	53804156	5000	chr18		53080000	chr10 chr18	54240000	54245000	1.2518088	0.05274656	0.44168428
chr2				chr2	199180000	199190000	chr2		200730000	0.51119745	2.03E-08	
chr2		201309547	10000	chr2	200320000	200330000	chr2	200720000	200730000	1.5162903	0.00013409	1.5750872
chr22	39840130	40091818	10000	chr22	39720000	39730000	chr22	39910000	39920000	0.7976765	6.04E-05	0.8887403
chr4	176717618		10000		176480000	176490000			176740000	1.0397903	6.22E-05	
chr5	87676693	88195380	5000	chr5	88025000	88030000	chr5	88595000	88600000	1.1398252	0	1.3084083
chr5 chr5	88580998 137838122	88748452 137948140	5000 10000	chr5	88025000 137930000	88030000 137940000	chr5 chr5	88595000 138200000	88600000 138210000	1.1398252 1.2840025	1.70E-05	1.3084083
chr6	24988105	33842877	10000	chr6	25260000	25270000	chr6	25590000	25600000	0.20888025	0.05431648	0.40973878
chr6	24988105	33842877	10000		25260000	25270000	chr6	25590000	25600000	0.20888025	0.05431648	0.4097387
chr6	24988105	33842877	5000		26120000	26125000	chr6	27110000	27115000	1.0039818	0	1.2458384
chr6	24988105	33842877	5000	chr6	26120000	26125000	chr6	27110000	27115000	1.0039818	0	1.2458384
chr6	24988105	33842877	10000	chr6	28460000	28470000	chr6	28630000	28640000	1.5588554	5.11E-05	
chr6	24988105	33842877	10000		28460000	28470000		28630000	28640000	1.5588554	5.11E-05	0.98528725
chr7	110850439	111180544	10000	chr7	108260000	108270000	cnr/	111120000	111130000	1.4305197	0.01244852	1.6058803
GLIA	Diels start	Diele en d	Diele evenlen	a h a f		x2	- h - O			GliafdrDonut	NeufdaDeau	
Riskchr chr1	Risk start 8392592	Risk end 8701288	Risk overlap 10000	chr1	8430000	xz 8440000	chr2 chr1	8770000	y2 8780000	3.85E-06	0.44497925	0.792593
chr1	98341152	98559093	9093	chr1	98550000	98560000	chr1	98660000	98670000	0.00097779	1.5657233	1.724188
chr1	149998923	150214166		chr1	149935000	149940000	chr1	150020000	150025000	2.52E-05	1.060373	0.842383
chr1	200253612	200269903	9903	chr1	200260000	200270000	chr1	200450000	200460000	0.00100757	1.5745742	1.0504069
chr10	104570118		10000	chr10	104480000	104490000	chr10	104810000	104820000	0.00022482	0.90376765	0.3040932
chr10	104570118	105059896			104485000				104670000	0.01798354	1.2801908	
chr10		105059896	5000		104485000	104490000	chr10	104660000	104665000	6.75E-05	0.5831852	0.114014
chr10 chr10		105059896	5000		104590000	104595000	chr10		104680000	0.00142198	1.3248246	1.347813
chr10 chr10	104570118 104570118	105059896	5000 10000	chr10	104590000 104850000	104595000 104860000	chr10 chr10	104675000 105000000	104680000 105010000	0.00142198 0.00171292	1.3248246	1.347813
chr10		105059896	10000	chr10		104860000	chr10	105000000	105010000	0.00171292	1.5044254	0.85600626
chr11		113451229	10000			113430000		113480000		0.00300015	1.1429281	1.0907023
chr12	123447928	123902361	10000	chr12		123530000	chr12	123710000	123720000	2.77E-08	1.508456	0.630625
chr12	123447928	123902361	10000	chr12		123530000	chr12	123710000	123720000	2.77E-08	1.508456	0.630625
chr13	79855297	80162555	10000			80140000		80930000	80940000	3.23E-05	0.90376765	
chr14	30000405	30190316	5000			30020000		30680000	30685000	1.14E-06	1.363864	0.44168428
chr15 chr15	82827938 84703470	83391537 85392298	10000	chr15		83260000 85135000		83650000 85585000	83660000 85590000	0.00023944 0.07324936	1.5335083 1.363864	1.2896612
chr16	29924422	30117253				29715000		30030000	30035000	0.08700617	1.3652908	1.2216492
chr16	29924422	30117253	10000			30080000		30400000	30410000	0.00022482	1.5745742	1.688994
chr16	67708897	68305708	5000	chr16	67550000	67555000	chr16	67960000	67965000	0	1	1.3505517
chr16	67708897	68305708	5000	chr16		67855000	chr16	67960000	67965000	0.01798354	1.3262548	
chr16	67708897	68305708		chr16		67855000		67960000	67965000	0.01798354	1.3262548	0.5672295
chr16 chr16	67708897 67708897	68305708 68305708	10000 5000			68130000 68295000		68360000 68360000	68370000 68365000	0.00817463 0.02715562	1.5335083	1.634100
chr17	17649172	17967397	10000	chr16 chr17	17810000	17820000	chr16 chr17	17930000	17940000	8.20E-07	1.1149824	0.5261287
chr17	17649172	17967397	10000			17820000	chr17	17930000	17940000	8.20E-07	1.1149824	0.5261287
chr17	17649172	17967397	5000	chr17	17825000	17830000	chr17	17870000	17875000	0.00082981	1.521011	0.96269840
chr17	17649172	17967397	5000	chr17	17825000	17830000	chr17	17870000	17875000	0.00082981	1.521011	0.96269846
chr18	52747689	53804156		chr18		53175000		53205000	53210000	0.00299272	1.6471775	
chr18	52747689	53804156				53175000	chr18	53205000	53210000	0.00299272	1.6471775	1.3091516
chr19	19358332	19657632		chr19		19260000	chr19	19600000	19605000	0 05050400	1.061395	1.3407764
chr2 chr2	57950104 57950104	58484172 58484172	10000		58130000 58130000	58140000 58140000	chr2 chr2	58460000 58460000	58470000 58470000	0.05253198 0.05253198	1.5455453 1.5455453	1.6341001
chr2	57950104	58484172	10000		58350000	58360000	chr2	58670000	58680000	2.43E-08	1.5479501	0.5771854
chr2	198146381	198940251	5000		197975000	197980000		198245000	198250000	9.33E-05	1.3912318	0.1050422
chr2	198146381	198940251	5000	chr2	198055000	198060000	chr2	198175000	198180000	0.06144834	0.9483586	0.7659539
chr2	198146381	198940251			198055000	198060000		198175000	198180000	0.06144834	0.9483586	0.7659539
chr2		201309547		chr2	200835000	200840000		201560000	201565000	2.32E-05	0.110981	0.6510733
chr22 chr22	39840130	40091818	10000	chr22	39800000	39810000	chr22	39890000	39900000	0.00029721	1.1993746	1.4838825
chr22 chr22	42315790 42315790	42689370 42689370	10000			42370000		42480000 42480000	42490000 42490000	0.02962909 0.02962909	1.5869178 1.5869178	
chr3	17221017	17888256	5000		17300000	17305000		17455000	17460000	0.00032593	1.3415203	0.19510272
chr3	17221017	17888256		chr3	17300000	17305000	chr3	17455000	17460000	0.00032593	1.3415203	0.19510272
chr3	36843149	36945794	5000	chr3	36770000	36775000	chr3	36895000	36900000	0.00071711	1.3308439	1.3718083
chr3	52965713	53175017	10000	chr3	52880000	52890000	chr3	53090000	53100000	8.95E-05	0.84550905	0.504883
chr3	135807609	136615268	5000	chr3	135785000	135790000	chr3	136470000	136475000	0.00010323	0.27722827	0.44168428
chr3 chr3	135807609 180571624	136615268 181207851	5000 5000		135790000 180510000	135795000 180515000	chr3	136475000 180890000	136480000 180895000	0.00967255	1.312808	
chr3	180571624	404007054	5000	chr3 chr3	180510000		chr3 chr3	101105000	180895000	0.00659433	1.1958251	1.351187
chr4	170357792				169930000				170490000	2.37E-09	1.3162088	
chr5	87676693		10000			87080000	chr5	87840000		3.70E-05	0.22795595	
chr5	87676693	88195380	10000	chr5	87680000	87690000	chr5		87790000	0.00987681	1.4089538	0.95696616
chr5	87676693		10000						87790000	0.00987681	1.4089538	
chr5	137838122		5000		137910000				138605000	0.00010323	0.5248362	0.1145801
chr6 chr6	24988105 24988105		5000		26610000 26610000				27055000	0.09176145	1.1728417 1.1728417	
chr6 chr6	24988105	33842877	5000 10000		26610000 27140000	26615000		27050000	27055000 27440000	0.09176145 9.69E-05	1.1728417 1.5393742	
chr6	24988105		10000		27140000	27150000		27430000		9.69E-05 9.69E-05	1.5393742	
chr6	24988105		5000		28235000	28240000		28300000	28305000	0.04845583	1.2806816	
chr6	24988105	33842877	5000		28235000	28240000		28300000	28305000	0.04845583	1.2806816	
	24988105	33842877	5000	chr6	30710000	30715000	chr6	30845000	30850000	3.10E-05	0.768764	0.2124772
chr6		00040077	5000	chr6	30710000	30715000	chr6	30845000	30850000	3.10E-05	0.768764	0.21247724
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Frad. 52786771 5110000 52180000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 55100000 5510000 5510000 5510000 5510000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5550000 5570000 </td <td>chr3</td> <td>17221017</td> <td>17888256</td> <td>5000</td> <td>chr3</td> <td>17470000</td> <td>17475000</td> <td>chr3</td> <td>17875000</td> <td>17880000</td> <td>0.34376985</td> <td>1.0329467</td> <td>1.63E-05</td>	chr3	17221017	17888256	5000	chr3	17470000	17475000	chr3	17875000	17880000	0.34376985	1.0329467	1.63E-05
134 ESYT22668 #6003985 10000chrd 2280000 2380000 2380000 2380000 1283472 12423472 1348077069 35867000 35867000 35867000 35867000 35867000 358775000 3587700 3587700	chr3	17221017	17888256	5000	chr3	17470000	17475000	chr3	17875000	17880000	0.34376985	1.0329467	1.63E-05
Hrd 15560760136615268 5000Errd 3552000Errd 1556600015370000 0.31253472 1.3427322 Hrd 15560760136615268 10000Errd 3550000175370000 1.5790071 1.3394774 Hrd 15560760136615268 10000Errd 13560001756710000 1.5790071 1.3394774 Hrd 15560760136615268 5000Errd 1362600013527000Errd 13647000013475000 1.2760337 1.2669171 Hrd 13560760136615268 5000Errd 1362600013527000Errd 136470000134750000 1.2760337 1.2669171 Hrd 13560760136615268 5000Errd 13627000Errd 1364700013475000 1.2760337 1.2669171 Hrd 136571524611277651 10000Errd 1307700018701000Errd 1803900011399000 1.4305107 1.573824 Hrd 1037278217064003 10000Errd 17250000170900Errd 18039000012399000 2.444782 1.445334 Hrd 1738777217064003 10000Errd 17250000170900Errd 1790000077400000 2.444782 1.445334 Hrd 1747737727017064003 10000Errd	chr3	52965713	53175017	10000	chr3	52880000	52890000	chr3	53100000	53110000	0.31834856	1.5495831	4.66E-05
Prid 155607601 56615268 5000/Prid 1556200000 155620000 1573077 13394774 Prid 155607601 36615268 10000/Prid 135600000 1570000 15730007 13394774 Prid 155607601 36615268 10000/Prid 13610000 137407000 1573007 13394774 Prid 155607601 36615268 5000/Prid 136265001 35270000 137407000 14750337 1.266171 Prid 35607601 36617268 5000/Prid 136256001 35270000 17310307 1.726137 1.726117 Prid 35607601 5600/Prid 180750001 1500000 1823000 1.828037 1.728117 Prid 15007162 110000 11000000 110000000 1.22000 11330000 1.222007 1133000 1.22817 1.728117 Prid 1437727 17646003 100000000 1410000000 1.222000 11330000 1.23337 1.564112 1.5741413 1.443388 1.5744133 1.443338<	chr3	63792668	64003983	10000	chr3	62600000	62610000	chr3	63800000	63810000	1.2053969	0.6132447	0.00451189
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Brd3 1386076091368115208 5000ptrd3 13826500138270001ptrd3 1384700001 34847200 1.2789417 Brd3 160571624181207851 5000ptrd3 180750000180710000trd3 1803900001 80898000 0.384727 1.1728417 Brd3 160571624181207851 10000ptrd3 1813200001 14300001 4.335197 1.5741624 Brd4 103001441 123080001 131300000 1.3314486 1.5741638 Brd4 103057782170646003 10000chrd 1437600001 4.37600000 4.37600000 4.3760000 4.376000	chr3	135807609	136615268	10000	chr3	136110000	136120000	chr3	136720000			1.5335083	0.06340116
High 190571624[181207851] 5000[Hrd] 180750000[Hrd] 180890000[18089000] 0.884727 1.1728417 Hird 100571624[181207851] 10000[Hrd] 181700000[Hrd] 181430000[18140000] 1.5276841 Hird 100571624[181207851] 10000[Hrd] 181430000[Hrd] 1532000[18130000] 1.5226448 1.5741838 Hird 10330164[Hr31398082] 10000[Hrd] 10280000[17019000] C.1242752 1.666538 Hird 103357792[70044003 10000[Hrd] 17030000[Hr3180000] 0.51119745 A433300 Hird 170357792[70044003 10000[Hrd] 17030000[Hr5180000] 4950000 49510000 7.400383 196.37476 Hird 49441777 49844022 10000[Hr5 453000[Hr5 4950000 4950000 4950000 4950000 4950000 14038358 160.43748 Hird 87676693 8195380 5000[Hr5 87240000 87440000 87440000 87440000 87440000 8744000 8744000 8744000 8744000 87440000 514443 1.266715 <td>chr3</td> <td>135807609</td> <td>136615268</td> <td></td> <td>chr3</td> <td>136265000</td> <td>136270000</td> <td>chr3</td> <td></td> <td>136475000</td> <td></td> <td></td> <td>7.88E-06</td>	chr3	135807609	136615268		chr3	136265000	136270000	chr3		136475000			7.88E-06
hrd 180571624[181207851] 15000[hrd 180780000[hrd] 1804900001 1804900001 180490001 14300001 143010001 143010001 15378524 hrd 180571624[181207851] 110000[hrd] 1813200001[ht/31800001 153800001 15384865 15741838 hrd 130377782170644003 10000[hrd] 1703000[hrd] 170400000 170400000 12384850 1.4433388 hrd 170357782170644003 10000[hrd] 1703000[hrd] 170400000 170400000 12484308 hrd 49441773 49844022 10000[hrd] 46330000[hrd] 4590000 4500000 4700000 170480000 17048000 14032838 hrd 947776693 88195380 5000[hrd] 68770000 6786000 7786000 87786000 17042828 hrd 947776693 88195380 5000[hrd] 68770000 67740000 87780000 7786000 87780000 7788000 7788000 7788000 7788000 7788000 7788000 7788000 77880000 1333018 134670													7.88E-06
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chr6 24988105 33842877 5000chr6 30450000 30455000chr6 30565000 30570000 0.61549026 1.2254188													0.00023257
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chr6 24988105 33842877 5000chr6 31510000 s155000chr6 31630000 31635000 1.2857124 0.76937705													0.00682281
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chr6 24988105 33842877 5000chr6 31760000 31765000chr6 31800000 0.9526036 1.4984149													0.00296772
chr6 24988105 33842877 5000chr6 31760000 31765000chr6 31800000 31805000 0.9526036 1.4984149	chr6	24988105	33842877	5000	chr6	31760000	31765000	chr6	31800000	31805000	0.9526036	1.4984149	0.00296772

chr6	24988105	33842877	10000	chr6	32080000	32090000	chr6	32140000	32150000	0.44272268	1.6237167	4.96E-05
chr6	24988105	33842877	10000	chr6	32080000	32090000	chr6	32140000	32150000	0.44272268	1.6237167	4.96E-05
chr6	24988105	33842877	5000	chr6	32380000	32385000	chr6	32665000	32670000	0.38935196	0.5867754	4.44E-10
chr6	24988105	33842877	10000	chr6	32380000	32390000	chr6	32830000	32840000	0.8012636	1.1520559	1.76E-05
chr6	24988105	33842877	10000	chr6	32380000	32390000	chr6	32830000	32840000	0.8012636	1.1520559	1.76E-05
chr6	24988105	33842877	5000	chr6	32380000	32385000	chr6	32665000	32670000	0.38935196	0.5867754	4.44E-10
chr6	24988105	33842877	10000	chr6	32380000	32390000	chr6	32830000	32840000	0.8012636	1.1520559	1.76E-05
chr6	24988105	33842877	10000	chr6	32380000	32390000	chr6	32830000	32840000	0.8012636	1.1520559	1.76E-05
chr6	24988105	33842877	5000	chr6	32850000	32855000	chr6	32940000	32945000	0.4254983	1.3397491	0.01301809
chr6	24988105	33842877	5000	chr6	32850000	32855000	chr6	32925000	32930000	0.58261454	1.3912318	5.14E-07
chr6	24988105	33842877	5000	chr6	32850000	32855000	chr6	32925000	32930000	0.58261454	1.3912318	5.14E-07
chr6	24988105	33842877	5000	chr6	32850000	32855000	chr6	32940000	32945000	0.4254983	1.3397491	0.01301809
chr6	24988105	33842877	5000	chr6	33550000	33555000	chr6	33675000	33680000	0.21398759	0.18502843	1.39E-07
chr6	24988105	33842877	5000	chr6	33550000	33555000	chr6	33675000	33680000	0.21398759	0.18502843	1.39E-07
chr6	24988105	33842877	5000	chr6	33735000	33740000	chr6	33995000	34000000	0.16174258	1.214352	0.00071491
chr6	24988105	33842877	5000	chr6	33735000	33740000	chr6	34120000	34125000	1.2104913	0.17672323	6.03E-05
chr6	24988105	33842877	5000	chr6	33735000	33740000	chr6	34120000	34125000	1.2104913	0.17672323	6.03E-05
chr6	84264202	84409255	10000	chr6	84320000	84330000	chr6	84930000	84940000	0.21959442	0.31036413	0.0002823
chr7	86403263	86948073	5000	chr7	86565000	86570000	chr7	86780000	86785000	0.32161906	1.0722134	0.00021275
chr7	86403263	86948073	5000	chr7	86565000	86570000	chr7	86780000	86785000	0.32161906	1.0722134	0.00021275
chr7	86403263	86948073	10000	chr7	86690000	86700000	chr7	87580000	87590000	74.00363	1.4095246	0.04428426
chr8	18396405	18429406	9406	chr8	18050000	18060000	chr8	18420000	18430000	1.4860443	1.1758273	1.23E-07
chr8	26190836	26279173	5000	chr8	26230000	26235000	chr8	26510000	26515000	0.34376985	1.2175577	0.00071491
chr8	27327841	27453762	3762	chr8	27170000	27175000	chr8	27450000	27455000	0.7089102	1.3237675	5.86E-05
chr8	27327841	27453762	3762	chr8	27170000	27175000	chr8	27450000	27455000	0.7089102	1.3237675	5.86E-05
chr8	38014429	38310910	10000	chr8	37630000	37640000	chr8	38020000	38030000	1.3000284	0.2903848	4.66E-05
chr8	38014429	38310910	5000	chr8	37975000	37980000		38030000	38035000	1.2364298	1.3860595	3.32E-06
chr8	38014429	38310910	5000	chr8	37975000	37980000	chr8	38030000	38035000	1.2364298	1.3860595	3.32E-06
chr8	38014429	38310910	5000	chr8	38035000	38040000		38575000	38580000	0.34376985	1.0722134	0.00021275
chr8	60475926	60954059	5000	chr8	59630000	59635000	chr8	60835000	60840000	0.10347593	1.2623683	2.74E-05
chr8	60475926	60954059	5000	chr8	59720000	59725000	chr8	60830000	60835000	0.8606538	0.8023862	9.22E-05
chr8	89221915	89462854	10000		87710000	87720000		89330000	89340000	1.3222934	0.45391688	3.55E-07
chr9	101065115	101076627	1627	chr9	101075000	101080000	chr9	101535000	101540000	0.17700288	1.2175577	5.86E-05

Table 3.9 | Loops anchored in schizophrenia (SZ) risk sequence.

Cell-type-specific HiCCUPS loops (i.e., FDR < 0.1 in one cell type and FDR > 0.1 in the remaining two) overlapping SZ risk-associated GWAS loci in at least one anchor. Riskchr, Risk start, Risk end = coordinates of SZ risk locus overlapping loop anchor; Risk overlap = # bp overlap between loop anchor and risk locus; chr1, x1, x2 = anchor 1 coordinates; chr2, y1, y2 = anchor 2 coordinates; Glia/Neu/NPC-fdrDonut = HiCCUPS FDR value from the "donut" local neighborhood.

		GWAS						1			1	1				Develop	
		Locus	GWAS	eSNP					target.bin.							mental	Primary/C
eSNP	Chr	Start	Locus End	position	Gene	ROI	.coord	in.genes	coord	genes	Glia	NPC	Neuron	Tissue	Туре	Period	onditional
						chr1:23724	chr1.23800		chr1:16700	SLC35E2B							
rs1203						02-	01-		01-	SLC35E2						early mid-	
7821	1	2372401	2402501	2387715	SLC35E2		2390000		1680000	0-4 -	NA	NA	NA	-	-	prenatal	conditional
rs1380						chr1:84111 85-	chr1:84600 01-		chr1:88800 01-								
50288	1	8355697	8638984	8460247	RERE	8638984	8470000	RERE I -	8890000	RERE 0 -	1.57E+00	2.25E+00	1.78E+00	-	-	-	primary
rs2015						chr1:30412 552-	chr1:30420 001-		chr1:29560 001-	PTPRU 0- 1 +,MECR	1.679003	1 130746	1.760513			early mid-	
244	1	30412551	30443951	30428943		30437271	30430000		29570000	0 -	1.07 3003	22	61	_	neurons	prenatal	primary
						chr2:19814			chr2:19829								
rs1262 1129	2	198148577	198835577	198265350	SE3B1	8578- 198835577	0001- 198270000	SF3B1 1 3-26 -	0001- 198300000	SF3B1 0- 1I-	6 28E+00	7.76E+00	1 82E+00	_	_	L	primary
		100110011	100000011	100200000	FTCDNL			0 20			0.202100		1.022100				printidity
					1,	chr2:20071	chr2:20078	00	chr2:20071	ETODNI AL		0 000400					
rs3522 0450	2	200715237	201247789	200780737	AC07304		0001- 200790000	C2orf69 2		FTCDNL1 0-3 -	5.62E-01	0.038190 44	0.15	_	_	adult	primary
					LINC017					LOC10050							
rs1865					92, AC00716	chr2:20071 5238-	chr2:20077 0001-	C2orf69 0	chr2:20160	7140 0 - ,AOX2P 7-				putamen (basal			
46506	2	200715237	201247789	200777930		200848037	200780000	-1 +	201610000	11 +	1.33E+00	2.21E-01	4.18E-01		_	adult	conditional
						chr3:36843			chr3:36780	D 01 1/010							
rs9834 970	3	36843183	36945783	36856030	DCI K3	184- 36945783	001- 36860000		001- 36790000	DCLK3 0- 2 -	1.572272 52	5.13E-01	0.960857	nerve - tibial	neurons	infant	primary
0.0	0	00010100	00010100	0000000	DOLINO	chr3:52965			chr3:52270					libitai	nourono	man	printidity
rs6801	~	50004070	52520200	52000505	DDMAM	713-	001-	SFMBT1		PPM1M 0-		0.285430				late	
235	3	52281078	53539269	53009595		53175017 chr3:63792	53010000 chr3:63800	<u>~ </u> -	52280000 chr3:63850	ATXN7I0-	68	16	09	-	neurons	prenatal	conditional
rs1133						651-	001-	C3orf49 0	001-	2 +,THOC7							
86200	3	63792650	64004050	63832212	I HOC7	64004050 chr3:13580	63810000 cbr3:13615	-2 +	63860000 chr3:13596	0 -	7.46E-03	7.35E-02	2.19E-01	F	F	F	primary
rs1093						7406-	0001-	STAG1 2		PCCB 0-	0.038329	0.101138	0.015655				
5184	3	135807405	136615405	136153468	PCCB	136615405	136160000	0 -	135970000	1 +	76	13	63	_	-	-	primary
						chr4:17035 7553-	chr4:17064 0001-	CLCN3 1	chr4:17054 0001-	CLCN3 0-			2.016706				
rs7438	4	170357552	170646052	170642246	CLCN3	170646052	170650000	6 +	170550000	1 +	4.61E+00	1.10E+01	55	_	_	-	primary
						chr5:45291			chr5:44740		0.050040		0 740404				
rs9292 918	5	45291475	46404116	45301035		476- 45393775	001- 45310000	HCN1 6 -	001- 44750000	(BRCAT54)	0.659013	2.21E-01	0.712464	_	_	adult	primary
						chr6:84279	chr6:84280		chr6:84420	(=				cerebellar			
rs2016 358	6	83779798	84407274	84283733		923- 84407274	001- 84290000	SNAP91 32-33 -	001- 84430000	SNAP91 0 -	0.083956	0.058077 64	0.550498 17	hemisphe			primary
550	0	03113130	04407274	04203733	SINALSI	04407274	04230000	52-55	04430000	PPP2R2A	30	04	17		_	F	primary
						chr8:26190			chr8:26230								
rs1705 5186	8	26181524	26279124	26260910		836- 26279173	001- 26270000	BNIP3L 6 -8l+	001- 26240000	L 0 +, SDAD1P1	0.207700		3.28E-01	testis	_	adult	conditional
0100	0	20101024	20213124	20200310		chr8:38014			chr8:38240			12	0.20E 01	103113		addit	contaitionai
rs2019	8	20020424	20240024	20204044	WHSC1L	429-	001-	FGFR1 5		3 +,WHSC1		0.005.04	0.221717			early	
99919	0	38020424	38310924	38291844		38310910	38300000	-	38250000	L1 0 - MIR4688 0-	73	6.29E-01	08	-	-	prenatal	primary
						chr11:4634			chr11:4639	1 +,MDK 0							
rs1693 8506	11	46340213	46751213	46454313		2944- 46751213	0001- 46460000	AMBRA1 17-18 -	0001- 46400000	+,DGKZ 8,1 2-34 +	9.32E-02	0.000108 69	0.094945			early mid- prenatal	primary
0000		40040210	40701210	40404010	MBR		chr12:5748				0.02L 02					prenatai	printiary
4550	10	57400044	57407044	57 4000 40	07470	8315-	0001-	7 +,STAT		1 +,STAT6	4.445.00	0.075.00	0.745.00			adolesce	
rs4559	12	57428314	57497814	57489648	STATE	57682971 chr15:7880	57490000 chr15:7890	6 25 -	57530000 chr15:7872	0-1 -	4.14E-02	3.87E-02	2.74E-02	-	microglia	nı	conditional
rs7171						3033-	0001-	CHRNA3	0001-			1.042098				early	
869	15	78803032	78926732	78900909	IREB2 LOC1019	78926732	78910000	4 -	78730000	IREB2 0 +	52	59	13	-	-	prenatal	conditional
					29479,	chr15:8466	chr15:8493		chr15:8574								
rs3567			85153461	0.4000000	RP11-	1162-	0001-	LOC6424		LOC64242						early mid-	
7834	15	84661161	85153461	84933868	56105.3	85153461 chr15:9141	84940000 chr15:9142	23 I - FES 0-	85750000 chr15:9140	3 0-1 -	NA	NA	NA	ovary		prenatal	primary
						6561-	0001-	4 +,FURI	0001-						endotheli	adolesce	
rs4702	15	91416560	91436560	91426560	FURIN	91429040	91430000	N 5-16 + SLX1B-	91410000	FURIN 0 +	3.41E-02	3.43E-01	2.53E-01	-	al cells	nt	primary
								SULT1A4		SLX1B-							
								III+,TME		SULT1A4 I							
								M219 2- 6 +,SMG		+, TMEM21 9 0 +,KCTD							
								1P2 -		13 0 -							
								,SLX1A I +,BOLA2		,SMG1P2 I							
								ų-		- ,SLX1A I +,							
								LOC606		BOLA2 I -							
rs4788					TMEM21	chr16:2992 4378-	chr16:2997 0001-	724 I +,L OC61303	chr16:2994 0001-	,LOC60672 4 I +,LOC6							
203	16	29924377	30144877	29978827	9	30144877	29980000	8 I -	29950000	13038 I -	3.52E+00	5.98E+00	4.65E+00	-	-	_	primary
								SLX1B- SULT1A4		SLX1B- SULT1A4 I							
								I +,INO8		+,INO80E 0							
								0E 4-		-							
								11 +,SM G1P2 I -		3 +,SMG1P 2 I -							
								SLX1A I		.SLX1AIII+.							
								+,BOLA2		BOLA2 I - LOC60672							
						chr16:2992			chr16:3000	4 I +,LOC6							
rs3935		2002 1277	204 4 40	20010500		4378-	0001-	724 I +,L	0001-	13038 -		0.073098					
873	16	29924377	30144877	30018500	INO80E	30144877	30020000	UC61303	30010000	HIRIP30-	01	34	01	-	neurons	-	primary

								8 I - ,DOC2A 11-17 -		9 - ,TAOK2 22- 24 +							
								,HIRIP3 0 - SLX1B-									
								SLX1B- SULT1A4 I +,INO8 0E 4- 11 +,SM G1P2 I - ,SLX1A I +,BOLA2 I - ,LOC606 724 I +,L OC61303		SLX1B- SULT1A4 I +,INO80E 0 - 3 +,SMG1P 2 I - ,SLX1A I +, BOLA2 I - LOC60672 4 I +,LOC6							
rs4787 491	16	29924377	30144877	30015337	DOC2A	4378- 30144877	chr16:3001 0001- 30020000	8 I - ,DOC2A 11-17 - ,HIRIP3 0	chr16:3000 0001- 30010000	13038 - ,HIRIP3 0- 9 - ,TAOK2 22- 24 +	0.141626	0.073098 34		brain - cortex		adolesce nt	conditional
rs1164 7976	16	58669293	58691393	58680936		9294-	chr16:5868 0001- 58690000		chr16:5866 0001- 58670000	CNOT1 0- 1 -	8.66E-01	9.55E-01	6.07E-01	-	_	_	primary
rs4072 739	17	17722402	18030202	17884660	DRG2		17890000	DRC3 4-		DRG2 0 +	0.376044	0.196744 81	0.376815 56		_	_	primary
rs7298 6630	19	11839736	11859736	11849736	ZNF823	9736- 11849736	11850000	ZNF823 0-1 -	11860000	ZNF823 0 -	0.007383 82	0.046477 34	7.22E-08		endotheli al cells		primary
rs2965 199	19	19374022	19658022		GATAD2 A	4023- 19658022	0001- 19480000		19500000	GATAD2A 0-1 +	2.81E+00	5.43E+00	1.442334 63		_	_	primary
rs5023 763	19	50067499	50135399		SNRNP7	7500-	0001-			SNRNP70 0-2 +		9.12E-01	5.46E-01	_		_	primary
rs2004 47424	22	41408556			RANGAP	chr22:4140 8557-	chr22:4158 0001- 41590000		chr22:4168 0001-		7.81E-01	0.392544			_	_	primary

Table 3.10 | Chromosomal contacts anchored in SZ GWAS co-localizedeQTLs.

HiC interactions overlapping GWAS co-localized eQTL SNP-gene pairs (Dobbyn *et al.*, 2018). Columns A-E are from Dobbyn et al (2018) Table 2. eSNP, eQTL SNP; Chr, GWAS Locus Start and GWAS Locus End together indicate the coordinates for the corresponding risk locus within which the eSNP resides; Gene, the gene corresponding to the eSNP; eSNP position, coordinate of the eSNP in hg19; ROI, region of interest corresponding to the SZ risk loci; anchor.bin.coord, coordinate of the 10kb used as anchor in the binomial test for significant interactions; anchor.bin.genes, genes overlapping the 10kb anchor bin; target.bin.coord, coordinate of the 10kb queried as a target of the anchor in the binomial test for significant interactions; target.bin.genes, genes overlapping the 10kb target bin; Glia, NPC, Neuron columns refer to the log (q-value) for each interaction determined by binomial statistics.

						Genotype 0	(n=150)	Genotype 1	(n=277)	Genotype 2	(n=152)
Ensembl ID	Gene ID	Gene Start (bp)	Gene End (bp)	p-value	p-adjusted			Mean	SE		SE
ENSG00000250120	PCDHA10	140235595			1.61E-12	-0.9450845	0.14005154	-0.3220529	0.0469093	-0.018637	0.03260897
ENSG00000204962		140220907	140391929						0.03233461		0.04282544
ENSG00000204963	PCDHA7	140213969	140391929	2.03E-11	1.36E-09	-1.2849008	0.04151695	-1.0832615	0.02988216	-0.9140139	0.04085353
ENSG00000204969	PCDHA2	140174444	140391929	1.87E-09	1.25E-07	-1.263745	0.03214275	-1.1634137	0.02443782	-0.9951632	0.0306389
ENSG00000239389	PCDHA13	140261793	140391929	2.65E-08	1.78E-06	-0.3379225	0.03420221	-0.4584249	0.02614051	-0.5841679	0.03465996
ENSG00000204967	PCDHA4	140186659	140391929	1.28E-05	0.0008576	-0.0940208	0.03133687	0.07152288	0.02254759	0.08552427	0.03129946
ENSG00000204961	PCDHA9	140227048	140391929	0.0003829	0.0256543	-0.8885906	0.13530321	-0.531843	0.04152144	-0.4422395	0.04931668
ENSG00000249158	PCDHA11	140248689	140391929	0.0008857	0.0593419	0.4383577	0.03023422	0.5256985	0.02074671	0.5673561	0.02727161
ENSG00000204970	PCDHA1	140165876	140391929	0.2997	1	-1.168368	0.03560745	-1.212149	0.02525289	-1.238437	0.03251574
ENSG00000255408	PCDHA3	140180783	140391929	0.09267	1	-0.3266051	0.02698158	-0.2652614	0.02019158	-0.2768451	0.03101834
ENSG00000204965	PCDHA5	140201222	140391929	0.3493	1	0.01375301	0.03100332	0.06426113	0.02347947	0.044756	0.03029636
ENSG0000081842	PCDHA6	140207563	140391929	0.4351	1	-0.0261231	0.0399647	-0.0368595	0.02697582	-0.0722259	0.03390551
ENSG00000249034	AC005609.1	140240341	140243224	0.8416	1	-1.651762	0.06461126	-1.734715	0.0462817	-1.657964	0.06411573
ENSG00000251664	PCDHA12	140255058	140391929	0.01968	1	-0.5597011	0.03440314	-0.4958071	0.02650548	-0.4345344	0.03334305
ENSG00000248383	PCDHAC1	140306302	140391929	0.04019	1	0.1123253	0.04139802	0.1829496	0.02673045	0.2158291	0.03283328
ENSG00000243232	PCDHAC2	140345820	140391936	0.589	1	2.361629	0.03826713	2.376794	0.03004392	2.326327	0.03968978
ENSG00000112852	PCDHB2	140474227	140476962	0.3131	1	0.6907204	0.03089572	0.6421203	0.02787192	0.6447874	0.0344836
ENSG00000272154	AC005754.7	140479829	140481794	0.881	1	-0.0156119	0.04615792	0.05565437	0.03715139	-0.0173929	0.06464357
ENSG00000113205	PCDHB3	140480234	140483406	0.02615	1	0.4022594	0.03103061	0.3829122	0.0218286	0.3135084	0.02927212
ENSG00000272108	AC005754.8	140498262	140500347	0.04383	1	0.6543306	0.04894567	0.5441159	0.03794151	0.5393526	0.05292806
ENSG0000081818	PCDHB4	140501581	140505201	0.8963	1	1.841228	0.02971057	1.815392	0.02339597	1.810416	0.03254467
ENSG00000113209	PCDHB5	140514800	140517703	0.224	1	1.567102	0.02299553	1.549667	0.02068541	1.51964	0.02580371
ENSG00000113211	PCDHB6	140529683	140532868	0.3184	1	0.265684	0.03583055	0.2689495	0.02605475	0.3133934	0.03659252
ENSG00000255622	PCDHB17	140535577	140538639	0.7226	1	-0.3756098	0.04920646	-0.4890143	0.03970313	-0.4072474	0.05548462
ENSG00000113212	PCDHB7	140552243	140555957	0.3499	1	0.4885163	0.03826745	0.4943283	0.03128195	0.5439796	0.04520774
ENSG00000120322	PCDHB8	140557371	140560081	0.9269	1	0.01001523	0.04438598	-0.0132715	0.03983904	0.02598359	0.05385032
ENSG00000196963	PCDHB16	140560980	140565793	0.4212		0.02597449	0.04194141	-0.0275194	0.03339609	0.09018198	0.04660624
ENSG00000177839	PCDHB9	140566893	140571111	0.03744	1	0.674292	0.0381622	0.7036802	0.02482808	0.7873397	0.03631906
ENSG00000120324	PCDHB10	140571942	140575215	0.0332	1	1.550995	0.02669294	1.558507	0.01814735	1.626481	0.02711403
ENSG00000197479	PCDHB11	140579183	140582618	0.7363	1	1.24371	0.02766519	1.198184	0.02279798	1.224654	0.02874177
ENSG00000120328	PCDHB12	140588269	140591696	0.5195	1	1.578292	0.02579869	1.568764	0.01984231	1.613458	0.02583847
ENSG00000187372	PCDHB13	140593509	140596993	0.5773	1	1.144594	0.03861705	1.117381	0.02907133	1.186299	0.04333161

ENSG00000120327P0	CDHB14	140602931	140605858	0.6245	1	2.37246	0.01787361	2.345718	0.01396584	2.359431	0.01816899
ENSG00000146001P0	CDHB18	140613938	140617101	0.7843	1	0.5287241	0.04483872	0.4868465	0.03350162	0.556907	0.04718459
ENSG00000262096P0	CDHB19P	140619518	140621864	0.6678	1	0.5610128	0.05018575	0.5222347	0.036843	0.6020646	0.04864062
ENSG00000113248P0	CDHB15	140625147	140627799	0.2016	1	1.275723	0.02478772	1.259874	0.02024715	1.326387	0.0259284
ENSG00000178913T/	AF7	140698057	140700330	0.6472	1	5.287593	0.02872701	5.307101	0.02267996	5.242052	0.03666379
ENSG00000272070A0	C005618.6	140705777	140708924	0.3824	1	0.2658314	0.02889967	0.2744418	0.02361841	0.2928478	0.03017335
ENSG00000204956P0	CDHGA1	140710252	140892546	0.08634	1	-0.9367046	0.04188522	-0.8589053	0.0321514	-0.8368188	0.03893686
ENSG0000081853P0	CDHGA2	140718539	140892546	0.3214	1	-0.4044892	0.04268331	-0.4066491	0.03242702	-0.3469972	0.03727468
ENSG00000254245P0		140723601	140892546	0.699	1	0.10353173	0.04510637	0.08295321	0.03484245	0.15444447	0.0494062
ENSG0000254221P0		140729828	140892546		1		0.03450643		0.02540657		
ENSG0000262576P0		140734768	140892546	0.6408	1		0.03192729		0.02606557		
ENSG00000253910P0		140739703	140892546	0.01694		0.3736417	0.037329		0.03197805		0.04426136
ENSG00000253485P0		140743898	140892546	0.6391	1		0.03938976		0.03426557		
ENSG00000262209P0		140749831	140892546	0.1391			0.04431799		0.03587637		
ENSG00000253731P0		140753651	140892546	0.6019	1	-0.3094883			0.03370213		
ENSG00000253537P0		140762467	140892546	0.485	1	-0.4691286					
ENSG00000253953P0	CDHGB4	140767452	140892546	0.2756	1	-0.4760418	0.03919649	-0.4802712	0.02919548	-0.412954	0.04021008
ENSG00000253767P0	CDHGA8	140772381	140892546	0.363	1	-0.9928499	0.05395769	-0.9891268	0.0443002	-0.8983144	0.06304307
ENSG00000261934P0	CDHGA9	140782520	140892546	0.419	1	0.7850766	0.03107475	0.7785777	0.0252662	0.8391602	0.03686301
ENSG00000253305P0	CDHGB6	140787770	140892546	0.438	1	0.08134237	0.04342428	0.09391992	0.03328246	0.14412456	0.04685704
ENSG00000253846P0	CDHGA10	140792743	140892546	0.9656	1	0.7094627	0.03317664	0.7157928	0.02714398	0.7139836	0.03741461
ENSG00000254122P0	CDHGB7	140797427	140892546	0.2273	1	-0.0062796	0.04391279	0.0008358	0.03474804	0.10182631	0.05136511
ENSG00000253873P0	CDHGA11	140800762	140891835	0.7571	1	0.1503583	0.03974685	0.1630275	0.03141137	0.1841329	0.04277974
ENSG00000248449P0	CDHGB8P	140805853	140808219	0.4083	1	0.1226163	0.05903111	0.0846888	0.0463076	0.2108079	0.06345998
ENSG00000253159P0	CDHGA12	140810185	140892546	0.5601	1	0.4870029	0.04397322	0.4898922	0.03426719	0.521935	0.04629193
ENSG00000240184P0	CDHGC3	140855580	140892542	0.246	1	3.335496	0.04493861	3.390439	0.03367271	3.400317	0.04292565
ENSG00000242419P0	CDHGC4	140864741	140892546	0.6337	1	1.511794	0.04101957	1.537931	0.02512287	1.522494	0.03315068
ENSG00000240764P0	CDHGC5	140868808	140892546	0.8556	1	2.872448	0.04664224	2.888523	0.03236965	2.84832	0.04243448
ENSG00000131504DI	IAPH1	140894583	140998622	0.8943	1	2.475123	0.02367114	2.446633	0.01934177	2.486694	0.02561167
ENSG00000248106A0	C005609.2	140143695	140144406	NA	NA	NA	NA	NA	NA	NA	NA
ENSG00000249504P0	CDHA14	140240860	140243104	NA	NA	NA	NA	NA	NA	NA	NA
ENSG00000171815P0	CDHB1	140430979	140433512	NA	NA	NA	NA	NA	NA	NA	NA
ENSG00000120329SL	LC25A2	140682196	140683612	NA	NA	NA	NA	NA	NA	NA	NA
ENSG00000255729A0	C005618.1	140699661	140700339	NA	NA	NA	NA	NA	NA	NA	NA
ENSG0000242020Rf		140858883	140859190			NA			NA	NA	NA

 Table 3.11 | Gene-level single-SNP eQTLs for clustered PCDH.

Gene-level single-SNP eQTL analysis testing for association of clustered PCDH 240 gene expression with SZ risk SNP rs111896713. Significant genes are highlighted in green.

bp	credsnp	indexsnp	R_to_index	hanc CP	hanc GZ	hanc ES	hgnc_IMR90	NPC	Neuron	Glia
	rs10860949	rs10860964	0.976583	0	0	J	0	ASCL1, C12orf42	NA	NA
	rs7306170	rs10860964	0.910154					ASCL1, C12orf42		NA
103575583	rs10778221	rs10860964	0.973776	C12orf42	NA	NA	NA	ASCL1, C12orf42	NA	NA
	rs10860950	rs10860964	0.936245		NA	NA	NA	ASCL1, C12orf42	NA	NA
			0.930243	EFNB1				EFNB1		EFNB1
	rs5937157	rs5937157	0 912256		NA	NA	NA		NA	
	rs62606711	rs5937157	0.012200	EFNB1	NA	NA	NA	EFNB1	NA	EFNB1
68377205	rs62606712	rs5937157	0.90829		NA	NA	NA	EFNB1	NA	EFNB1
	rs2361468	rs5937157	0.993235		NA	NA	NA	EFNB1	NA	EFNB1
68377499	rs2885287	rs5937157	0.993235	EFNB1	NA	NA	NA	EFNB1	NA	EFNB1
68379039	rs5937159	rs5937157	0.92888	EFNB1	NA	NA	NA	EFNB1	NA	EFNB1
				CTNNA1,L RRTM2,SIL 1,MATR3,P	SIL1,MATR 3,PAIP2,SL	CTNNA1,SI		CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24,	CTNNA1, LRRTM2, SIL1, SNHG4, MATR3,	CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24,
137913882		rs3849046 rs3849046	0.887143	CTNNA1,L RRTM2,SIL 1,MATR3,P	C23A1 SIL1,MATR 3,PAIP2,SL C23A1	L1,MATR3 CTNNA1,SI L1,MATR3	1 CTNNA1,SIL 1	PROB1, DNAJC18 CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24, PROB1, DNAJC18	SNORA74A, PAIP2 CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2	DNAJC18 CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24, DNAJC18
	rs11957778	rs3849046	0.880865	CTNNA1,L RRTM2,SIL 1,MATR3,P			CTNNA1,SIL 1	CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24, PROB1, DNAJC18		CTNNA1, LRRTM2, SIL1, SNHG4, MATR3, SNORA74A, PAIP2, SLC23A1, SPATA24, DNAJC18
				CCDC39,S	CCDC39,S					
	rs10804885	rs33972009	0.769784	OX2	OX2	NA	NA	SOX2, SOX-OT	SOX2, SOX-OT	SOX2, SOX-OT, FLJ46066
113364647	rs4245150	rs2514218	0.952883	DRD2	DRD2	NA	NA	TTC12	TTC12	TTC12
	rs17602038	rs2514218	0.952883	DRD2	DRD2	NA	NA	TTC12	TTC12	TTC12
113364803		rs2514218	0.952883		DRD2		NA	TTC12	TTC12	TTC12
113365084		rs2514218	0.952883	DRD2	DRD2	NA	NA	TTC12	TTC12	TTC12
113365141	rs4936276	rs2514218	0.952883	DRD2	DRD2	NA	NA	TTC12	TTC12	TTC12
27442127	rs73229090	rs73229090	1	35,CHRNA 2,EPHX2	PTK2B,TRI M35,CHRN A2	CHRNA2,E PHX2	EPHX2,SCA RA5	CHRNA2, PTK2B,TRIM35	CHRNA2, PTK2B,TRIM35, EPHX2	CHRNA2, PTK2B,TRIM35, EPHX2
27442329	rs73229093	rs73229090	0.849192	35,CHRNA 2,EPHX2	PTK2B,TRI M35,CHRN A2 PTK2B.TRI	PHX2	EPHX2,SCA RA5	CHRNA2, PTK2B,TRIM35	CHRNA2, PTK2B,TRIM35, EPHX2	CHRNA2, PTK2B,TRIM35, EPHX2
27452241	rs35598594	rs73229090	0.81184	M35,CHRN A2,EPHX2	M35,CHRN A2,EPHX2	RNA2,EPH X2	NA	CHRNA2, PTK2B,TRIM35	CHRNA2, PTK2B,TRIM35, EPHX2	CHRNA2, PTK2B,TRIM35, EPHX2
27453579	rs35236974	rs73229090	0.815871	M35,CHRN	PTK2B,TRI M35,CHRN A2,EPHX2	RNA2,EPH	NA	CHRNA2, PTK2B,TRIM35	CHRNA2, PTK2B,TRIM35, EPHX2	CHRNA2, PTK2B,TRIM35, EPHX2
140140239	re2563258	rs111896713	0.963304	PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1,P0 DHA3,PCDI A5,PCDHA8 PCDHA8,P0 DHA10,PCI HA12,PCDH GA1,PCDH GA3,PCDH GA4,PCDH	HA4,PCDH 6,PCDHA7, CDHA9,PC DHA11,PCD HA13,PCDH GA2,PCDH GB1,PCDH	NA	HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB8, PCDHB16, PCDHG81, PCDH GB2, PCDHG83, PCDHG41, PCDH GB2, PCDHG35, PCDHG34, PCDHG A4, PCDHGA5, PCDHGA5, PCDHG DHGA7, PCDHG34, PCDHG85, P CDHG86, PCDHG87, PCDHG CDHG86, PCDHG87, PCDHG A12, DIAPHL, IOC100505646	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG A11
140140529		rs111896713	0.987169	PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1,PC DHA3,PCD A5,PCDHA4 PCDHA8,PC DHA10,PCI HA12,PCDH GA1,PCDH GA3,PCDH GA4,PCDH	CDHA2,PC HA4,PCDH 6,PCDHA7, CDHA9,PC DHA11,PCD HA13,PCDH GA2,PCDH GB1,PCDH		PCDHB1,PCDHB12,PCDH B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB8, PCDHB16, PCDHG81, PCDH GB2, PCDHG83, PCDHG41, PCDHG42, PCDHG43, PCDHG43, PCDHG44, PCDHG45, PCDHG45, PCDHG46, PC DHG47, PCDHG45, PCDHG85, P CDHG86, PCDHG87, PCDHG CDHG86, PCDHG87, PCDHG CDHG87, PCDHG87, PCDHG87, PCDHG CDHG87, PCDHG87, PCDH67, PCDH67	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG
140140853	rs2337516_	rs111896713	0.965827	CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1,P0 DHA3,PCDH A5,PCDHA8,P0 DHA10,PCI HA12,PCDH GA1,PCDH GA3,PCDH GA4,PCDH	HA4,PCDH 6,PCDHA7, CDHA9,PC DHA11,PCD HA13,PCDH GA2,PCDH GB1,PCDH	NA	HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1; PCDHB2; PCDHB3; PCDHB5; PCDHB7; PCDHB3; PCDHB16; PCDHG81; PCDH GB2; PCDHG83; PCDHG41; PCDHG45; PCDHG45; PCDHG45; PCDHG46; PCD HG47; PCDHG45; PCDHG46; PCDHG46; PCDHG86; PCDHG87; PCDHG CDHG86; PCDHG87; PCDHG8 A12; DIAPHL, IQC100505648	PCDHB7, PCDHB8, PCDHB 16, SLC25A2, TAF7, PCDHG A9
140141779	rs2563256	rs111896713	0.987169					HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	A4,PCDHGA5,PCDHGA6,PC DHGA7,PCDHGA9,PCDHGA 10,PCDHGA11,PCDHGB5,P CDHGB6,PCDHGB7,PCDHG	PCDHB7, PCDHB8, PCDHB 16, SLC25A2, TAF7, PCDHG A8

								PCDHB1,PCDHB2,PCDHB3,	
140142547	rs2563255	rs111896713	0.987169				B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	CDHGB6,PCDHGB7,PCDHG A12,DIAPH1,LOC100505650	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG A7
140142701	rs2563254	rs111896713	0.982092				B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	CDHGB6,PCDHGB7,PCDHG A12,DIAPH1,LOC100505651	PCDHB7, PCDHB8, PCDHB 16, SLC25A2, TAF7, PCDHG A6
140143664	rs111896713	rs111896713		CDHA3,PC DHA1,PCD	PCDHA1,PCDHA2,PC DHA3,PCDHA4,PCDH A5,PCDHA6,PCDHA7, PCDHA8,PCDHA9,PC DHA10,PCDHA11,PCD HA12,PCDHA11,PCDH GA1,PCDHGA2,PCDH GA3,PCDHGB1,PCDH GA3,PCDHGB2	NA	B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1,PCDHB2,PCDHB3, PCDHB5,PCDHB7,PCDHB8, PCDHB7,PCDHG31,PCDH GB2,PCDHG83,PCDHG41,PCDH GB2,PCDHG83,PCDHG4,PCDHG45,PCDHG45,PCDHG462,PCDHG464,PCDHG47,PCDHG 10,PCDHG411,PCDHG85,P CDHG86,PCDHG87,PCDHG A12,DIAPHL JLC0100505652	
140145206		rs111896713		PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1,PCDHA2,PC DHA3,PCDHA4,PCDH A5,PCDHA6,PCDHA7,PCDHA8,PCDHA8,PCDHA9,PC DHA10,PCDHA11,PCD HA12,PCDHA11,PCDH GA1,PCDHGA2,PCDH GA3,PCDHGB1,PCDH GA3,PCDHGB2	NA	PCDHB1,PCDHB12,PCDH B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1,PCDHB2,PCDHB3, PCDHB6,PCDHB7,PCDHB8, PCDHB16,PCDHGB1,PCDH GB2,PCDHGB3,PCDHGA1,P CDHGA2,PCDHGA3,PCDHG A4,PCDHGA5,PCDHGA6,PC DHGA7,PCDHGA9,PCDHGA	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG
	rs11741994	rs111896713		PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1, PCDHA2, PC DHA3, PCDHA4, PCDH A5, PCDHA6, PCDHA7, PCDHA8, PCDHA9, PC DHA10, PCDHA11, PCD HA12, PCDHA13, PCDH GA1, PCDHGA2, PCDH GA3, PCDHGB1, PCDH GA3, PCDHGB2	NA	PCDHB1,PCDHB12,PCDH B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB8, PCDHB6, PCDHGB1, PCDH GB2, PCDHGB3, PCDHGA1, PC CDHGA2, PCDHGA3, PCDHGA A4, PCDHGA5, PCDHGA5, PCDHGA DHGA7, PCDHGA5, PCDHGB, PCDHGB6, PCDHGB7, PCDHG 10, PCDHGA11, PCDHGB5, PC CDHGB6, PCDHGB7, PCDHG A12, DIAPHL 10, C010505654	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG A3
140146345	rs7737424	rs111896713		PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1,PCDHA2,PC DHA3,PCDHA4,PCDH A5,PCDHA6,PCDHA7, PCDHA8,PCDHA9,PC DHA10,PCDHA11,PCD HA12,PCDHA11,PCDH GA1,PCDHGA2,PCDH GA3,PCDHGB1,PCDH GA3,PCDHGB2	NA	B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB8, PCDHB5, PCDHG81, PCDH GB2, PCDHG83, PCDHG41, PC DHGA2, PCDHGA3, PCDHG A4, PCDHGA5, PCDHGA6, PC DHGA7, PCDHGA5, PCDHG DHGA7, PCDHGB5, PCDHG CDHGB6, PCDHGB7, PCDHG A12, DIAPHL, LOC100505655	
140146821	rs13168514	rs111896713	0.924772	PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1, PCDHA2, PC DHA3, PCDHA4, PCDH A5, PCDHA6, PCDHA7, PCDHA8, PCDHA9, PC DHA10, PCDHA11, PCD HA12, PCDHA12, PCDH GA1, PCDHGA2, PCDH GA3, PCDHGB1, PCDH GA3, PCDHGB2	NA	B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB, PCDHB5, PCDHB7, PCDH GB2, PCDHGB3, PCDHG41, PC DHGA2, PCDHGA3, PCDHG A4, PCDHGA5, PCDHGA5, PCDHGA DHGA7, PCDHGA5, PCDHGB, PCDHGB6, PCDHGB7, PCDHG CDHGB6, PCDHGB7, PCDHG A12, DIAPHL, LOC100505656	
		rs111896713		PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1, PCDHA2, PC DHA3, PCDHA4, PCDH A5, PCDHA6, PCDHA7, PCDHA8, PCDHA9, PC DHA10, PCDHA11, PCD HA12, PCDHA12, PCDH GA1, PCDHGA2, PCDH GA3, PCDHGB1, PCDH GA3, PCDHGB2	NA	PCDHB1,PCDHB12,PCDH B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC	РСОНВ1, РСОНВ2, РСОНВ3, РСОНВ5, РСОНВ7, РСОНВ8, РСОНВ6, РСОНGB1, РСОН GB2, РСОНGB3, РСОНGA1, Р, СОНGA2, РСОНGA3, РСОНGA4, РСОНGA4, РСОНGA5, РСОНGA9, РСОНGA 10, РСОНGA11, РСОНGB5, Р СОНGB6, РСОНGB7, РСОНG А12, DIAPHI, LOC 100505657	PCDHB7,PCDHB8,PCDHB
	rs12659129			PCDHA2,P CDHA3,PC DHA1,PCD HB1,PCDH	PCDHA1, PCDHA2, PC DHA3, PCDHA4, PCDH A5, PCDHA6, PCDHA7, PCDHA8, PCDHA9, PC DHA10, PCDHA11, PCD HA12, PCDHA11, PCD HA12, PCDHA13, PCDH GA1, PCDHGA1, PCDH GA3, PCDHGB1, PCDH GA3, PCDHGB2		PCDHB1,PCDHB12,PCDH B13,PCDHGA1,PCDHGA2, PCDHGA3,PCDHGA4,PCD HGA5,PCDHGA6,PCDHGA 7,PCDHGA8,PCDHGA9,PC DHGB1,PCDHGB2,PCDHG	PCDHB1, PCDHB2, PCDHB3, PCDHB5, PCDHB7, PCDHB PCDHB6, PCDHG81, PCDH GB2, PCDHG83, PCDHG41, PCDH GB2, PCDHG83, PCDHG4, PCDHG40, PCDHG40, PCDHG40, PCDHG40, PCDHG40, PCDHG40, PCDHG40, PCDHG50, PCDH500, PC	PCDHB7,PCDHB8,PCDHB 16,SLC25A2,TAF7,PCDHG

Table 3.12 | Locus-specific chromosomal contacts in fetal brain compared to hiPSC-derived cells.

Comparison of genes identified as interacting with 36 credible SNPs of interest (Won *et al.*, 2016) between fetal brain and hiPSC Hi-C interactions. Credsnp, credible SNP called by CAVIAR; indexsnp, index SNP of the haplotype in which

credible SNP lies; R_to_index, correlation between credible SNP and its index SNP; hgnc_CP, gene interacting with credible SNP in cortical plate samples; hgnc_GZ, gene interacting with credible SNP in germinal zone samples; hgnc_ES, gene interacting with credible SNP in embryonic stem cell samples; hgnc_IMR90, gene interacting with credible SNP in IMR90 samples; NPC, Neuron, Glia – genes interacting with credible SNPs in cell types from the present study.

CRISPRa

D	ТҮРЕ	COORDINATES (Region, gRNA)		REVERSE COMPLEMENT	IVT F1 PRIMER	IVT R1 PRIMER	GENE
S1	Loop-SNP1	(chr12:103160000-104160000) 103573681-103573700 FWD	TTCTAGTTTAGACAAC AGGA	TCCTGTTGTCTAAACT AGAA	TAATACGACTCACTATAGCTAGTTTAG ACAACAGGA	TTCTAGCTCTAAAACTCCTGTT GTCTAAACTAG	ASCL
S2	Loop-SNP2	(chr12:103160000-104160000) 103573733-103573752 REV	CTTCAGATCATTCTTC CTGG	CCAGGAAGAATGATC TGAAG	TAATACGACTCACTATAGTCAGATCAT TCTTCCTGG	TTCTAGCTCTAAAACCCAGGAA GAATGATCTGA	ASCL
S3	Loop-SNP3	(chr12:103160000-104160000) 103573810-103573829 FWD	ACAGTGGGACAGAGT TTAGG	CCTAAACTCTGTCCC ACTGT	TAATACGACTCACTATAGAGTGGGACA GAGTTTAGG	TTCTAGCTCTAAAACCCTAAAC TCTGTCCCACT	ASCL
S4	Loop-SNP4	(chr12:103160000-104160000) 103574251-103574270 REV	AACTGTACTAAGCTAT CCAG	CTGGATAGCTTAGTA CAGTT	TAATACGACTCACTATAGCTGTACTAA GCTATCCAG	TTCTAGCTCTAAAACCTGGATA GCTTAGTACAG	ASCL
S5	Loop-SNP5	(chr12:103160000-104160000) 103574252-103574271 REV		TGGATAGCTTAGTAC AGTTC	TAATACGACTCACTATAGACTGTACTA AGCTATCCA	TTCTAGCTCTAAAACTGGATAG CTTAGTACAGT	ASCL
S6	Control1	(chr12:103160000-104160000) 103469557-103469576 REV	CTCTCCCATAAACAC TACCC	GGGTAGTGTTTATGG GAGAG	TAATACGACTCACTATAGCTCCCATAA ACACTACCC	TTCTAGCTCTAAAACGGGTAGT GTTTATGGGAG	ASCI
S7	Control2	(chr12:103160000-104160000) 103469613-103469632 FWD	TAAGACTCTATACATG ACCA	TGGTCATGTATAGAG TCTTA	TAATACGACTCACTATAGAGACTCTAT ACATGACCA	TTCTAGCTCTAAAACTGGTCAT GTATAGAGTCT	ASCI
S8	Control3	(chr12:103160000-104160000) 103469622-103469641 FWD	ATACATGACCATGGG TGATG	CATCACCCATGGTCA TGTAT	TAATACGACTCACTATAGACATGACCA TGGGTGATG	TTCTAGCTCTAAAACCATCACC CATGGTCATGT	ASCI
S9	Control4	(chr12:103160000-104160000) 103470279-103470298 REV	CCAGTTCTAGTATGA ATCTG	CAGATTCATACTAGA ACTGG	TAATACGACTCACTATAGAGTTCTAGT ATGAATCTG	TTCTAGCTCTAAAACCAGATTC ATACTAGAACT	ASCI
S10	Control5	(chr12:103160000-104160000) 103470309-103470328 FWD	AATTTTACAAAGTCGC AAGG	CCTTGCGACTTTGTA AAATT	TAATACGACTCACTATAGTTTTACAAA GTCGCAAGG	TTCTAGCTCTAAAACCCTTGCG ACTTTGTAAAA	ASCI
S11	Promoter1	(chr12:103160000-104160000) 103350547-103350566 REV	TATGGCAGGGACGTC CCCCT	AGGGGGACGTCCCT GCCATA	TAATACGACTCACTATAGTGGCAGGG ACGTCCCCCT	TTCTAGCTCTAAAACAGGGGG ACGTCCCTGCCA	ASCI
S12	Promoter2	(chr12:103160000-104160000) 103350598-103350617 FWD	TCCTCGGTGTCGCTT CCCCG	CGGGGAAGCGACAC CGAGGA	TAATACGACTCACTATAGCTCGGTGTC GCTTCCCCG	TTCTAGCTCTAAAACCGGGGA AGCGACACCGAG	ASCI
S13	Promoter3	(chr12:103160000-104160000) 103350602-103350621 REV	GCCGCGGGGGAAGCG ACACCG	CGGTGTCGCTTCCCC GCGGC	TAATACGACTCACTATAGCGCGGGGA AGCGACACCG	TTCTAGCTCTAAAACCGGTGTC GCTTCCCCGCG	ASCI
S14	Promoter4	(chr12:103160000-104160000) 103350875-103350894 FWD	TCCAATTTCTAGGGT CACCG	CGGTGACCCTAGAAA TTGGA	TAATACGACTCACTATAGCAATTTCTA GGGTCACCG	TTCTAGCTCTAAAACCGGTGAC CCTAGAAATTG	ASC
S15	Promoter5	(chr12:103160000-104160000) 103351166-103351185 REV	CTCCCGGCTGAATAA ACAGG		TAATACGACTCACTATAGCCCGGCTGA ATAAACAGG		ASC
F1	Loop-SNP1	(chrX:67810000-68810000) 68378608-68378627 REV		CCTTAGGTGGTGCCC GTCGG	TAATACGACTCACTATAGGACGGGCA CCACCTAAGG	TTCTAGCTCTAAAACCCTTAGG TGGTGCCCGTC	EFNI
F2	Loop-SNP2	(chrX:67810000-68810000) 68378823-68378842 REV	CACTGCAAGTACAGA AACGT	ACGTTTCTGTACTTG CAGTG	TAATACGACTCACTATAGCTGCAAGTA CAGAAACGT	TTCTAGCTCTAAAACACGTTTC TGTACTTGCAG	EFNI
F3	Loop-SNP3	(chrX:67810000-68810000) 68379023-68379042 REV	GGCCGGGGTTACTTC TAGGG	CCCTAGAAGTAACCC CGGCC	TAATACGACTCACTATAGCCGGGGTTA CTTCTAGGG	TTCTAGCTCTAAAACCCCCTAGA AGTAACCCCGG	EFNE
F4	Loop-SNP4	(chrX:67810000-68810000) 68379038-68379057 REV	ACATGCCTAACTGAT GGCCG	CGGCCATCAGTTAGG CATGT	TAATACGACTCACTATAGATGCCTAAC TGATGGCCG	TTCTAGCTCTAAAACCGGCCAT CAGTTAGGCAT	EFNE
F5	Loop-SNP5	(chrX:67810000-68810000) 68379044-68379063 REV	AAGGCGACATGCCTA ACTGA	TCAGTTAGGCATGTC GCCTT	TAATACGACTCACTATAGGGCGACATG CCTAACTGA	TTCTAGCTCTAAAACTCAGTTA GGCATGTCGCC	EFNE
F6	Control1	(chrX:67810000-68810000) 68249998-68250017 REV		CACATATTTTGGGGG AGGCC		TTCTAGCTCTAAAACCACATAT TTTGGGGGGAGG	EFNE
F7	Control2	(chrX:67810000-68810000) 68250062-68250081 REV	GCCCTAGTGCTAGGG GAATG			TTCTAGCTCTAAAACCATTCCC CTAGCACTAGG	EFNE
F8	Control3	(chrX:67810000-68810000) 68250069-68250088 REV		CTAGCACTAGGGCTT			EFNE
F9	Control4	(chrX:67810000-68810000) 68250158-68250177 FWD		TGTTGGTTAGAGTGG CCCAG	TAATACGACTCACTATAGGGGCCACTC TAACCAACA		EFN
	Control5	(chrX:67810000-68810000) 68250187-68250206 FWD	CCACTTAACAGTCCT	TGACAAGGACTGTTA AGTGG	TAATACGACTCACTATAGACTTAACAG		EFN
F10		(chrX:67810000-68810000)			TAATACGACTCACTATAGACGTGGGTA CGTCCTCTG	TTCTAGCTCTAAAACCAGAGGA	
	Promoter1		CTCTG				
F11	Promoter1 Promoter2	68047822-68047841 FWD (chrX:67810000-68810000) 68048066-68048085 REV	CTCTG GATAAAGAAAGACAC CTCGA		TAATACGACTCACTATAGTAAAGAAAG ACACCTCGA	TTCTAGCTCTAAAACTCGAGGT GTCTTTCTTTA	EFN
F11 F12	Promoter2	68047822-68047841 FWD (chrX:67810000-68810000) 68048066-68048085 REV (chrX:67810000-68810000)	GATAAAGAAAGACAC CTCGA AGCCCACTAAAGCCT	TCGAGGTGTCTTTCT TTATC ACGTAAGGCTTTAGT	ACACCTCGA TAATACGACTCACTATAGCCCACTAAA	GTCTTTCTTTA TTCTAGCTCTAAAACACGTAAG	EFNE
F11 F12 F13	Promoter2 Promoter3	68047822-68047841 FWD (chrx;67810000-68810000) 68048066-68048085 REV (chrx:67810000-68810000) 68048398-68048417 FWD (chrx:67810000-68810000)	GATAAAGAAAGACAC CTCGA AGCCCACTAAAGCCT TACGT ACAGGCTGTCCTTGA	TCGAGGTGTCTTTCT TTATC ACGTAAGGCTTTAGT GGGCT CCGTGTCAAGGACAG	ACACCTCGA TAATACGACTCACTATAGCCCACTAAA GCCTTACGT TAATACGACTCACTATAGAGGCTGTCC	GTCTTTCTTTA TTCTAGCTCTAAAACACGTAAG GCTTTAGTGGG TTCTAGCTCTAAAACCCGTGTC	EFN
F10 F11 F12 F13 F14 F15	Promoter2	68047822-68047841 FWD (chrX:67810000-68810000) 68048066-68048085 REV (chrX:67810000-68810000) 68048398-68048417 FWD	GATAAAGAAAGACAC CTCGA AGCCCACTAAAGCCT TACGT ACAGGCTGTCCTTGA CACGG	TCGAGGTGTCTTTCT TTATC ACGTAAGGCTTTAGT GGGCT CCGTGTCAAGGACAG CCTGT	ACACCTCGA TAATACGACTCACTATAGCCCACTAAA GCCTTACGT TAATACGACTCACTATAGAGGCTGTCC	GTCTTTCTTTA TTCTAGCTCTAAAACACGTAAG GCTTTAGTGGG TTCTAGCTCTAAAACCCGTGTC AAGGACAGCCT	

			-				
MA1	Loop-SNP1	(chr5:137860000-138860000) 137914322-137914341 FWD	TACCGATGCAAAGAA GACAG	CTGTCTTCTTTGCATC GGTA	TAATACGACTCACTATAGCCGATGCAA AGAAGACAG	TTCTAGCTCTAAAACCTGTCTT CTTTGCATCGG	MATR3
MA2	Loop-SNP2	(chr5:137860000-138860000) 137914389-137914408 FWD	TGTTCTTACGTTGTAC ATGA	TCATGTACAACGTAA GAACA	TAATACGACTCACTATAGTTCTTACGT TGTACATGA	TTCTAGCTCTAAAACTCATGTA CAACGTAAGAA	MATR3
MA3	Loop-SNP3	(chr5:137860000-138860000) 137914391-137914410 FWD	TTCTTACGTTGTACAT GACG	CGTCATGTACAACGT AAGAA	TAATACGACTCACTATAGCTTACGTTG TACATGACG	TTCTAGCTCTAAAACCGTCATG TACAACGTAAG	MATR3
MA4	Loop-SNP4	(chr5:137860000-138860000) 137914394-137914413 FWD	TTACGTTGTACATGA CGGGG	CCCCGTCATGTACAA CGTAA	TAATACGACTCACTATAGACGTTGTAC ATGACGGGG	TTCTAGCTCTAAAACCCCCGTC ATGTACAACGT	MATR3
MA5	Loop-SNP5	(chr5:137860000-138860000) 137914401-137914420 FWD	GTACATGACGGGGC GGAGAA	TTCTCCGCCCCGTCA TGTAC	TAATACGACTCACTATAGACATGACGG GGCGGAGAA	TTCTAGCTCTAAAACTTCTCCG CCCCGTCATGT	MATR3
MA6	Control1	(chr5:137860000-138860000) 138278537-138278556 REV	CCTCACACAAGGGAG CCGCA	TGCGGCTCCCTTGTG TGAGG	TAATACGACTCACTATAGTCACACAAG GGAGCCGCA	TTCTAGCTCTAAAACTGCGGCT CCCTTGTGTGA	MATR3
MA7	Control2	(chr5:137860000-138860000) 138278547-138278566 REV	GGCCTGGGAACCTCA CACAA	TTGTGTGAGGTTCCC AGGCC	TAATACGACTCACTATAGCCTGGGAAC CTCACACAA	TTCTAGCTCTAAAACTTGTGTG AGGTTCCCAGG	MATR3
MA8	Control3	(chr5:137860000-138860000) 138278824-138278843 REV	AAGTGTGCAGACTCC AGATG	CATCTGGAGTCTGCA CACTT	TAATACGACTCACTATAGGTGTGCAGA CTCCAGATG	TTCTAGCTCTAAAACCATCTGG AGTCTGCACAC	MATR3
MA9	Control4	(chr5:137860000-138860000) 138278931-138278950 REV	GGTGAGAATAGATGA CACAG	CTGTGTCATCTATTCT CACC	TAATACGACTCACTATAGTGAGAATAG ATGACACAG	TTCTAGCTCTAAAACCTGTGTC ATCTATTCTCA	MATR3
MA10	Control5	(chr5:137860000-138860000) 138278933-138278952 REV	GTGGTGAGAATAGAT GACAC	GTGTCATCTATTCTCA CCAC	TAATACGACTCACTATAGGGTGAGAAT AGATGACAC	TTCTAGCTCTAAAACGTGTCAT CTATTCTCACC	MATR3
MA11	Promoter1	(chr5:137860000-138860000) 138608459-138608478 REV	TAAAACCAGGCAGAT TGGGT	ACCCAATCTGCCTGG TTTTA	TAATACGACTCACTATAGAAACCAGGC AGATTGGGT	TTCTAGCTCTAAAACACCCCAAT CTGCCTGGTTT	MATR3
MA12	Promoter2	(chr5:137860000-138860000) 138608898-138608917 REV	CCATTGCCAGGTGAA CCCCT	AGGGGTTCACCTGGC AATGG	TAATACGACTCACTATAGATTGCCAGG TGAACCCCT	TTCTAGCTCTAAAACAGGGGTT CACCTGGCAAT	MATR3
MA13	Promoter3	(chr5:137860000-138860000) 138609219-138609238 FWD	CGGCGAAGAATCCCA CTGCA	TGCAGTGGGATTCTT CGCCG	TAATACGACTCACTATAGGCGAAGAAT CCCACTGCA	TTCTAGCTCTAAAACTGCAGTG GGATTCTTCGC	MATR3
MA14	Promoter4	(chr5:137860000-138860000) 138609310-138609329 FWD	TGGAAAGATCCCGAA GACCG	CGGTCTTCGGGATCT TTCCA	TAATACGACTCACTATAGGAAAGATCC CGAAGACCG	TTCTAGCTCTAAAACCGGTCTT CGGGATCTTTC	MATR3
MA15	Promoter5	(chr5:137860000-138860000) 138609317-138609336 FWD	ATCCCGAAGACCGTG GACCA	TGGTCCACGGTCTTC GGGAT	TAATACGACTCACTATAGCCCGAAGAC CGTGGACCA	TTCTAGCTCTAAAACTGGTCCA CGGTCTTCGGG	MATR3
SO1	Loop-SNP1	chr3:180560000-181560000 180928342-180928361 REV	TAGGAACAGAAAATT ATGCT	AGCATAATTTTCTGTT CCTA	TAATACGACTCACTATAGGGAACAGAA AATTATGCT	TTCTAGCTCTAAAACAGCATAA TTTTCTGTTCC	SOX2
SO2	Loop-SNP2	chr3:180560000-181560000 180928424-180928443 FWD	ATGAAGTTCCACCCA GTTTG	CAAACTGGGTGGAAC TTCAT	TAATACGACTCACTATAGGAAGTTCCA CCCAGTTTG	TTCTAGCTCTAAAACCAAACTG GGTGGAACTTC	SOX2
SO3	Loop-SNP3	chr3:180560000-181560000 180928425-180928444 FWD	TGAAGTTCCACCCAG TTTGG	CCAAACTGGGTGGAA CTTCA	TAATACGACTCACTATAGAAGTTCCAC CCAGTTTGG	TTCTAGCTCTAAAACCCAAACT GGGTGGAACTT	SOX2
SO4	Loop-SNP4	chr3:180560000-181560000 180928530-180928549 REV	TATTAGGAAAAATCC GCCAG	CTGGCGGATTTTTCC TAATA	TAATACGACTCACTATAGTTAGGAAAA ATCCGCCAG	TTCTAGCTCTAAAACCTGGCG GATTTTTCCTAA	SOX2
SO5	Loop-SNP5	chr3:180560000-181560000 180928608-180928627 FWD	ACTCTAAAGTTTCATC AGGA	TCCTGATGAAACTTTA GAGT	TAATACGACTCACTATAGTCTAAAGTT TCATCAGGA	TTCTAGCTCTAAAACTCCTGAT GAAACTTTAGA	SOX2
SO6	Control1	(chr3:180560000-181560000) 181180191-181180210 FWD	TGGCTTAATAGTGAG TTACG	CGTAACTCACTATTAA GCCA	TAATACGACTCACTATAGGCTTAATAG TGAGTTACG	TTCTAGCTCTAAAACCGTAACT CACTATTAAGC	SOX2
SO7	Control2	(chr3:180560000-181560000) 181180624-181180643 FWD	TGAGCCATAAAATAG TCACT	AGTGACTATTTTATG GCTCA	TAATACGACTCACTATAGAGCCATAAA ATAGTCACT	TTCTAGCTCTAAAACAGTGACT ATTTTATGGCT	SOX2
SO8	Control3	(chr3:180560000-181560000) 181180998-181181017 FWD	TCTGTTGTGTATCAG GACCA	TGGTCCTGATACACA ACAGA	TAATACGACTCACTATAGTGTTGTGTA TCAGGACCA	TTCTAGCTCTAAAACTGGTCCT GATACACAACA	SOX2
SO9	Control4	(chr3:180560000-181560000) 181181058-181181077 FWD	ACTTTAAAAAATCCCA CACG	CGTGTGGGGATTTTTT AAAGT	TAATACGACTCACTATAGTTTAAAAAAT CCCACACG	TTCTAGCTCTAAAACCGTGTGG GATTTTTTAAA	SOX2
SO10	Control5	(chr3:180560000-181560000) 181181105-181181124 FWD	AATAACTAATAGCTAC AAGt	aCTTGTAGCTATTAGT TATT	TAATACGACTCACTATAGTAACTAATA GCTACAAGt	TTCTAGCTCTAAAACaCTTGTA GCTATTAGTTA	SOX2
SO11	Promoter1	(chr3:180560000-181560000) 181428583-181428602 FWD	CACCACAATGGAAAT CTACG	CGTAGATTTCCATTG TGGTG	TAATACGACTCACTATAGCCACAATGG AAATCTACG	TTCTAGCTCTAAAACCGTAGAT TTCCATTGTGG	SOX2
SO12	Promoter2	(chr3:180560000-181560000) 181428799-181428818 FWD	CCGAAACCCTTCTTA CGGGG	CCCCGTAAGAAGGGT TTCGG	TAATACGACTCACTATAGGAAACCCTT CTTACGGGG	TTCTAGCTCTAAAACCCCCGTA AGAAGGGTTTC	SOX2
SO13	Promoter3	(chr3:180560000-181560000) 181429039-181429058 FWD	TAAGAACAGAGCAAG TTACG	CGTAACTTGCTCTGT TCTTA	TAATACGACTCACTATAGAGAACAGAG CAAGTTACG	TTCTAGCTCTAAAACCGTAACT TGCTCTGTTCT	SOX2
SO14	Promoter4	(chr3:180560000-181560000) 181429420-181429439 REV	AAACAGCACTAAGAC TACGT	ACGTAGTCTTAGTGC TGTTT	TAATACGACTCACTATAGACAGCACTA AGACTACGT	TTCTAGCTCTAAAACACGTAGT CTTAGTGCTGT	SOX2
SO15	Promoter5	(chr3:180560000-181560000) 181429421-181429440 REV	TAAACAGCACTAAGA CTACG	CGTAGTCTTAGTGCT GTTTA	TAATACGACTCACTATAGAACAGCACT AAGACTACG	TTCTAGCTCTAAAACCGTAGTC TTAGTGCTGTT	SOX2
5015	Promoter5	101429421-181429440 REV	CTACG	GIIIA	AAGACTACG	TTAGTGCTGTT	SOX

Cas9

ID	TYPE	COORDINATES (Region, gRNA)		REVERSE COMPLEMENT	IVT F1 PRIMER	IVT R1 PRIMER		Targeted credible SNP rsID
PC1		(chr5:140109000-141100000) 140142674-140142693 REV	agagttatccttctgccaaa	tttggcagaaggataactct		aaggataact	PCDH	rs2563254
PC2		(chr5:140109000-141100000) 140142705-140142724 REV	gaggccatatgtccaagat c	gatcttggacatatggcctc	TAATACGACTCACTATAGggcca tatgtccaagatc	TTCTAGCTCTAAAACgatcttgg acatatggcc		rs2563254
PC3				tgtacaggagacacatttta		gagacacattt	PCDH	rs6891559
PC4		(chr5:140109000-141100000) 140146895-140146914 REV	ttcccagatagggccagg cg		TAATACGACTCACTATAGcccag atagggccaggcg	TTCTAGCTCTAAAACcgcctgg ccctatctggg		rs13168670
PC5		(chr5:140109000-141100000) 140147218-140147237 FWD	ttgatgtctgtcattaaatg		TAATACGACTCACTATAGgatgtc tgtcattaaatg		PCDH	rs12659129

-								
		(chr12:103350000-103580000)	ATTCAGAATGACG	CCAAATCCGTCAT	TAATACGACTCACTATAGTCAG			
AS16		103573566-103573585 FWD	GATTTGG	TCTGAAT	AATGACGGATTTGG	TCCGTCATTCTGA	ASCL1	rs10860949
	Loop-SNP-	(chr12:103350000-103580000)	CGGATTTGGTGGT	CAACTGCACCACC	TAATACGACTCACTATAGGATT	TTCTAGCTCTAAAACCAACT		
AS17	del2	103573577-103573596 FWD	GCAGTTG	AAATCCG	TGGTGGTGCAGTTG	GCACCACCAAATC	ASCL1	rs10860949
	Loop-SNP-	(chr12:103350000-103580000)	TCAAAGAGAGTTG	ACGAAGTCAACTC	TAATACGACTCACTATAGAAAG	TTCTAGCTCTAAAACACGAA		
AS18	del3	103574184-103574203 FWD	ACTTCGT	TCTTTGA	AGAGTTGACTTCGT	GTCAACTCTCTTT	ASCL1	rs7306170
	Loop-SNP-	(chr12:103350000-103580000)	GAGTGGTCATTCT	AGTAATTAGAATG	TAATACGACTCACTATAGGTG	TTCTAGCTCTAAAACAGTAA		
AS19	del4	103575773-103575792 REV	AATTACT	ACCACTC	GTCATTCTAATTACT	TTAGAATGACCAC	ASCL1	rs10860950
	Loop-SNP-	(chr12:103350000-103580000)	TAGAATGACCACT	CCAGAAGAGTGGT	TAATACGACTCACTATAGGAAT	TTCTAGCTCTAAAACCCAGA		
AS20	del5	103575779-103575798 FWD	CTTCTGG	CATTCTA	GACCACTCTTCTGG	AGAGTGGTCATTC	ASCL1	rs10860950
	Loop-SNP-	chr5:137911382-137916382	ggagaaaaggaaataag		TAATACGACTCACTATAGagaaa	TTCTAGCTCTAAAACtccttctttc		
MA16	del1	137913870-137913889 FWD			aggaaataaggaa	cctccttc	MATR3	rs409273
	Loop-SNP-	chr5:137915326-137920326			TAATACGACTCACTATAGaattag	TTCTAGCTCTAAAACcttgtaaa		
MA17	del2	137917793-137917812 FWD	ggaattagtgcctttacaag	cttgtaaaggcactaattcc	tgcctttacaag	ggcactaatt	MATR3	rs11957778
	Loop-SNP-	chrX:68374626-68379626	TTTTCAGGAGCTG	AATTTGCCAGCTC	TAATACGACTCACTATAGTTCA	TTCTAGCTCTAAAACAATTT		
EF16	del1	68377097-68377116 FWD	GCAAATT	CTGAAAA	GGAGCTGGCAAATT	GCCAGCTCCTGAA	EFNB1	rs5937157
	Loop-SNP-	chrX:68374704-68379704	AGAGCTGGAAAGG	GAAATGTCCTTTC	TAATACGACTCACTATAGAGCT	TTCTAGCTCTAAAACGAAAT		
EF17	del2	68377479-68377498 FWD	ACATTTC	CAGCTCT	GGAAAGGACATTTC	GTCCTTTCCAGCT	EFNB1	rs2361468
	Loop-SNP-	chrX:68374704-68379704	GAAAGGACATTTC	CTGACCCGAAATG	TAATACGACTCACTATAGAAG	TTCTAGCTCTAAAACCTGAC		
		68377486-68377505 FWD	GGGTCAG	TCCTTTC	GACATTTCGGGTCAG	CCGAAATGTCCTT	EFNB1	rs2885287
	Loop-SNP-	(chr22:41400000-41600000)	tcactgcgaactccgcccc	aaaaaaaattcacaat	TAATACGACTCACTATAGactgc	TTCTAGCTCTAAAACaaaaac		
		41427455-41427474 FWD					EP300	rs9607768

Table 3.13 | Oligonucleotide sequences for gRNA in vitro transcription.

Oligos used to generate gRNAs through in vitro transcription (IVT). ID, unique oligo name; Type, condition of the experiment; Name, contains coordinate and strand information (from Benchling); Sequence, gRNA sequence; F1 and R1 Primer, blue corresponds to T7 Promoter sequence (F1) and tracrRNA (R1) as per manual, p.9-11

(https://assets.thermofisher.com/TFS-

Assets/LSG/manuals/geneart_precision_gRNA_synthesis_kit_man.pdf)

NAME	SEQUENCE
ASCL1-1-F	GGAGCTTCTCGACTTCACCA
ASCL1-1-R	AACGCCACTGACAAGAAAGC
EFNB1-1-F	CTCCCTGGGTGACTCTGATG
EFNB1-1-R	TGATGAGCAGGAAGATGACG
MATR3-3-F	AAGCAAGAGCTTGGACGTGT
MATR3-3-R	AACCATTGCCATTGCATCTT
SOX2-F	GCTAGTCTCCAAGCGACGAA
SOX2-R	GCAAGAAGCCTCTCCTTGAA
EP300-F	ATGACACACTGCCAGTCAGG
EP300-R	TTTTTGAGGGGGAGACACAC
PCDHA7-F	ACCCAAGACAGACCTCATGG
PCDHA7-R	AATGCCAGCCTCCTCTAGGT
PCDHA8-F	AGAGGGTGTGCTCTGGTGAG
PCDHA8-R	GAGGCAGAGTAACGCCAGTC
PCDHA10-F	GTAGATGTGGACGGGGAAGA
PCDHA10-R	CTCAGGGAGGCAGAGTAACG

Table 3.14 | qPCR primer sequences for RNA quantification in CRISPR experiments.

Primer sequences to measure gene expression of target genes from CRISPR experiments.

	d in 10kb bins corresp actions anchored in F			n 10kb bins corresp anchored in PGC2-		in PGC2+ CLOZUK randomly	b bins corresponding to speciloci; with clusters of co-local removed such that only one g	ized genes (e.g., PCDH) gene is present
NPC (309)	Neurons (328)	Glia (165)	NPC (386)	Neurons (385)	Glia (201)	NPC (clusters removed)	Neurons (clusters removed)	Glia (clusters removed)
AADAT	ABCB9	ABCB9	AADAT	ABCB9	ABCB9	AADAT	ABCB9	ABCB9
ADSS	ACTR5	ADSL	ABCD3	ACTG1P17	ADSL	ABCD3	ACTG1P17	ADSL
AKT3	ADAMTS9-AS2	ADSS	ACTR8	ACTR5	ADSS	ACTR8	ACTR5	ADSS
AMBRA1 ANKRD44	ADAMTSL3 ADGRA3	AKT3 ANKHD1	ADGRG6 ADSS	ACTR8 ADAMTS9-AS2	AKT3 ALMS1	ADGRG6 ADSS	ACTR8 ADAMTS9-AS2	AKT3 ALMS1
	ADGRAS	ANKHD1-	AD33	ADAMI 39-A32	ALIVIS I	AD33	ADAMT 39-A32	ALIVIS I
ANP32E	ADIG	EIF4EBP3	AIG1	ADAMTSL3	ALMS1P1	AIG1	ADAMTSL3	ALMS1P1
AOX1	ADSS	ANKRD44	AKT3	ADGRA3	ANKHD1	AKT3	ADGRA3	ANKHD1
		1			ANKHD1-			
AOX2P	AKAP13	AOX1	ALAS1	ADIG	EIF4EBP3	ALAS1	ADIG	ANKHD1-EIF4EBP3
AP1S3	AKT3	AOX2P	ALMS1	ADSS	ANKRD44	ALMS1	ADSS	ANKRD44
ARNT	AMBRA1	ARL6IP4	ALMS1P1	AIG1	AOX1	ALMS1P1	AIG1	AOX1
ASB5 ASTN1	ANKRD44 ANP32E	ARTN ATF7IP2	AMBRA1 ANKRD44	AKAP13 AKT3	AOX2P AP3B2	AMBRA1 ANKRD44	AKAP13 AKT3	AOX2P AP3B2
ATP2A2	AP1S3	BOLA2	ANKRD45	ALAS1	ARL6IP4	ANKRD45	ALAS1	ARL6IP4
ATP6V0A2	ARHGAP40	BOLL	ANP32E	ALKBH5	ARTN	ANP32E	ALKBH5	ARTN
B4GALT2	ARL6IP4	C12orf65	AOX1	ALMS1	ATF7IP2	AOX1	ALMS1	ATF7IP2
BANK1	ASTN1	C1orf100	AOX2P	ALMS1P1	AUP1	AOX2P	ALMS1P1	AUP1
BNIPL	ATG13	C1orf101	AP1S3	AMBRA1	BOLA2	AP1S3	AMBRA1	BOLA2
BOLL	ATPAF2	C2orf69	ARL14	ANKRD44	BOLL	ARL14	ANKRD44	BOLL
BRD8	ATXN7	C7orf50	ARNT	ANKRD45	BRINP3	ARNT	ANKRD45	BRINP3
BRINP2	BAHD1	CCDC150	ASB5	ANP32E	C12orf65	ASB5	ANP32E	C12orf65
C11orf49	BAZ2A	CCDC39	ASH2L	AP1S3	C1orf100	ASH2L	AP1S3	C1orf100
C1orf101 C2orf69	BOLA2 BOLL	CD46 CNBD1	ASTN1 ATP2A2	AP3B2 ARHGAP15	C1orf101 C2orf69	ASTN1 ATP2A2	AP3B2 ARHGAP15	C1orf101 C2orf69
C4BPA	BRINP2	CNKSR2	ATP6V0A2	ARHGAP40	C7orf50	ATP6V0A2	ARHGAP40	C7orf50
C4DFA C7orf31	C1orf100	CNK3K2 CNTN4	B4GALT2	ARL6IP4	CCDC150	B4GALT2	ARL6IP4	CCDC150
CA6	C1orf101	COQ10B	BANK1	ASTN1	CCDC39	BANK1	ASTN1	CCDC39
CACNA1C	C1orf116	CSMD1	BNIPL	ATG13	CCT7	BNIPL	ATG13	CCT7
CACNA1D	C2orf69	CTNND1	BOLA3	ATPAF2	CD46	BOLA3	ATPAF2	CD46
CBR4	C2orf82	CUL3	BOLA3-AS1	ATXN7	CHRM3	BOLA3-AS1	ATXN7	CHRM3
CCDC150	C3orf49	DHX35	BOLL	BAHD1	CNBD1	BOLL	BAHD1	CNBD1
CCDC24	C7orf50	DNAH10	BRD8	BAZ2A	CNKSR2	BRD8	BAZ2A	CNKSR2
CCDC30	CA6	DOCK10	BRINP2	BNIP3L	CNTN4	BRINP2	BNIP3L	CNTN4
CCDC39	CA8 CACNA1C	DPP4 DPYD	BRINP3	BOLA2 BOLL	COQ10B CPEB1-AS1	BRINP3	BOLA2 BOLL	COQ10B CPEB1-AS1
CD46 CDC25C	CACNA1C-AS4	EP300	C11orf49 C1orf101	BRINP2	CPEBT-AST CSMD1	C11orf49 C1orf101	BRINP2	CSMD1
CEP170	CACNA1C-IT3	ERMAP	C2orf69	BRINP3	CTNND1	C2orf69	BRINP3	CTNND1
CETN3	CBR4	EAM57B	C4BPA	C1orf100	CUL3	C4BPA	C1orf100	CUL3
CFAP57	CCDC150	FAM83D	C7orf31	C1orf101	DAAM1	C7orf31	C1orf101	DAAM1
CHST12	CCDC39	FANCL	CA6	C1orf116	DHX35	CA6	C1orf116	DHX35
CLCN3	CCDC62	FAT2	CACNA1C	C2orf69	DNAH10	CACNA1C	C2orf69	DNAH10
CLIP1	CD46	FCAMR	CACNA1D	C2orf78	DOCK10	CACNA1D	C2orf78	DOCK10
CLU	CDC20	FLJ40288	CACNA2D3	C2orf82	DPP4	CACNA2D3	C2orf82	DPP4
CNKSR2 COQ10B	CDC25C CDHR3	FLJ46066 FPGT-TNNI3K	CBR4	C3orf49 C7orf50	DPYD DQX1	CBR4	C3orf49 C7orf50	DPYD DQX1
CREB3L2	CEP162	GALNT10	CCDC150 CCDC24	CA6	EMX1	CCDC150 CCDC24	CA6	EMX1
CSMD1	CHRNA3	GBA3	CCDC30	CA8	EP300	CCDC30	CA8	EP300
CUL3	CNKSR2	GIGYF2	CCDC39	CACNA1C	ERMAP	CCDC39	CACNA1C	ERMAP
CYP26B1	CNNM2	GPER1	CCNH	CACNA1C-AS4	EXOC6B	CCNH	CACNA1C-AS4	EXOC6B
CYSTM1	COQ10B	GPM6A	CD46	CACNA1C-IT3	FAM57B	CD46	CACNA1C-IT3	FAM57B
DAZL	COX20	GPX5	CDC25C	CACNA1D	FAM83D	CDC25C	CACNA1D	FAM83D
DDX60L	CR1L	GRAMD1B	CENPL	CACYBP	FANCL	CENPL	CACYBP	FANCL
DENR	CREB3L1	GRIA1	CEP170	CBR4	FAT2	CEP170	CBR4	FAT2
DESI2	CSMD1	GRIN2A	CETN3	CCDC150	FCAMR	CETN3	CCDC150	FCAMR EL 140288
DFNA5 DGKD	CTNNA1 CTNND1	HCN1 HECW2	CFAP57 CHDH	CCDC39 CCDC62	FLJ40288 FLJ46066	CFAP57 CHDH	CCDC39 CCDC62	FLJ40288 FLJ46066
DGKD	CTRL	HIRIP3	CHST12	CCDC62 CCT7	FDXP1	CHST12	CCDC62 CCT7	FDXP1
DNAJC18	CTSS	HSPE1-MOB4	CLCN3	CD46	FPGT-TNNI3K	CLCN3	CD46	FPGT-TNNI3K
DNAJC19	CUL3	IMMP2L	CLIP1	CDC20	GALNT10	CLIP1	CDC20	GALNT10
DOCK10	CYP26B1	INO80E	CLU	CDC25C	GALNT15	CLU	CDC25C	GALNT15
DPH3	DDX60L	ITGA9	CMTR2	CDHR3	GBA3	CMTR2	CDHR3	GBA3
DPP4	DESI2	JUND	CNKSR2	CENPL	GIGYF2	CNKSR2	CENPL	GIGYF2
DPYD	DGKD	KCNJ13	COQ10B	CEP162	GPER1	COQ10B	CEP162	GPER1
DPYD-AS1	DGKZ	KDM4A	CREB3L2	CHDH	GPM6A	CREB3L2	CHDH	GPM6A
DUS2 EBNA1BP2	DIS3L2 DNAJC19	LINC01122	CSGALNACT1 CSMD1	CHRNA3 CNKSR2	GPX5 GRAMD1B	CSGALNACT1 CSMD1	CHRNA3 CNKSR2	GPX5 GRAMD1B
EFHD1	DOCK10	LOC100507091	CUL3	CNNM2	GRIA1	CUL3	CNNM2	GRIA1
EIF2B1	DPEP3	LOC606724	CYP26B1	COQ10B	GRIN2A	CYP26B1	COQ10B	GRIN2A
EIF3B	DPH3	LOC613038	CYSTM1	COX20	HCN1	CYSTM1	COX20	HCN1
EP300	DPYD	LOC729987	DARS2	CPEB1	HECW2	DARS2	CPEB1	HECW2
EPB41	DPYD-AS1	LRRIQ3	DAZL	CPEB1-AS1	HIRIP3	DAZL	CPEB1-AS1	HIRIP3
EPC2	DUS2	LSMEM1	DCP1A	CR1L	HSPE1-MOB4	DCP1A	CR1L	HSPE1-MOB4
EPHB1	DYSF	MAD1L1	DCTN1	CREB3L1	HTRA2	DCTN1	CREB3L1	HTRA2
ERI3	EFHD1	MAN2A1	DCTN1-AS1	CSMD1	HYDIN	DCTN1-AS1	CSMD1	HYDIN
ESAM	EIF4E2	MAPK3	DDX60L	CTNNA1	IBTK	DDX60L	CTNNA1	IBTK
ETF1	ELOVL1	MBTPS2	DENR	CTNND1	IMMP2L	DENR	CTNND1	IMMP2L
FAM114A2 FAM13B	ENO1 EPC2	MEF2C MIR137	DESI2 DFNA5	CTRL CTSS	INO80E ITGA9	DESI2 DFNA5	CTRL CTSS	INO80E ITGA9
FAM53C	EPC2 ERI3	MIR137 MIR137HG	DGKD	CUL3	JUND	DGKD	CUL3	JUND
	FAM124B	MIR2682	DGKZ	CYP26B1	KCNJ13	DGKZ	CYP26B1	KCNJ13
FANCL					KDM4A	DNAJC18		

FCHSD1 FER	FAM57B	MIR3188	DNAJC19	DCDC1	KLHL20	DNAJC19	DCDC1	KLHL20
TL 14000C	FANCL	MIR3714	DOCK10	DCDC5	KMT5A	DOCK10	DCDC5	KMT5A
FLJ46066	FLJ46066	MIR4304	DPH3	DCP1A	LINC00461	DPH3	DCP1A	LINC00461
FPGT-TNNI3K	FPGT-TNNI3K	MKL1	DPP4	DCTN1	LINC01122	DPP4	DCTN1	LINC01122
FTCDNL1	FSIP2	MOB4	DPYD	DDHD2	LOC100506023	DPYD	DDHD2	LOC100506023
FXR1	FTCDNL1	MPHOSPH9	DPYD-AS1	DDX60L	LOC100507091	DPYD-AS1	DDX60L	LOC100507091
GALNT10	FXR1	MPP6	DUS2	DESI2	LOC153910	DUS2	DESI2	LOC153910
GALNT15	G3BP1	MYLPF	EBNA1BP2	DGKD	LOC440704	EBNA1BP2	DGKD	LOC440704
GATAD2A	GABBR1	MYO1A	EFHD1	DGKZ	LOC606724	EFHD1	DGKZ	LOC606724
GBF1	GALNT10	NDUFA13	EIF2B1	DIS3L2	LOC613038	EIF2B1	DIS3L2	LOC613038
GFRA3	GATAD2A	NEK1	EIF3B	DNAJC19	LOC729987	EIF3B	DNAJC19	LOC729987
GIGYF2	GFRA3	NFATC3	EMB	DNAJC6	LOXL3	EMB	DNAJC6	LOXL3
GLRA1	GID4	NLGN4X	EMX1	DOCK10	LRRIQ3	EMX1	DOCK10	LRRIQ3
GPM6A	GIGYF2	NMUR2	EP300	DPEP3	LSMEM1	EP300	DPEP3	LSMEM1
GPX5	GLRA1	NRN1L	EPB41	DPH3	MAD1L1	EPB41	DPH3	MAD1L1
GPX6	GPM6A	NUGGC	EPC2	DPYD	MAN2A1	EPC2	DPYD	MAN2A1
GRIA1	GPX5	OGFOD2	EPHB1	DPYD-AS1	MAPK3	EPHB1	DPYD-AS1	MAPK3
GRIN2A	GRIA1	OR1S1	ERI3	DUS2	MBTPS2	ERI3	DUS2	MBTPS2
GTF2H3	GRIN2A	OR1S2	ESAM	DUSP11	MEF2C	ESAM	DUSP11	MEF2C
GTF3C3	GRM3	OR8B3	ETF1	DYSF	MIR137	ETF1	DYSF	MIR137
HCN1	H6PD	OR9Q1	EXOC6B	EFHD1	MIR137HG	EXOC6B	EFHD1	MIR137HG
HDAC3	HARS	PAPPA2	FAM114A2	EGR4	MIR2682	FAM114A2	EGR4	MIR2682
HECW2	HARS2	PDE6D	FAM13B	EIF4E2	MIR29C	FAM13B	EIF4E2	MIR29C
HPF1	HCN1	PITPNM2	FAM53C	ELOVL1	MIR3188	FAM53C	ELOVL1	MIR3188
ISPA9	HECW2	PLCL1	FANCL	EMX1	MIR3714	FANCL	EMX1	MIR3714
HSPD1	HSPA9	PLCL2	FAT2	ENO1	MIR4304	FAT2	ENO1	MIR4304
ISPE1-MOB4	IL19	PLD5	FCHSD1	EPC2	MIR4304 MIR9-2	FCHSD1	EPC2	MIR4304 MIR9-2
K	INPP5D	PLEKHO1	FER	EPG2 EPHA7	MKL1	FER	EPG2 EPHA7	MKL1
n L19	INPP5D IPO11	PLEKHUT PLXNA4	FGFR1	EPHA7 EPN2	MOB1A	FGFR1	EPHA7 EPN2	MOB1A
L19 MMP2L	ITIH1	PPP1R16B	FGFR1 FLJ46066	EPN2 EPN2-IT1	MOB1A MOB4	FGFR1 FLJ46066	EPN2 EPN2-IT1	MOB1A MOB4
NPP5D	ITIH3	PSKH1	FPGT-TNNI3K	ERI3	MPHOSPH9	FPGT-TNNI3K	ERI3	MPHOSPH9
QCE	ITIH4	PTCHD1-AS	FTCDNL1	EXOC6B	MPP6	FTCDNL1	EXOC6B	MPP6
QCF1	KCNH7	PTPRF	FXR1	FAM124B	MYLPF	FXR1	FAM124B	MYLPF
TGA9	KCNJ13	RERE	GAB1	FAM53C	MYO1A	GAB1	FAM53C	MYO1A
TIH3	KDM4A	RFTN2	GALNT10	FAM57B	NAT8	GALNT10	FAM57B	NAT8
TIH4	KMT2E	RILPL1	GALNT15	FANCL	NDUFA13	GALNT15	FANCL	NDUFA13
KCNH7	KMT2E-AS1	RNF220	GATAD2A	FBXO41	NEK1	GATAD2A	FBXO41	NEK1
CNJ13	LAPTM5	SAP30L-AS1	GBF1	FLJ46066	NFATC3	GBF1	FLJ46066	NFATC3
(DM3B	LINC00634	SATB2	GFRA3	FOXP1	NLGN4X	GFRA3	FOXP1	NLGN4X
KDM4A	LINC00698	SBNO1	GIGYF2	FPGT-TNNI3K	NMUR2	GIGYF2	FPGT-TNNI3K	NMUR2
(MT2E	LINC01004	SDCCAG8	GLRA1	FRMD8	NRN1L	GLRA1	FRMD8	NRN1L
(NTC1	LINC01122	SF3B1	GPM6A	FSIP2	NUGGC	GPM6A	FSIP2	NUGGC
INC01122	LOC100129620	SH3RF1	GPR52	FTCDNL1	OGFOD2	GPR52	FTCDNL1	OGFOD2
OC100129620	LOC100652758	SLC4A10	GPX5	FXR1	OPCML	GPX5	FXR1	OPCML
OC100130452	LOC339529	SLC6A9	GPX6	G3BP1	OR1S1	GPX6	G3BP1	OR1S1
OC100130880	LOC339862	SLX1A	GRIA1	GABBR1	OR1S2	GRIA1	GABBR1	OR1S2
OC100506085	LOC606724	SLX1B-SULT1A4	GRIN2A	GALNT10	OR8B3	GRIN2A	GALNT10	OR8B3
OC100507091	LOC613038	SMG1P2	GTF2H3	GALNT15	OR9Q1	GTF2H3	GALNT15	OR9Q1
OC100507140	LOC642423	SMS	GTF3C3	GAS5	PAPPA2	GTF3C3	GAS5	PAPPA2
OC148696	LRP1	SNAP91	GUSBP5	GAS5-AS1	PDE4B	GUSBP5	GAS5-AS1	PDE4B
OC339529	LRRIQ3	SOX2-OT	HCN1	GATAD2A	PDE6D	HCN1	GATAD2A	PDE6D
OC339862	LYPD6B	ST3GAL3	HDAC3	GFRA3		HDAC3	GFRA3	PGM3
OC729987	MAD1L1	STK31	HECW2	GID4		HECW2	GID4	PITPNM2
_RRC43	MAN2A1	TAOK2	HPF1	GIGYF2		HPF1	GIGYF2	PLCL1
_RRIQ3	MARS	TBC1D10B	HSPA9	GLRA1	PLCL2	HSPA9	GLRA1	PLCL2
YPD6	MBTPS2	TBC1D5	HSPD1	GLT8D1	PLOL2 PLD5	HSPD1	GLT8D1	PLD5
/AD1L1	MDK	TCF4	HSPE1-MOB4	GNL3	PLEKHO1	HSPE1-MOB4	GNL3	PLEKHO1
MAN2A1	MED8	TMX2-CTNND1	HYDIN	GPM6A	PLXNA4	HYDIN	GPM6A	PLXNA4
/ARS2	MIR137HG	TNFRSF9	IK	GPR52	PPP1R16B	IK ISBB	GPR52	PPP1R16B
	MIR22	TNNI3K	IL17RB	GPX5	PSKH1	IL17RB	GPX5	PSKH1
				GRIA1	PTCHD1-AS			
/IBTPS2	MIR22HG	TNRC6B	L19			IL19	GRIA1	PTCHD1-AS
MBTPS2 MDK	MIR29C	TNRC6B TOX	IMMP2L	GRIN2A	PTPRF	IL19 IMMP2L	GRIN2A	PTPRF
/IBTPS2 /IDK /IFAP3	MIR29C MIR339	TNRC6B TOX TRANK1	IMMP2L INO80B	GRIN2A GRM3	PTPRF RABGAP1L	IL19 IMMP2L INO80B	GRIN2A GRM3	PTPRF RABGAP1L
MAU2 MBTPS2 MDK MFAP3 MIR137HG	MIR29C MIR339 MIR33B	TNRC6B TOX TRANK1 TSSK6	IMMP2L NO80B INO80B-WBP1	GRIN2A GRM3 H6PD	PTPRF RABGAP1L RC3H1	L19 IMMP2L INO80B INO80B-WBP1	GRIN2A GRM3 H6PD	PTPRF RABGAP1L RC3H1
MBTPS2 MDK MFAP3 MIR137HG MIR2682	MIR29C MIR339 MIR33B MIR4677	TNRC6B TOX TRANK1 TSSK6 TTYH3	MMP2L NO80B NO80B-WBP1 NPP4B	GRIN2A GRM3 H6PD HARS	PTPRF RABGAP1L RC3H1 RERE	L19 MMP2L NO80B NO80B-WBP1 NPP4B	GRIN2A GRM3 H6PD HARS	PTPRF RABGAP1L RC3H1 RERE
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1	MIR29C MIR339 MIR33B MIR4677 MIR4688	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D	GRIN2A GRM3 H6PD HARS HARS2	PTPRF RABGAP1L RC3H1 RERE RFTN2	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D	GRIN2A GRM3 H6PD HARS HARS2	PTPRF RABGAP1L RC3H1 RERE RFTN2
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR3160-2	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE	GRIN2A GRM3 H6PD HARS HARS2 HCN1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE	GRIN2A GRM3 H6PD HARS HARS2 HCN1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR3160-2 MIR3160-2 MIR4688	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2 MOG	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR3160-2 MIR3160-2 MIR4688	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE	GRIN2A GRM3 H6PD HARS HARS2 HCN1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE	GRIN2A GRM3 H6PD HARS HARS2 HCN1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1
IBTPS2 IDK IFAP3 IIR137HG IIR2682 IIR3160-1 IIR3160-2 IIR4688 IOB4	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2 MOG	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220
IBTPS2 IDK IFAP3 IIR137HG IIR2682 IIR3160-1 IIR3160-2 IIR4688 IOB4 IPHOSPH9	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1	L19 MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TGA9	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1
IBTPS2 IDK IFAP3 IFAP3 IFAP3 IR137HG IIR2682 IIR2688 IIR4688 IIR4688 IOB4 IPHOSPH9 IPP6	MIR29C MIR339 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TiH3	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2	L19 MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTH3	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2
IBTPS2 IDK IFAP3 IIR137HG IIR1682 IIR3160-2 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 II	MIR29C MIR339 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2	TNRC6B TOX TRANK1 TTSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SAB01	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HDAC2 HECW2 HIVEP2 HSPA9 HSPA9	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBNO1
IBTPS2 IDK IFAP3 IIR137HG IIR1682 IIR3160-2 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR4688 IIR3160-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3170-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 II	MIR29C MIR339 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8 UGT1A8 UGT1A9	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCF1 TGA9 TTIH3 TTIH4 KCNH7	GRIN2A GRM3 H6PD HARS HARS HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17RB	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAT82 SBNO1 SDCCAG8	L19 MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TIGA9 TIH3 TIH4 KCNH7	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8
IBTPS2 IDK IFAP3 IIR137HG IIR2682 IIR3160-1 IIR3160-2 IIR31	MIR29C MIR339 MIR3677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8 UGT1A9 UGT1A9 UGT1A9 UGT1A9 UGT1A9	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTH4 TTH4 KCNH7 KCNH7 KCNJ3	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L17RB L19	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SBN01 SDCCAG8 SF3B1	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCF QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ13	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SBN01 SDCCAG8 SF3B1
ABTPS2 ADK MFAP3 AIR137HG AIR2682 AIR2682 AIR3682 AIR3688 AIR3688 AIR3688 AIR4	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1 MYO1A	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A4 UGT1A5 UGT1A7 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 UJS2	MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCF1 IGA9 TIH3 TIH4 KCNH7 KCNJ13 KCNJ3B	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17RB IL19 IL34	PTPRF RABGAP1L RC3H1 RER1 REFN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBNO1 SDCCAG8 SF3B1 SFXN5 SH3RF1	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNJ13 KDM3B	GRIN2A GRM3 H6PD HARS HARS2 HON1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34	PTPRF RABGAP1L RC3H1 RER4 REFN2 RFTN2 RLPL1 RNF220 SAP30L-AS1 SAF32 SBNO1 SDCCAG8 SF3B1 SFXN5 SH3RF1
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR3682 MIR360-2 MIR360-2 MIR3688 MOB4 MPHOSPH9 MPOSPH9 MPOSPH9 MPOS MOT MOT ICAN ICK1	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MRAP2 MSL2 MUSTN1 MSL2 MUSTN1 MYO1A NDUFA4L2	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8 UGT1A9 UGT1A9 UGT1A9 UGT1A9 UGT1A9	MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNJ13 KDM3B KDM4A KLHL20	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17 L34 NO800B	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF381 SF385 SHX5 SHX6 SHX6 SLC4A10	L19 MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TIGA9 TIH3 TIH4 KCNH7 KCNH7 KCNH3 KCNH3 KCNH3 KCNH3 KCNH3 KCNH3 KCNH3 KCNH4 KCNH	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 L19 L34 NO80B	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF381 SF381 SF381 SHXP5 SHXP5
IBTPS2 IDK IFAP3 IIR137HG IIR2682 IIR3160-1 IIR3160-1 IIR3160-2 IIR3160-2 IIR3160-2 IIR3688 MOB4 IIR4688 MOB4 IIR4688 MOB4 IIR4688 IIR4688 IIR468 III	MIR29C MIR339 MIR33B MIR4677 MIR4687 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1 MSL2 MUSTN1 MYO1A NDUFA4L2 NEK1 NEK4	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8 UGT1A9 UGT1A9 UGT1A9 UGT1A9 VPS40 UTS2 VPS45 VRK2	MMP2L N080B N080B-WBP1 NPP6D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH7 KCNH3 KDM3A KDM4A KLHL29	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L134 NO808-WBP1 NPP48	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SBN01 SDCCAG8 SF381 SFXN5 SH2C4A10 SLC4A5	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TIH3 TIH4 KCNH7 KCNJ13 KDM4A KDM4A KLHL20 KLHL29	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L134 NO80B-WBP1 NOPP4B	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SHQF11 SLC4A5
MBTP32 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR2682 MIR2682 MIR2688 MOB4 MPHOSPH9 MPHOSPH9 MPHOSPH9 MPHOSPH9 MPTO1 MYOTA MYOTA CAN CAN CAN ICK1 EK1 IFATC3	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR4688 MOG MPL0SPH9 MRL MR482 MUSTN1 MV01A MUSTN1 MV01A NDUFA4L2 NEK1 NEK4 NEK4 NEMP1	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A6 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 UTS2 VPS45 VRK2 WDFY1	MMP2L NO80B NO80B-WBP1 NPP5D QCE QCF1 TGA9 TTH3 TTH4 KCNJ13 KCNJ13 KCNJ13 KCNJ13 KCNJ13 KCNJ13 KLHL29 KLHL29 KMT2E	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL19 IL34 IN080B NO80B-WBP1 NPP4B NPP5D	PTPRF RABGAP1L RC3H1 RERE RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCC-AG8 SF3B1 SH3RF1 SLC4A10 SLC4A5 SLC649	L19 MMP2L NN80B NN80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ13 KDM3B KDM3B KDM4A KLHL20 KLHL29 KMT2E	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 N080B-WBP1 N080B-WBP1 NPP4B NPP5D	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30LAS1 SATB2 SBN01 SDCCAG8 SF3B1 SH3RF1 SLC4A10 SLC6A9
IBTPS2 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3160-1 IIR3160-2 IIR3160-2 IIR3160-2 IIR46888 IIR4688 IIR46888 IIR46888 IIR46888 IIR46888 IIR46888 IIR46888	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR6488 MIR648A2 MOG MPHOSPH9 MPL MPL MRAP2 MSL2 MUSTN1 MVO1A NDUFA4L2 NEK1 NEK4 NEK4 NEK4 NEK4 NEK7 NEATC3	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A6 UGT1A7 UGT1A8 UGT1A9 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 VJEFN3	MMP2L N080B N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH3 KDM4A KLHL20 KLHL20 KUT2E	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 L17RB L178 L19 L34 NO80B NOP48 NPP48 INPP5D	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF3B1 SFXN5 SH2C4A10 SLC4A5 SLC6A9 SLC52	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ13 KCNJ13 KDM3B KDM3B KDM4A KLHL20 KLHL20 KLH29 KMT2E KNTC1	GRIN2A GRM3 H6PD HARS HARS2 HON1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L34 NO80B NO90B-WBP1 NPP4B NPP5D PO11	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILP11 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF3B1 SFXN5 SH2C4A10 SLC4A5 SLC6A9 SLC22
IBTPS2 IDK IFAP3 IIR137HG IIR137HG IIR3160-1 IIR3160-1 IIR3160-2 IIR3	MIR29C MIR339 MIR33B MIR4677 MIR4687 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MRAP2 MRAP2 MSL2 MUSTN1 MSL2 MUSTN1 MYO1A NDUFA4L2 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A9 UNCX USP40 UTS2 VPS45 WDFY1 YJEFN3 ZNF165	MMP2L N080B ND80B-WBP1 NPP5D QCE QCF1 TIGA9 TIH4 KCNH7 KCNH7 KCNH4 KCM4A KLHL20 KLHL29 KMT2E KNTC1 LINC00461	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17R IL34 NO800B-WBP1 NOP4B NPP5D IPO111 TIH1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAF30L-AS1 SAF30L-AS1 SBN01 SDCCAG8 SF381 SFXN5 SLC4A10 SLC4A5 SLC6A9 SLC9C2 SLX1A	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNJ13 KDM4A KLHL20 KLHL29 KMT2E KNTC1 LINC00461	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 L19 L34 NO80B-WBP1 NPP4B NPP5D PO11 TIH1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF381 SFXN5 SLC4A5 SLC6A9 SLC9C2 SLX1A
IBTP52 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR468 IIR47 II	MIR29C MIR339 MIR4839 MIR4677 MIR4688 MIR4688 MOG MPHOSPH9 MPL MR48A2 MUSTN1 MR42 MUSTN1 MV01A NDUFA4L2 NEK1 NEK4 NEK4 NEK4 NEFATC3 NGEF NLGN4X	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USF2 VPS45 VRK2 WDFY1 YJEFN3 ZNF165 ZNF48	MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNJ13 KDM4A KLHL29 KMT2E KNTC1 LINC00461 LINC01122	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17 IL34 NO80B NO80B-WBP1 NPP5D PO11 TIH1 TIH3	PTPRF RABGAP1L RC3H1 RERE RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF3B1 SH3RF1 SLC4A10 SLC4A5 SLC6A9 SLXIA SLX1A SLX1A SLX1A	L19 MMP2L NO80B-WBP1 NO80B-WBP1 NPP4B NPP5D QCF QCF1 TGA9 TH3 TH4 KCNJ13 KCNJ13 KCNJ13 KDM3B KDM4A KLHL20 KLHL20 KLH29 KMT2E KNTC1 LINC00461 LINC01122	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 N0806-WBP1 N0808-WBP1 NPP4B NPP5D PO11 TIH3	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30LAS1 SATB2 SBN01 SDCCAG8 SF3B1 SH3RF1 SLC4A10 SLC4A5 SLC6A9 SL2622 SLX1A SLX1A SLX1B-SULT1A4
IBTPS2 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3160-1 IIR3160-2 IIR3160-2 IIR3160-2 IIR46888 IIR46888 IIR46888 IIR46888 IIR4688 IIR46888 II	MIR29C MIR339 MIR4339 MIR4677 MIR4687 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MSL2 MUSTN1 MVC1A NOUFA4L2 NEK1 NEK1 NEK4 NEK1 NEK4 NEFATC3 NGEF NLGN4X NT5C2	TNRC6B TOX TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A7 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 UTS2 VPS45 VRK2 WDFY1 YJEFN3 ZNF165 ZNF691	MMP2L N080B N080B-WBP1 N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH7 KCNH3 KDM4A KLHL20 KLHL20 KMT21 INC00461 INC01122 OC0129620	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 IL17RB IL34 NO80B-WBP1 NPP4B NPP5D PO11 TIH1 TIH3	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILP11 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SLC4A10 SLC6A9 SLC345	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNJ7 KCNJ7 KCNJ3 KDM3B KDM4A KLHL20 KLH29 KMT2E KNTC1 LINC00461 LINC01122 LOC100129620	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B-WBP1 NPPAB NPPPD PP011 THH1 TIH3 TIH4	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SH2C4A10 SLC6A9 SLC9C2 SLX1A SLX1B-SULT1A4 SMG1P2
IBTP52 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR4688 NOB4 IIR4688 IIR4688 IIR4688 IVOTA IVOTA IVOTA IVOTA ICAN	MIR29C MIR339 MIR338 MIR4677 MIR4688 M	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 UTS2 VPS45 VRK2 WDFY1 YJEFN3 ZNF465 ZNF48 ZNF691 ZNF804A	MMP2L N080B N080B-WBP1 NPP4B NPP5D QCE QCF1 TTGA9 TTH3 TTH4 KCNH7 KCNH7 KCNH3 KDM4A KLHL29 KMT2E KNTC1 LINC00461 LINC00461 COC100129620 OC100130452	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 L19 L34 NN800B-WBP1 NN800B-WBP1 NPP4B INPP5D IPO111 TIH1 TIH3 TIH4 JAK1	PTPRF RABGAP1L RC3H1 RER1 REFN2 RILPL1 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF3B1 SLC4A5 SLC6A9 SLX1A SLX1A SLX1B-SULT1A4 SMS	L19 MMP2L NN808 NN808-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ3 KDM38 KDM4A KLHL29 KMT2E KNTC1 LINC00461 LINC01122 LOC100130452	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 L19 L34 NO806B-WBP1 NOPP4B NPP5D PO111 TIH4 JAK1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SLC4AS1 SLC4AS1 SLC4A5 SLC6A9 SLX18-SULT1A4 SMS
IBTPS2 IDK IFAP3 IIFN37HG IIFN37HG IIFN37HG IIFN360-2 IIFN360-2 IIFN360-2 IIFN4688 IIFN60-2 IIFN4688 IIFN60-2 IIFN4688 IIFN4688 IIFN4688 IIFN468 IIFN468 IIFN47 IIF	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR6488 MIR648A2 MOG MPHOSPH9 MPL MPL MRAP2 MSL2 MUSTN1 MV01A MV01A NDUFA4L2 NEK1 NEK4 NEK4 NEK4 NEFATC3 NGEF NLGN4X NT5C2 NT5M NYAP2	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N080B N080B-WBP1 N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCN13 KDM4A KLHL20 KLHL20 KIT2E INC00461 INC01122 LOC100120620 LOC100130452 OC100130880	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 L17RB L14 N0808 NN0808-WBP1 N0808-WBP1 PP748 IP011 TTIH1 TTIH3 TTIH4 JAK1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30LAS1 SAP30LAS1 SAP30LAS1 SBN01 SDCCAG8 SF3B1 SFXN5 SLC4A10 SLC4A5 SLC4A5 SLC4A5 SLX18-SULT1A4 SMAP1 SMAP31	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTH4 KCNH7 KCNH7 KCNH7 KCNH7 KCNH7 KCNJ3 KDM38 KDM4A KLHL20 KLHL20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 CC100129620 LOC100130880	GRIN2A GRM3 H6PD HARS HARSS HOAC2 HECW2 HIVEP2 HSPA9 L17RB L178 L19 L34 NO80B NO90B-WBP1 NPP4B PO11 TTH4 JAK1 KAT5	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILP11 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF3B1 SFXN5 SH2C4A10 SLC4A5 SLC4A5 SLC4A5 SLX18-SULT1A4 SMG1P2 SMS SNAP91
IBTP52 IDK IFAP3 IRP37HG IIR137HG IIR2682 IIR3160-1 IIR3688 IIR3688 IIR3688 IIR4688 IOB4 IIR4688 IIIR4688 IIIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIR4688 IIIIIR4688 IIIIIR4688 IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	MIR29C MIR339 MIR338 MIR4677 MIR4687 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MRAP2 MSL2 MUSTN1 MSL2 MUSTN1 MY01A NDUFA4L2 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1 NEK1	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 UTS2 VPS45 VRK2 WDFY1 YJEFN3 ZNF465 ZNF48 ZNF691 ZNF804A	MMP2L N080B NN20B-WBP1 NPP4B NPP5D QCE QCF1 TIH3 TIH4 KCNH7 KCNH7 KCNH3 KDM4A KLHL29 KMT2E KNTC1 LINC00461 LINC01122 OC100130452 OC100130452 OC1000306023	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17RB IL34 NO80B NO80B-WBP1 NNPP4B INPP5D PO11 TTH1 TTH3 JAK1 KAT5 KCNH7	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF381 SFXN5 SH3RF1 SLC4A5 SLC6A9 SLX1A SNAP91 SOX2-OT	L19 MMP2L NN080B NN080B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TTIH3 THH4 KCNJ13 KDM4A KLHL29 KMT2E KNTC1 LINC00461 LINC00461 LINC0129620 LOC100130452 LOC10030880 LOC10030880	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B NO80BWBP1 NPP4B INPP5D PO11 TTH3 TH4 JAK1 KAT5 KCNH7	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAF30L-AS1 SAF30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF381 SFXN5 SLC4A10 SLC4A5 SLC6A9 SLC4A5 SLX1A SMG1P2 SMAP91 SOX2-OT
IBTP52 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR360-2 IIR360-2 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR469 IIR47	MIR29C MIR339 MIR33B MIR4677 MIR4688 MIR688 MIR688 MIR688 MR0G MOG MR497 MSL2 MUSTN1 MY01A NDUFA4L2 NEK1 NEK4 NEFF NGEF LGN4X NT5C2 NT5M NYAP2 OGFOD2 P2RX3	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N080B NN080B-WBP1 NPP5D QCE QCF1 TTGA9 TTIH3 TTIH4 KCNH7 KCNH7 KCNH7 KCNH7 KCNH7 KCNH7 LINC0461 LINC00461 LINC00461 LINC01122 OC100130880 LOC100130880 LOC100506023 OC100506085	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17 IL34 NO80B-WBP1 NPP48 NPP5D IPO111 TIH3 TIH4 JAK1 KAT5 KCNH7 KCNJ3	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SLCAAD SUCCAG8 SLCAAD SLC4A10 SLC4A5 SLX1A SLX1A SUX1A SMS SNAP91 SOX2-OT ST3GAL3	L19 MMP2L NN80B NN80B-WBP1 NPP4B NPP5D QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ13 KDM3B KDM4A KLHL20 KLH20 KLH20 KLH29 KMT2E KNTC1 LINC00461 LINC01122 LOC100129620 OC100130452 OC100130880 LOC100506023 LOC100506023	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L178 L19 IS4 NO806 NO806 NO806 HPP4B NPP4B NPP5D PO11 TTH3 TTH4 JAK1 KAT5 KCNH7 KCN13	PTPRF RABGAP1L RAC3H1 RER2 RFTN2 RIFP120 SAP30L-AS1 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SF381 SLC4A10 SLC4A5 SLX18-SUL71A4 SMS SMAP91 SOX2-OT ST3GAL3
IBTPS2 IDK IFAP3 IIR137HG IIR137HG IIR2882 IIR3160-1 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIR4688 IIG41 IIG41 IIG41 IIG41 IIG41 IIG41 IIG41 IIG41 IIG41 IIG52 IIUBP1 IYAP2 IYAP2 IYZ82 IIG82 IIG4 IIG41	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MPL MRAP2 MSL2 MUSTN1 MV01A NOUFA4L2 NEK1 NUFA12 NEK4 NEMP1 NEK4 NEFATC3 NGEF NLGN4X NT5C2 NT5M NYAP2 OGFOD2 P2RX3 PALLD	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N080B N080B-WBP1 N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH7 KCNH3 KDM4A KLHL29 KMTC1 INC00461 INC00429620 OC1001296201 OC100506023 COC100506023 COC100507091	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 IL17RB IL34 IN080B NNPP4B NPP5D IP011 TIH1 TIH3 TIH4 JAK1 KAT5 KCNH7 KCDM4A	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATS0L-AS1 SATS0L-AS1 SAT30L-AS1 SAT81 SBNO1 SDCCAG8 SF381 SFXN5 SLC4A5 SLC6A9 SLX18-SULT1A4 SMAP91 SOX2-OT ST3GAL3 STAB2	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNH7 KCNH7 KCNJ3 KDM3B KDM4A KLHL20 KLH29 KMT2E KNTC1 LINC00461 LINC01122 LOC100129620 LOC100506023 LOC100506025 LOC100507091	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B-WBP1 NPPAB NPPPBD PO11 TTH4 JAK1 KAT5 KCNH7 KCMMAA	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SLC4A10 SLC4A5 SLC4A5 SLC345 SLC345 SLC345 SLC345 SLC345 SLC345 SLC345 SLC3410 SLC342 SLC343 SLC343 SLC342 SMG1P2 SMAP91 S0X2-OT ST3GAL3 STAB2
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IBTP52 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3160-2 IIR316	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MPL MRAP2 MSL2 MUSTN1 MV01A NOUFA4L2 NEK1 NUFA12 NEK4 NEMP1 NEK4 NEFATC3 NGEF NLGN4X NT5C2 NT5M NYAP2 OGFOD2 P2RX3 PALLD	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N080B N080B-WBP1 N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH7 KCNH3 KDM4A KLHL29 KMTC1 INC00461 INC00429620 OC1001296201 OC100506023 COC100506023 COC100507091	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 L17RB L17R L178 N0808 N0P48 NPP5D PO11 TTH3 TTH4 JAK1 KAT5 KCNH7 KCM13 KDM4A KLH29	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATS0L-AS1 SATS0L-AS1 SAT30L-AS1 SAT81 SBNO1 SDCCAG8 SF381 SFXN5 SLC4A5 SLC6A9 SLX18-SULT1A4 SMAP91 SOX2-OT ST3GAL3 STAB2	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNH7 KCNH7 KCNJ3 KDM3B KDM4A KLHL20 KLH29 KMT2E KNTC1 LINC00461 LINC01122 LOC100129620 LOC100506023 LOC100506025 LOC100507091	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B-WBP1 NPPAB NPPPBD PO11 TTH4 JAK1 KAT5 KCNH7 KCMMAA	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SLC4A10 SLC4A5 SLC4A5 SLC4A5 SLC345 SLC4A5 SLC345 SMG1P2 SMAP91 S0X2-OT ST3GAL3 STAB2
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IBTP52 IDK IFAP3 IDK IFAP3 IIR137HG IIR2682 IIR3160-1 IIR2682 IIR3160-2 IIR3160-2 IIR3160-2 IIR3160-2 IIR3688 IIR3688 IIR3688 IIR4688 IIIR4688 IIIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIIR4688 IIIIR4688 IIIR4688 IIIR4688 IIIR4688 IIIR46	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1 MVO1A MVO1A MVO1A NDUFA4L2 NEK1 NEK4 NEK4 NEK4 NEK4 NEK4 NEK4 NEFATC3 NGEF NLGN4X NT5C2 NT5M NYAP2 OGFOD2 PARK7 PALLD PARK7 PBRM1	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N0808 N0808-WBP1 N0808-WBP1 NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNH3 KDM38 KUHL20 KLHL20 KLHL20 CC100129620 OC100306023 OC100506085 OC100506085 OC100507140 OC100507140	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 L17RB L17R L178 N0808 N0P48 NPP5D PO11 TTH3 TTH4 JAK1 KAT5 KCNH7 KCM13 KDM4A KLH29	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SUPCAG8 SF381 SFXN5 SH3RF1 SLC4A10 SLC4A5 SNAP91 SOX2-OT STA82 STA82 STA82	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNJ13 KDM3B KDM4A KLHL20 KLHL20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 CC100129620 OC100130452 LOC100130880 OC10050085 OC10050085 OC10050091 LOC100507140 LOC14896	GRIN2A GRM3 H6PD HARS HARSS HOAC2 HECW2 HECW2 HIVEP2 HSPA9 L17RB L178 L34 NO808-WBP1 NO908-WBP1 PP48 NPP48 TITH3 TIH4 JAK1 KAT5 KCNH7 KCNH7 KCM4A KUH29 KMT2E	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILP11 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF3B1 SFXN5 SH3RF1 SLC4A10 SLC4A5 SLC6A9 SLX16-SULT1A4 SMG1P2 SMS SNAP91 SOX2-OT ST4A2 ST43 TAOK2
MBTP52 MDK MFAP3 MDK MFAP3 MDK MFAP3 MIR137HG MIR2682 MIR3160-1 MIR2682 MIR2682 MIR2682 MIR269 MOSA	MIR29C MIR339 MIR4877 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1 MSL2 MUSTN1 MYO1A NOUFA4L2 NEK1 NEK1 NEK1 NEK1 NEFATC3 NGEF NLGN4X NT5C2 NT5M NYAP2 OGFOD2 P2RX3 PALLD PARK7 PBRM1 PCCB	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N0808 N0808-WBP1 N0808-WBP1 NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNJ3 KDM38 KDM4A KLHL20 KLHL20 CC100129620 OC10006085 OC1000506035 OC100506035 OC100506035 OC100507140 LOC148696 LOC339629	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO800B-WBP1 NPP4B NPP5D PO11 TTH1 JAK1 KAT5 KCNH7 KCNH7 KCDM4A KLH29 KMT2E KMT2E	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SLARA SBNO1 SDCCAG8 SF381 SFXN5 SH3RF1 SLC4A10 SLC4A5 SLC6A9 SLX1A SLTB-SULT1A4 SMG1P2 SMS SNAP91 SOX2-OT STAB2 STK31 TAOK2 TBC1D10B	L19 MMP2L NN80B NN80BWB1 NNP84B NPP4B NPP5D QCF1 TGA9 TIH3 TH4 KCNH7 KCNH7 KCNH7 KCN13 KDM4A KLHL20 KLHL20 KLTC1 INC00461 INC01012620 OC100130680 JOC100506023 OC100507091 OC100507091 OC148696 JOC339529 OC339529	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 N080B-WBP1 NPP4B NPP5D PP011 TIH1 TIH4 JAK1 KAT5 KCNH7 KCNH4 KLH29 KMT2E-AS1	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SH24A10 SLC4A5 SLC6A9 SLC92 SLX1A SLC92 SMG1P2 SMS SNAP91 SOX2-0T STAB2 STK31 TAOK2 TEC1D10B
IBTPS2 IDK IFAP3 IDK IFAP3 IIR137HG IIR137HG IIR1360-1 IIR3160-1 IIR3160-1 IIR3160-2 IIR	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MSL2 MSTN1 MY01A NOUFA4L2 NEK1 NUFA12 NEK4 NEK4 NEK4 NEK4 NEK4 NEK4 NEK4 NEK4	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L NN080B NN080B-WBP1 NN080B-WBP1 NNPP5D QCE QCF1 TGA9 TTIH4 KCNH7 KCNH7 KCN13 KDM4A KLHL29 KMTC1 LINC00461 LINC01122 OC100506023 OC100506085 OC100507140 OC148696 OC339862 OC34962	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 IL17RB IL17R IL178 IL178 IP94 NO808-WBP1 NPP48 NPP5D IP011 TIH3 TIH4 JAK1 KAT5 KCNH7 KCNJ13 KDM4A KLH29 KMT2E KMT2E KMT2E LAPTM5 LEPR	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SATS0L-AS1 SAP30L-AS1 SFXN5 SHARF1 SUC4A5 SUC4A5	L19 MMP2L NO80B NO80B-WBP1 NPP4B NPP5D QCE QCF1 TGA9 TIH3 TIH4 KCNH7 KCNH7 KCNH7 KCN13 KDM38 KDM4A KLHL20 KLH20 KLH20 KLH20 KLH20 KLH20 KLH20 CC100129620 LOC100129620 LOC10050091 LOC10050091 LOC100507140 COC148696 LOC139862 LOC339862 LOC339862 LOC339862 LOC339862 LOC339862 LOC339862 LOC339862 LOC440704	GRIN2A GRM3 H6PD HARS HARSS HCN1 HDAC2 HECW2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B NOPP4B NPP5D PC011 TTH1 JAK1 KAT5 KCNH7 KCNH4 KMT2E KMT2E KMT2E LAPTM5 LEPR	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAT81L SAT91LAS1 SAT91LAS1 SAT930L-AS1 SAP30L-AS1 SFXBS SH3RF1 SUC4A5 SLC4A5 SNAP91 SOX2-OT STAB2 STAB2 </td
IBTP52 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR3682 IIR3160-2 IIR3160-2 IIR3160-2 IIR3688 IIR3688 IIR46888 IIR4688 IIR46888 IIR4688 IIR46888 IIR46888 IIR46888 IIR46888 IIR46888 IIR46888 IIR4688	MIR29C MIR339 MIR339 MIR339 MIR339 MIR4677 MIR4687 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MUTA NDUFA4L2 NEK4 NEK1 NEK4 NEK4 NEK7 PALLD PARK7 PBRM1 PCCB PCDHA10 PCDHA11	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L NN080B NN080B-WBP1 NNPP6D QCE QCF1 TIGA9 TIH4 KCNH7 KCNH7 KCNH7 KCNH7 KCNH13 KDM4A KLHL29 KMT2E KNTC1 LINC00461 DC100129620 OC100130452 OC100506053 OC100506023 OC100506023 OC1398629 OC339862 OC440704 OC729887	GRIN2A GRM3 H6PD HARS HARS1 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17R IL178 IN080B NO80B-WBP1 INPP4B INPP5D IPO11 TTH1 TH4 JAK1 KAT5 KCNH7 KCNH7 KMT2E-AS1 LAPTM5 LEPR LINC00634	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SH3RF1 SLC4A5 SLC6A9 SLX1A SNAP91 SOX2-OT STAB2 STK31 TAC42 TBC1D10B TBC1D10B TBC44 TMEM161B-AS1	L19 MMP2L NN808 NN808-WBP1 NNPP4B NPP5D QCE QCF1 TTGA9 TTIH3 TTH4 KCNH7 KCNH7 KCNH7 KCN13 KDM4A KLHL29 KNTC1 LINC00461 LINC00461 LINC00461 COT100506085 LOC100506085 LOC100507140 OC3398529 OC339862 LOC400704 OC729887	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B NO80BWP1 NPP4B INPP5D PO11 TTH4 JAK1 KAT5 KCNH7 KOMAA KMT2E-KS1 APTM6 LEPR LINC00634	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SHS SUC4A68 SFXN5 SLC4A410 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC42 SMS SNAP91 SOX2-0T STK31 TA0K2 TBC1D10B TEC1D5 TCF4 TMEM161B-AS1
IBTP52 IDK IFAP3 IDK IFAP3 IIR137HG IIR137HG IIR2682 IIR3160-1 IIR2682 IIR3160-2 IIR3160-2 IIR3160-2 IIR360-2 IIR360-2 IIR360-2 IIR360-2 IIR360-2 IIR360-2 IIR460-2 II	MIR29C MIR339 MIR4877 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MSL2 MUSTN1 MVO1A MVO1A MVO1A MVO1A MVO1A NDUFA4L2 MUSTN1 NEK1 NEK4 NEK4 NEK4 NEK4 NEK4 NEK4 NEF7 NGEF NLGN4X NT5C2 NT5M NYAP2 OGFOD2 PARK7 P2RX3 PALLD PARK7 PBRM1 PCDHA1 PCDHA11 PCDHA12	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L N080B N080B-WBP1 N080B-WBP1 NPP5D QCE QCF1 TGA9 TTIH3 TTIH4 KCNH7 KCNH3 KDM3B KDM4A KLHL20 KLHL20 CC100129620 OC100506085 OC100506085 OC100506085 OC100506085 OC339862 OC440704 OC730159	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 ISPA9 L17RB L14 N0808 IN0808-WBP1 IN0808-WBP1 IPP48 IPO11 TTIH3 THH4 JAK1 KAT5 KCNH7 KCNH73 KDM4A KMT2E LAPTM5 LEPR LINC00634 LINC00638	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SAP30L-AS1 SBNO1 SDCCAG8 SF3B1 SFXN5 SH3RF1 SLC4A10 SLC4A5 SLC6A9 SLX16 SUC42 SMG1P2 SMS SNAP91 SOX2-OT ST36AL3 STAB2 STK31 TAOK2 TEC1D05 TCF4 TMEM161B-AS1	L19 MMP2L N080B N080B-WBP1 NPP4B NPP5D QCF1 TGA9 TIH4 KCNH7 KCNJ13 KDM4A KLHL20 KLHL20 KLHL20 CO100129620 COC100129620 COC100506085 COC100506085 COC100507091 COC339529 COC339529 COC339529 COC339529 COC339529 COC730159	GRIN2A GRM3 H6PD HARS HARSS HOAC2 HECW2 HIVEP2 HSPA9 L17RB L178 L19 L34 NO806 NO806B NO906PWBP1 PP4B PO11 TTH4 JAK1 KAT5 KCNH7 KCN13 KDMAA KMT2E LAPTM5 EPR LINC00698	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILP11 RNF220 SAP30L-AS1 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF3B1 SFXN5 SH3RF1 SLC4A10 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLX18-SULT1A4 SMG1P2 SMS SNAP91 SOX2-OT ST3GAL3 STAB2 STK31 TAOK2 TEC1D10B TCF4 TME161B-AS11 TMX2-CTNND1
MBTPS2 MDK MFAP3 MIR137HG MIR2682 MIR3160-1	MIR29C MIR339 MIR339 MIR339 MIR338 MIR4677 MIR4688 MIR548A2 MOG MPHOSPH9 MPL MRAP2 MUTA NDUFA4L2 NEK4 NEK1 NEK4 NEK4 NEK7 PALLD PARK7 PBRM1 PCCB PCDHA10 PCDHA10	TNRC6B TOX TRANK1 TSSK6 TTYH3 UGT1A10 UGT1A4 UGT1A5 UGT1A6 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A8 UGT1A9 UNCX USP40 VFS45 VRK2 WDFY1 YJEFN3 ZNF691 ZNF691A ZNF64A ZSCAN16-AS1	MMP2L NN080B NN080B-WBP1 NNPP6D QCE QCF1 TIGA9 TIH4 KCNH7 KCNH7 KCNH7 KCNH7 KCNH13 KDM4A KLHL29 KMT2E KNTC1 LINC00461 DC100129620 OC100130452 OC100506053 OC100506023 OC100506023 OC1398629 OC339862 OC440704 OC729887	GRIN2A GRM3 H6PD HARS HARS1 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 IL17R IL17 IN080B NO80B-WBP1 INPP4B INPP5D IPO11 TTH1 TH44 JAK1 KAT5 KCNH7 KCNH7 KMT2E-AS1 LAPTM5 LEPR LINC00634	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SAP30L-AS1 SATB2 SBN01 SDCCAG8 SF381 SFXN5 SH3RF1 SLC4A5 SLC6A9 SLX1A SNAP91 SOX2-0T STGAL3 STK31 TAC105 STK31 TAC105 TGC105 TCF4 TMEMf61B-AS1	L19 MMP2L NN808 NN808-WBP1 NNPP4B NPP5D QCE QCF1 TTGA9 TTIH3 TTH4 KCNH7 KCNH7 KCNH7 KCN13 KDM4A KLHL29 KNTC1 LINC00461 LINC00461 LINC00461 COT100506085 LOC100506085 LOC100507140 OC3398529 OC339862 LOC400704 OC729887	GRIN2A GRM3 H6PD HARS HARS2 HCN1 HDAC2 HECW2 HIVEP2 HSPA9 L17RB L19 L34 NO80B NO80BWP1 NPP4B INPP5D PO11 TTH4 JAK1 KAT5 KCNH7 KOMAA KMT2E-KS1 APTM6 LEPR LINC00634	PTPRF RABGAP1L RC3H1 RERE RFTN2 RILPL1 RNF220 SAP30L-AS1 SHS SUC4A68 SFXN5 SLC4A410 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC4A5 SLC42 SMS SNAP91 SOX2-0T STK31 TA0K2 TBC1D10B TEC1D5 TCF4 TMEM161B-AS1

PCDHA13	PCDHA4		M1AP	LOC100506023	тох	M1AP	LOC100506023	тох
PCDHA2	PCDHA5		MAD1L1	LOC100652758	TRANK1	MAD1L1	LOC100652758	TRANK1
PCDHA3	PCDHA6		MAN2A1	LOC338963	TSSK6	MAN2A1	LOC338963	TSSK6
PCDHA4	PCDHA7		MARS2	LOC339529	TTYH3	MARS2	LOC339529	TTYH3
PCDHA5	PCDHA8		MAU2	LOC339862	UGT1A10	MAU2	LOC339862	UGT1A10
PCDHA6	PCDHA9		MBTPS2	LOC440704	UGT1A4	MBTPS2	LOC440704	UGT1A5
PCDHA7	PCDHAC1		MDK	LOC606724	UGT1A5	MDK	LOC606724	UGT1A8
PCDHA8	PCDHAC2		MEF2C	LOC613038	UGT1A6	MEF2C	LOC613038	UNCX
PCDHA9	PCDHGA1		MFAP3	LOC642423	UGT1A7	MFAP3	LOC642423	USP40
PCDHAC1	PCDHGA10		MIR137HG	LRP1	UGT1A8	MIR137HG	LRP1	UTS2
PCDHAC2	PCDHGA11		MIR2682	LRRIQ3	UGT1A9	MIR2682	LRRIQ3	VPS45
PCDHGA1	PCDHGA12		MIR3160-1	LYPD6B	UNCX	MIR3160-1	LYPD6B	VRK2
PCDHGA10	PCDHGA2		MIR3160-2	MAD1L1	USP40	MIR3160-2	MAD1L1	WDFY1
PCDHGA11	PCDHGA3		MIR4688	MAN2A1	UTS2	MIR4688	MAN2A1	YJEFN3
PCDHGA12	PCDHGA4		MOB4	MAP3K11	VPS45	MOB4	MAP3K11	ZNF165
PCDHGA2	PCDHGA5		MPHOSPH9	MARS	VRK2	MPHOSPH9	MARS	ZNF48
PCDHGA3	PCDHGA6		MPP6	MBTPS2	WDFY1	MPP6	MBTPS2	ZNF691
PCDHGA4	PCDHGA7		MSRA	MDK	YJEFN3	MSRA	MDK	ZNF804A
PCDHGA5	PCDHGA8		MUSTN1	MED8	ZNF165	MUSTN1	MED8	ZSCAN16-AS1
PCDHGA6	PCDHGA9		MYO1A	MEF2C	ZNF48	MYO1A	MEF2C	ZSWIM6
PCDHGA7	PCDHGB1		MYOT	MIR137HG	ZNF691	MYOT	MIR137HG	
								_
PCDHGA8	PCDHGB2		NAT8	MIR22	ZNF804A	NAT8	MIR22	
PCDHGA9	PCDHGB3		NCAN	MIR22HG	ZSCAN16-AS1	NCAN	MIR22HG	
PCDHGB1	PCDHGB4		NCK1	MIR29C	ZSWIM6	NCK1	MIR29C	
PCDHGB2	PCDHGB5	1	NEK1	MIR339	1	NEK1	MIR339	1
		1			#			
PCDHGB3	PCDHGB6		NFATC3	MIR33B	l	NFATC3	MIR33B	
PCDHGB4	PCDHGB7		NISCH	MIR4677	I	NISCH	MIR4677	
PCDHGB5	PCDHGC3		NLGN4X	MIR4688		NLGN4X	MIR4688	
PCDHGB6	PCGF6	1	NME5	MIR548A2	1	NME5	MIR548A2	1
PCDHGB7	PDE4D	1	NMUR2		#	NMUR2	MIR548AI	
				MIR548AI	H			
PCGF6	PDE6D		NOSIP	MOG		NOSIP	MOG	-
PDE4D	PEMT		NPY6R	MOGS	U	NPY6R	MOGS	
PER3	PEX5L		NT5C2	MPHOSPH9		NT5C2	MPHOSPH9	
PEX5L	PFKFB2	1	NTM	MPL	1	NTM	MPL	1
		1		MRAP2	#			
PGAP1	PGAP1		NUBP1			NUBP1	MRAP2	-
PHEX	PGM3		NYAP2	MRPS14	I	NYAP2	MRPS14	
PHF21A	PHEX		OPCML	MSL2		OPCML	MSL2	
PIH1D1	PHF21A	Ĩ	OR2B2	MSRA	ii	OR2B2	MSRA	1
PITPNM2	PITPNM2	1			H			
			ORC5	MUSTN1	l	ORC5	MUSTN1	
PKD2L2	PLCB2		OTUD7B	MYO15A	ļ	OTUD7B	MYO15A	
PLCL1	PLCL1		OXNAD1	MYO1A		OXNAD1	MYO1A	
PLCL2	PLCL2		PAIP2B	NAT8		PAIP2B	NAT8	1
PLD5	PLD5		PALLD	NDUFA4L2		PALLD	NDUFA4L2	
		1			#			
PLEKHO1	PLEKHO1		PARP8	NEK1	l	PARP8	NEK1	
PLPPR5	PLPPR5		PCCB	NEK4		PCCB	NEK4	
PLXNA2	PLXNA2		PCDHA1	NEMP1		PCDHA1	NEMP1	
PODXL	POLDIP3	Ĩ	PCDHA10	NFATC3	ii	PCDHAC1	NFATC3	1
PPARGC1A	PPARGC1A	1	PCDHA11	NGEF	1	PCDHGA2	NGEF	1
PPIH	PPP2R3A		PCDHA12	NLGN4X		PCGF6	NLGN4X	
PPP1CC	PRUNE1		PCDHA13	NT5C2		PDE4B	NT5C2	
PPP2R3A	PSKH1		PCDHA2	NT5M		PDE4D	NT5M	
PSMD14	PSMB10		PCDHA3	NTM		PDGFRL	NTM	1
PTCHD1-AS	PSMG3		PCDHA4	NYAP2		PER3	NYAP2	-
FICHDI-AS								
PTPRF	PSMG3-AS1		PCDHA5	OGFOD2		PEX5L	OGFOD2	
RAD54L2	PTBP2		PCDHA6	OPCML		PGAP1	OPCML	
RANBP10	PTCHD1-AS		PCDHA7	OXNAD1		PHEX	OXNAD1	
RANGAP1	PTPRF		PCDHA8	P2RX3		PHF21A	P2RX3	1
RASAL2	R3HDM2		PCDHA9	PALLD		PIH1D1	PALLD	
REEP2	RAI1		PCDHAC1	PARK7		PITPNM2	PARK7	
RELL2	RASAL2		PCDHAC2	PBRM1		PKD2L2	PBRM1	
RERE	RERE	1	PCDHGA1	PCCB	1	PLCL1	PCCB	1
RFTN1	RFTN1	1	PCDHGA10	PCDHA1	1	PLCL2	PCDHA1	1
							PCDHAT PCDHAC1	
RFTN2	RFTN2	l	PCDHGA11	PCDHA10	 	PLD5		
RILPL2	RNF220		PCDHGA12	PCDHA11		PLEKHO1	PCDHGA10	
RPRD2	RPS6KA3		PCDHGA2	PCDHA12		PLPPR5	PCGF6	
RPS6KA3	RRP7BP	Ĩ	PCDHGA3	PCDHA13	ii	PLXNA2	PCNX3	1
	RTN4RL1	1	PCDHGA3	PCDHA13 PCDHA2	H	POC1A	PDE4B	
RSRC2								
SAP30L-AS1	RTN4RL2		PCDHGA5	PCDHA3		PODXL	PDE4D	-
SATB1	RWDD2A		PCDHGA6	PCDHA4	I	PPARGC1A	PDE6D	
SATB2	SAP30L-AS1		PCDHGA7	PCDHA5		PPIH	PEMT	
SBNO1	SATB2	Ĩ	PCDHGA8	PCDHA6	ii	PPM1L	PEX5L	1
SCG2	SBN01	1	PCDHGA9	PCDHA7	#	PPP1CC	PFKFB2	
					H			
SDCCAG8	SDCCAG8		PCDHGB1	PCDHA8	 	PPP2R2A	PGAP1	1
SEC16B	SEMA3G		PCDHGB2	PCDHA9		PPP2R3A	PGM3	
SF3B1	SEMA6C		PCDHGB3	PCDHAC1		PRDX6	PHEX	
GO2	SERHL2		PCDHGB4	PCDHAC2		PSD3	PHF21A	1
	SERPINE2	1	PCDHGB5	PCDHGA1	1	PSMD14	PITPNM2	1
SH3RF1	SERPINE2 SERPINF1							
			PCDHGB6	PCDHGA10	H	PTCHD1-AS	PLCB2	
SLC17A7			PCDHGB7	PCDHGA11	ļ	PTPRF	PLCL1	
SLC17A7 SLC25A42	SF3B1					DADOADU	PLCL2	
LC17A7 LC25A42 LC2A5			PCGF6	PCDHGA12		RABGAP1L	PLULZ	
LC17A7 LC25A42 LC2A5	SF3B1 SFMBT1		PCGF6 PDE4B					
LC17A7 LC25A42 LC2A5 LC35G2	SF3B1 SFMBT1 SFXN2		PDE4B	PCDHGA2		RAD54L2	PLD5	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8	SF3B1 SFMBT1 SFXN2 SH3GL3		PDE4B PDE4D	PCDHGA2 PCDHGA3		RAD54L2 RANBP10	PLD5 PLEKHO1	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8 SLC4A10	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A		PDE4B PDE4D PDGFRL	PCDHGA2 PCDHGA3 PCDHGA4		RAD54L2 RANBP10 RANGAP1	PLD5 PLEKHO1 PLPPR5	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8 SLC39A8 SLC4A10	SF3B1 SFMBT1 SFXN2 SH3GL3		PDE4B PDE4D	PCDHGA2 PCDHGA3		RAD54L2 RANBP10	PLD5 PLEKHO1	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8 SLC3A10 SLC4A10 SLC6A9	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4		PDE4B PDE4D PDGFRL PER3	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5		RAD54L2 RANBP10 RANGAP1 RASA1	PLD5 PLEKHO1 PLPPR5 PLXNA2	
SLC17A7 SLC25A42 SLC25A5 SLC35G2 SLC35G2 SLC39A8 SLC4A10 SLC6A9 SMPX	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC12A4 SLC25A33		PDE4B PDE4D PDGFRL PER3 PEX5L	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2	PLD5 PLEKHO1 PLPPR5 PLXNA2 POC1A	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8 SLC4A10 SLC6A9 SMPX SMS	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1	PLD5 PLEKHO1 PLPPR5 PLXNA2 POC1A POC1A POLDIP3	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC35G2 SLC36A9 SLC4A10 SLC6A9 SMPX SMPS SMPS SMPS	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA8		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1 REEP2	PLD5 PLEKHO1 PLPPR5 PLXNA2 POC1A POC1A POLDIP3 PPARGC1A	
SLC17A7 SLC25A42 SLC2A5 SLC36G2 SLC36G2 SLC36A8 SLC4A10 SLC6A9 SMPX SMPX SMS SNTB2	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA8 PCDHGA9		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1	PLD5 PLEKHO1 PLPPR5 PLXNA2 POC1A POC1A PDARGC1A PPARGC1A PPP2R2A	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC39A8 SLC4A10 SLC6A9 SMPX SMS SMTB2 SNTB2 SNX7	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7 SLC35G2		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA8 PCDHGA9		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1 REEP2 REEL2	PLD5 PLEKHO1 PLPPR5 PLXNA2 POC1A POC1A PDARGC1A PPARGC1A PPP2R2A	
SLC17A7 SLC25642 SLC2A5 SLC3662 SLC39A8 SLC4A10 SLC6A9 MPX SMS SMFX SNTB2 SNX7 SNX8	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC2A33 SLC2A5 SLC2A7 SLC3A5 SLC3A5 SLC3A10		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PIH1D1	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA8 PCDHGA9 PCDHGB1		RAD54L2 RANBP10 RANGAP1 RASA1 RASA1 RC3H1 REEP2 RELL2 REEL2 RERE	PLD5 PLEKHO1 PLPPR5 PLXNA2 POCIA POLDIP3 PPARGC1A PPP2R2A PPP2R3A	
SLC17A7 SLC25A42 SLC2A5 SLC35G2 SLC35G2 SLC39A8 SLC4A10 SLC6A9 SMS SMS SMS SMTB2 SNX7 SNX8 SNX8 SOX2	SF3B1 SFMBT1 SFXN2 SF3X02 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7 SLC2A7 SLC35G2 SLC4A10 SLC1A1		PDE4B PDE4D PDE7RL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PIHT01 PIHT01 PITPNM2	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA9 PCDHGA9 PCDHGB1 PCDHGB2		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1 REEP2 RELL2 REERE RERE RFT1	PLD5 PLEFKHO1 PLPPR5 PLVPR5 POLDIP3 POLDIP3 PPARGC1A PPP2R2A PPP2R3A PRADC1	
LC17A7 SLC25A42 SLC2A5 SLC3A62 SLC3A63 SLC3A44 SLC3A5 SLC3A43 SLC3A44 SLC3A44 SLC3A45 SLC3A43 SLC4A10 SLC4A10 SLC6A9 SMPX SMS SNTB2 SNX7 SNX8 SOX2 SOX2-OT	SF3B1 SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7 SLC35G2 SLC3410 SLX18-SULT1A4		PDE4B PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PHF21A PHF1D1 PITPNM2 PKD2L2	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA7 PCDHGA8 PCDHGA8 PCDHGB1 PCDHGB1 PCDHGB2 PCDHGB3		RAD54L2 RANBP10 RANGAP1 RASA1 RASA12 RC3H1 REEP2 REEL2 RERE RFTN1	PLD5 PLEKHO1 PLPPR5 PLNA2 POC1A POCDIP3 PPARSC1A PPP2R2A PPP2R3A PRADC1 PRKCB	
SLC17A7 SLC25A42 SLC2A5 SLC3A62 SLC3A63 SLC4A10 SLC6A9 SMPX SMS SNTB2 SNX7 SNX8 SOX2 SOX2-OT	SF3B1 SFMBT1 SFXN2 SF3X02 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7 SLC2A7 SLC35G2 SLC4A10 SLC1A1		PDE4B PDE4D PDE7RL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PIHT01 PIHT01 PITPNM2	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA9 PCDHGA9 PCDHGB1 PCDHGB2		RAD54L2 RANBP10 RANGAP1 RASA1 RASAL2 RC3H1 REEP2 RELL2 REERE RERE RFT1	PLD5 PLEFKHO1 PLPPR5 PLVPR5 POLDIP3 POLDIP3 PPARGC1A PPP2R2A PPP2R3A PRADC1	
SLC17A7 SLC25A42 SLC2A5 SLC2A5 SLC3A5 SLC3A5 SLC3A10 SLC4A10 S	SF3B1 SFMBT1 SFXN2 SH32ACL3 SH39XD2A SLC12A4 SLC22A3 SLC2A5 SLC2A7 SLC3410 SLX1A SLX1A SLX1B-SULT1A4 SLX1P-SULT1A4		PDE48 PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PHF21A PHF1D1 PITPNM2 PKD2L2 PLCL1	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA6 PCDHGA6 PCDHGA7 PCDHGA7 PCDHGA8 PCDHGB4 PCDHGB1 PCDHGB2 PCDHGB3 PCDHGB3 PCDHGB4		RAD54L2 RANBP10 RANGAP1 RASA1 RASA1 RASA1 RASA1 RASA1 REP1 RC3H1 REEP2 RELL2 REFT RFT1 RFTN1 RFTN2	PLD5 PLEKHO1 PLPPR5 PLXNA2 POCDIP3 PPARGC1A PPP2R2A PPP2R3A PRADC1 PRKD6 PRKD1	
SLC17A7 SLC25A42 SLC25A42 SLC2A5 SLC3A5 SLC3A5 SLC3A88 SLC3A98 SLC3A98 SLC3A98 SLC3A98 SLC3A98 SLC3A98 SNT82 SNT82 SNT82 SNT82 SNT82 SNX7 SNX8 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2	SF3B1 SFMBT1 SFXN2 SF3X02A SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A5 SLC2A7 SLC3SG2 SLC4A10 SLC4A10 SLX14 SLX14-SULT1A4 SMG1P2 SMG6		PDE48 PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PIH1D1 PITPNM2 PKD2L2 PLCL1 PLCL2	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA7 PCDHGA7 PCDHGA8 PCDHGB1 PCDHGB1 PCDHGB2 PCDHGB2 PCDHGB4 PCDHGB5		RAD54L2 RANBP10 RANGAP1 RASA1 RASA12 RC3H1 RELL2 REFT RFT1 RFTN1 RFTN2 RILPL2	PLD5 PLEKHO1 PLPPR5 PLNA2 POC1A POLDIP3 PPARGC1A PPP2R2A PPP2R3A PRADC1 PRKCB PRKD1 PRUNE1	
SLC17A7 SLC25A42 SLC25A42 SLC2A5 SLC3A5 SLC3A50 SLC3A10 SLC6A9 SMPX SMPX SMS SMPX SMS SNX7 SNX8 SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3	SF3B1 SFMBT1 SFXN2 SH3QGJ3 SH3PXD2A SLC12A4 SLC2A5 SLC2A5 SLC2A5 SLC2A5 SLC2A5 SLC2A7 SLC3A5 SLC3A10 SLX14 SLC314 SLX18-SULT1A4 SMG1P2 SMG6 SMPX		PDE48 PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PIH1D1 PITPNM2 PKD2L2 PLCL1 PLCL1 PLCL2 PLD5	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA7 PCDHGA8 PCDHGB1 PCDHGB2 PCDHGB3 PCDHGB4 PCDHGB5 PCDHGB5 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6		RAD54L2 RANBP10 RANGAP1 RASA1 RASA1 RAS4 RC3H1 REL2 RERE RFT1 RFTN1 RFTN2 RILPL2 RPRD2	PLD5 PLEKHO1 PLPPR5 PLNR4 POC1A POLDIP3 PPARSC1A PPP2R2A PPP2R3A PRADC1 PRKCB PRKD1 PRUE1 PSD3	
SLC17A7 SLC25A42 SLC23642 SLC2365 SLC3662 SLC39A8 SLC3A10 SLC6A9 SMPX SMS SMPX SMS SMPX SMS SMPX SMS SMPX SMX8 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2 SOX2	SF3B1 SFMBT1 SFXN2 SH3QL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC3410 SLX1A SLX1A SLX1A SMG1P2 SMG6 SMS		PDE48 PDE40 PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PHF21A PHF21A PHTD1 PITPNM2 PKD2L2 PLCL1 PLCL1 PLCL2 PLD5 PLEKHO1	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA5 PCDHGA7 PCDHGA7 PCDHGA9 PCDHGB1 PCDHGB1 PCDHGB3 PCDHGB3 PCDHGB3 PCDHGB4 PCDHGB4 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB7		RAD54L2 RANBP10 RANGAP1 RASA1 RASA1 RASA1 RASA1 RASA1 RASA1 RASA1 RASA1 RASA1 RC3H1 REEP2 RELL2 RERE RFT1 RFTN1 RFTN2 RILPL2 RPRD2 RPS6KA3	PLD5 PLEKHO1 PLPPR5 PLXNA2 POCDIP3 PPARGC1A PPP2R2A PPP2R3A PRADC1 PRKCB PRKD1 PRUNE1 PSD3 PSKH1	
SH3RF1 SLC2FA42 SLC2FA42 SLC2FA42 SLC3A6 SLC36G2 SLC39A8 SLC39A8 SLC34A10 SLC6A9 SMS SMS SMS SMS SMS SMS SMS SMS SMS SM	SF3B1 SFMBT1 SFXN2 SH3QGJ3 SH3PXD2A SLC12A4 SLC2A5 SLC2A5 SLC2A5 SLC2A5 SLC2A5 SLC2A7 SLC3A5 SLC3A10 SLX14 SLC314 SLX18-SULT1A4 SMG1P2 SMG6 SMPX		PDE48 PDE4D PDGFRL PER3 PEX5L PGAP1 PHEX PHF21A PIH1D1 PITPNM2 PKD2L2 PLCL1 PLCL1 PLCL2 PLD5	PCDHGA2 PCDHGA3 PCDHGA4 PCDHGA5 PCDHGA6 PCDHGA7 PCDHGA7 PCDHGA8 PCDHGB1 PCDHGB2 PCDHGB3 PCDHGB4 PCDHGB5 PCDHGB5 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6 PCDHGB6		RAD54L2 RANBP10 RANGAP1 RASA1 RASA1 RAS4 RC3H1 REL2 RERE RFT1 RFTN1 RFTN2 RILPL2 RPRD2	PLD5 PLEKHO1 PLPPR5 PLNR4 POC1A POLDIP3 PPARSC1A PPP2R2A PPP2R3A PRADC1 PRKCB PRKD1 PRUE1 PSD3	

SUGP2	SNORD63	POC1A	PCNX3	SAP30L-AS1	PSMG3-AS1	
TAF5	SNX7	PODXL	PDE4B	SATB1	PTBP2	
TBC1D5	SNX8	PPARGC1A	PDE4D	SATB2	PTCHD1-AS	
TCF4	SOX2	PPIH	PDE6D	SBNO1	PTPRF	
TCTN2	SOX2-OT	PPM1L	PEMT	SCG2	R3HDM2	
TFAMP1	SPARC	PPP1CC	PEX5L	SDCCAG8	RAI1	
ТКТ	SPATS2L	PPP2R2A	PFKFB2	SEC16B	RASAL2	
TMEFF2	SREBF1	PPP2R3A	PGAP1	SEMA3G	RERE	
TMEM110	SRPK2	PRDX6	PGM3	SEMA4F	RFTN1	
TMEM110-						
MUSTN1	SRR	PSD3	PHEX	SERPINC1	RFTN2	
TMTC1	SSRP1	PSMD14	PHF21A	SF3B1	RNF220	
TNNI3K	ST3GAL3	PTCHD1-AS	PITPNM2	SFMBT1	RPS6KA3	
TRANK1	STAC3	PTPRF	PLCB2	SFXN5	RRP7BP	
TSPAN9	STAG1	RABGAP1L	PLCL1	SGO2	RTN4RL1	
TVP23A	STAT6	RAD54L2	PLCL2	SH3RF1	RTN4RL2	
TYW5	SUFU	RANBP10	PLD5	SLC17A7	RWDD2A	
JGT1A10	SYNPR	RANGAP1	PLEKHO1	SLC25A42	SAP30L-AS1	1
JGT1A6	TAF5	RASA1	PLPPR5	SLC2A5	SATB2	
JGT1A7	TBC1D5	RASAL2	PLXNA2	SLC35G2	SBNO1	
JGT1A8	THOC7	RC3H1	POC1A	SLC39A8	SDCCAG8	
JGT1A9	TLCD2	REEP2	POLDIP3	SLC4A10	SEMA3G	
JTP4	TLR9	RELL2	PPARGC1A		SEMA6C	
				SLC4A5		
/PS33A	TMEFF2	RERE	PPP2R2A	SLC6A9	SERHL2	
VPS45	TMEM110	RFT1	PPP2R3A	SLC9C2	SERPINE2	
	TMEM110-					
/RK2	MUSTN1	RFTN1	PRADC1	SMPX	SERPINF1	
/SIG2	TMEM161A	RFTN2	PRKCB	SMS	SF3B1	
WBP1L	TMX2-CTNND1	RILPL2	PRKD1	SNTB2	SFMBT1	
NDR55	TNNI3K	RPRD2	PRUNE1	SNX7	SFXN2	1
ZBED9	TOM1L2	RPS6KA3	PSD3	SNX8	SH3GL3	T.
ZBTB18	TOX	RSRC2	PSKH1	SOX2	SH3PXD2A	1
ZKSCAN4	TTC14	RTKN	PSMB10	SOX2-OT	SLC12A4	1
ZNF391	TWF2			SPATA24		
		SAP30L-AS1	PSMG3		SLC25A33	
ZNF804A	TYW5	SATB1	PSMG3-AS1	SRPK2	SLC2A5	
ZSCAN23	USP40	SATB2	PTBP2	ST3GAL3	SLC2A7	
ZSWIM6	VPS37B	SBN01	PTCHD1-AS	STAG1	SLC35G2	
	VPS45	SCG2	PTPRF	STK31	SLC4A10	
	VRK2	SDCCAG8	R3HDM2	SUGP1	SLX1A	
	VTRNA1-1	SEC16B	RAI1	SUGP2	SLX1B-SULT1A4	
	WBP1L	SEMA3G	RASAL2	TAF5	SMG1P2	1
	WDFY1	SEMA4F	RERE	TBC1D5	SMG6	ii
	WDR81	SERPINC1	RFTN1	TCF4	SMPX	1
	YY2	SF3B1	RFTN2		SMS	
				TCTN2		
	ZBED9	SFMBT1	RNF220	TET3	SNAP91	
	ZBTB18	SFXN5	RPS6KA3	TFAMP1	SNORD32B	
	ZDHHC5	SGO2	RRP7BP	ТКТ	SNORD63	
	ZFP57	SH3RF1	RTN4RL1	TMEFF2	SNX7	
	ZKSCAN3	SLC17A7	RTN4RL2	TMEM110	SNX8	
	ZMAT2	SLC25A42	RWDD2A	TMEM110-MUSTN1	SOX2	1
	ZNF592	SLC2A5	SAP30L-AS1	TMTC1	SOX2-OT	1
	ZNF804A	SLC35G2	SATB2	TNFSF4	SPARC	
	ZSCAN12	SLC39A8	SBN01	TNN	SPATS2L	
	ZSCAN23	SLC4A10	SDCCAG8	TNNC1	SREBF1	
	ZSCAN31	SLC4A5	SEMA3G	TNNI3K	SRPK2	
	ZSWIM6	SLC6A9	SEMA6C	TNR	SRR	
		SLC9C2	SERHL2	TRANK1	SSRP1	
		SMPX	SERPINE2	TSPAN9	ST3GAL3	
		SMS	SERPINF1	TVP23A	STAC3	
			O E O D I			
		SNTB2	SF3B1	TYW5	STAG1	
					STAG1	
		SNX7	SFMBT1	UGT1A5	STAG1 STAT6	
		SNX7 SNX8	SFMBT1 SFXN2	UGT1A5 UGT1A8	STAG1 STAT6 SUFU	
		SNX7 SNX8 SOX2	SFMBT1 SFXN2 SH3GL3	UGT1A5 UGT1A8 UGT1A10	STAG1 STAT6 SUFU SYNPR	
		SNX7 SNX8 SOX2 SOX2-OT	SFMBT1 SFXN2 SH3GL3 SH3PXD2A	UGT1A5 UGT1A8 UGT1A10 USP38	STAG1 STAT6 SUFU SYNPR TAF5	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THOC7	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THOC7 TLCD2	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 STAG1	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS35 VPS45 VRK2	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THCC7 TLCD2 TLR9	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 STAG1 STK31	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC35G2	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2	STAG1 STAT6 SUFU SYNPR TAF5 TBC105 THOC7 TLC02 TLR9 TMEFF2	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 STAG1	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS35 VPS45 VRK2	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THCC7 TLCD2 TLR9	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 STAG1 STK31	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC35G2	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THOC7 TLCD2 TLR9 TMEFF2 TMEM10	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 STAG1 STK31 SUGP1	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC25A33 SLC2A5 SLC2A7 SLC36G2 SLC4A10	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L	STAG1 STAT6 SUFU SYNPR TAF5 TBC105 THOC7 TLC02 TLR9 TMEFF2	
		SNX7 SNX8 SOX2 SOX2-T SPATA24 SRPK2 ST3GAL3 STAG1 STK31 SUGP1 SUGP1	SFMBT1 SFXN2 SH3GL3 SH3FXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC3GG2 SLC4A10 SLX1A	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THC7 TLCD2 TRFF2 TMEM110-MUSTN1 TMEM161A	
		SNX7 SNX8 SOX2 SOX20 SPATA24 SRPK2 ST3GAL3 STA61 STK31 SUGP1 SUGP2 TAF5 TBC1D5	SFMBT1 SFXN2 SH3GL3 SH3FXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC3G22 SLC410 SLX1A SLX1A SLX1A SLX1A SLX1A SLX1A SLX1B-SULT1A4 SMG1P2	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZBED9	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 TLCD2 TLR9 TMEEFF2 TMEM110-MUSTN1 TMEM161A TMX2-CTNND1	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SPATA24 STAG1 STAG1 STK31 SUGP1 SUGP2 TAF5 TBC1D5 TCF4	SFMBT1 SFXN2 SH3GL3 SH3FXD2A SLC25A33 SLC2A5A33 SLC2A5 SLC2A5 SLC3SG2 SLX1A SLX1B-SULT1A4 SMG1P2 SMG6	UGT1A5 UGT1A1 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZBED9 ZBTB18	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THOC7 TLCD2 TMEFF2 TMEM110-MUSTN1 TMEM161A TMX2-CTNND1 TNNI3K	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 ST3GAL3 ST4G1 ST4G1 SUGP1 SUGP1 SUGP2 TAF5 TBC1D5 TCF4 TCTN2	SFMBT1 SFXN2 SH3GL3 SH3PXD2A SLC25A33 SLC2A5A33 SLC2A7 SLC2A7 SLC3G2 SLX1A SLX1A SLX1A SLX1A SMG1P2 SMG6 SMPX	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZBED9 ZBTB18 ZBTB37	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 TLCD2 TLR9 TMEFF2 TMEM10-MUSTN1 TMEM161A TMX2-CTNND1 TNNI3K	
		SNX7 SNX8 SOX2 SOX2-0T SPATA24 SRPK2 STAG1 STAG1 STAG1 SUGP1 SUGP1 SUGP2 TAF5 TEC105 TCF4 TCTN2 TET3	SFMBT1 SFXN2 SH3GL3 SH3GL3 SH3FXD2A SLC12A4 SLC25A33 SLC2A5 SLC2A7 SLC3G2 SLC4A10 SLX1B-SULT1A4 SMG6 SMPX SMS	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 WRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZBED9 ZBTB18 ZBTB37 ZEB2	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THC07 TLCD2 TMEM10 TMEM110 TMEM110 TMEX10000 TMX2-CTNND1 TMN3K TOM1L2	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SPATA24 STAG1 STAG1 STK31 SUGP1 TAF5 TBC1D5 TCFF4 TCTN2 TET3 TFAMP1	SFMBT1 SFXN2 SH3GL3 SH3GL3 SL212A4 SLC25A33 SLC2A5 SLC2A5 SLC2A6 SLC2A7 SLX18-SULT1A4 SMG6 SMPX SMAS SNAP91	UGT1A5 UGT1A6 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZBED9 ZBTB18 ZBTB37 ZEB2 ZKSCAN4	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THOC7 TLCD2 TMEFF2 TMEM110-MUSTN1 TMEM161A TMNI3K TOM1L2 TOX TTC14	
		SNX7 SNX8 SOX2 SOX2 SPATA24 SRPK2 ST3GAL3 STAG1 STAG1 STAG1 ST4G1 STGAL3 TAF5 TBC1D5 TCF4 TCTN2 TET3 TFAMP1 TKT	SFMBT1 SFXN2 SH3GL3 SH3GL3 SH2PXD2A SLC12A4 SLC2A5 SLC2A7 SLC3SG2 SLA10 SLX18-SULT1A4 SMG6 SMG6 SMS SNAP91 SNORD32B	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WH5C1L1 ZBED9 ZBTB18 ZBTB37 ZEB2 ZKSCAN4 ZNF391	STAG1 STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THCC7 TLCD2 TLR9 TMEM110-MUSTN1 TMEM161A TMX2-CTNND1 TMN3K TOM1L2 TOX TTC14 TWF2	
		SNX7 SNX8 SOX2 SOX2-OT SPATA24 SRPK2 STAG1 STAG1 STAG1 STAG1 STAG1 STAG1 TGC105 TCF4 TCTN2 TF33 TFAMP1 TKT TMEFF2	SFMBT1 SFXN2 SH3GL3 SH3GL3 SL212A4 SLC25A33 SLC2A7 SLC3G2 SLC4A10 SLX18-SULT1A4 SMG1P2 SMG6 SMX SMAP31 SNAP91 SNORD32B SNORD63	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WHSC1L1 ZEED9 ZBTB18 ZBTB37 ZEB2 ZKSCAN4 ZNF391 ZNF391 ZNF394A	STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THC07 TLCD2 TMEM10 TMEM110 TMEM110 TMEXTIN TMEXTIN TMEXTIN TMEXTIN TOX2 TOX TC14 TYW5	
		SNX7 SNX8 SOX2-OT SOX2-OT SPATA24 STAFA STGGL3 STGAL3 STAF5 TBC1D5 TCF4 TCTN2 TET3 TFAMP1 TKT TMEFF2 TMEH10	SFMBT1 SFXN2 SH3GL3 SH3GL3 SH2PXD2A SLC12A4 SLC2A5 SLC2A7 SLC3SG2 SLA10 SLX18-SULT1A4 SMG6 SMG6 SMS SNAP91 SNORD32B	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 VRK2 VSIG2 WBP1L WDR55 WH5C1L1 ZBED9 ZBTB18 ZBTB37 ZEB2 ZKSCAN4 ZNF391	STAG1 STAG1 STAT6 SUFU SYNPR TAF5 TBC1D5 THCC7 TLCD2 TLR9 TMEM110-MUSTN1 TMEM161A TMX2-CTNND1 TMN3K TOM1L2 TOX TTC14 TWF2	
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		SNX7 SNX8 SOX2 SOX2-0T SPATA24 SRPK2 STAG1 STAG1 STAG1 STAG1 STAG1 STAG1 TGC105 TCF4 TCTN2 TET3 TFAMP1 TKT TMEFF2 TMEM110-	SFMBT1 SFXN2 SH3GL3 SH3GL3 SH2PXD2A SLC12A4 SLC25A33 SLC2A7 SLC3SG2 SLC4A10 SLX1B-SULT1A4 SMG6 SMG6 SMS SNAP91 SNORD32B SNX7 SNX8 SOX2	UGT1A5 UGT1A8 UGT1A10 USP38 UTP4 VPS33A VPS45 WRK2 WSIG2 WBP1L WDR55 WHSC1L1 ZBED9 ZBTB18 ZBTB37 ZEB2 ZKSCAN4 ZNF831 ZNF804A ZSCAN23	STAG1 STAG6 SUFU SYNPR TAF5 TBC1D5 THCC7 TLCD2 TLR9 TMEM110 TMEM110 TMEXM110 TMMIN10 TMEXM110 TMEXM110 TMEXM110 TMEXCTIND1 TNN3X TOX12 TOX TTC14 TWF2 TYW5 USP40	
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VRK2	TMEFF2			
VSIG2	TMEM110			
	TMEM110-			
WBP1L	MUSTN1			
WDR55	TMEM161A			
WHSC1L1	TMX2-CTNND1			
ZBED9	TNNI3K			
ZBTB18	TOM1L2			
ZBTB37	тох			
ZEB2	TTC14			
ZKSCAN4	TWF2			
ZNF391	TYW5			1
ZNF804A	USP40			
ZSCAN23	VPS37B			1
ZSWIM6				

Table 3.15 | Genes located in cell-type specific SZ risk associatedchromosomal contacts.

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GO:0052897 kenobiotic glucuronidation GO. BiologicalProcess-GOA. 23.02.2017. 10h01 31.0E-15 93.0.0E-15 12.0E+0 77.78 7.00 GO:1904224 negative regulation of glucuronosyttransferase activity GO. BiologicalProcess-GOA. 23.02.2017. 10h01 33.5E-12 100.0E+12 10.0E+0 7.0E 6.00 GO:0000983 Parug metabolism KEGG. 01.03.2017 1.2E-4 33.0E+0 7.7E+0 25.33 7.00 GO:0000210 Glucuronate interconversions KEGG. 01.03.2017 1.2E-4 33.0E+0 6.6.3E+0 16.6.7 7.00 GO:0000080 Perphyrin and chlorophyll metabolism KEGG. 01.03.2017 19.0E-9 480.0E-9 6.3E+0 16.6.7 7.00 GO:0000802 Perphyrin and chlorophyll metabolism KEGG. 01.03.2017 19.0E-9 480.0E-9 6.3E+0 16.6.7 7.00 GO:0000802 Drug metabolism KEGG. 01.03.2017 19.0E-6 4.0E-6 11.44 8.00 GO:00002040 Bernical carcinogenesis KEGG. 01.03.2017 2.1E-6 4.0E-6 4.4E+0 7.00 7.00 7.00 <td< th=""><th></th><th></th><th></th><th>Term</th><th>Bonferroni</th><th>Neg log</th><th></th><th></th></td<>				Term	Bonferroni	Neg log		
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GC:00000053 Ascorbate and aldarate metabolism KEGG_01.03.2017 (99.0E-12 19.0E-9 7.7E+0 25.93 7.0C GO:0000083 Drug metabolism KEGG_01.03.2017 1.2E-9 33.0E-9 7.5E+0 17.39 8.00 GO:0000210 Glucuronate interconversions KEGG_01.03.2017 15.0E-9 380.0E-3 6.4E+0 25.00 6.0 GO:0000820 Derphyrin and chlorophyll metabolism KEGG_01.03.2017 19.0E-9 460.0E-9 6.3E+0 12.31 8.00 GO:0000820 Retinol metabolism KEGG_01.03.2017 21.0E-9 490.0E-9 6.3E+0 12.31 8.00 GO:0000820 Drug metabolism KEGG_01.03.2017 19.0E-9 4.0E-6 5.4E+0 12.07 7.00 GO:0000520 Chemical carcinogenesis KEGG_01.03.2017 1.0E-6 21.0E-6 4.7E+0 9.46 7.0C GO:0005200 Chemical carcinogenesis KEGG_01.03.2017 2.1E-6 4.0E+0 8.64 7.0C GO:0005200 Chemical carcinogenesis KEGG_01.03.2017 2.0E+0	GO:1904224	negative regulation of glucuronosyltransferase activity						
GC:0000040 Pentose and glucuronate interconversions KEGG_010.32017 4.0E-9 100.0E-9 7.0E+0 20.59 7.0C GO:0000210 Glucuronidation REACTOME_Pathways_01.03.2017 15.0E-9 380.0E-9 6.4E+0 25.00 6.07 00 GO:0000800 Periphyrin and chicrophyll metabolism KEGG_01.03.2017 19.0E-9 460.0E-9 6.3E+0 16.67 7.0C GO:0000803 Retinol metabolism KEGG_01.03.2017 21.0E-9 490.0E-9 6.3E+0 11.43 8.00 GO:0000804 Istericid hormone biosynthesis KEGG_01.03.2017 190.0E-9 4.0E-6 5.4E+0 12.07 7.00 GO:0000804 Metabolism of xenobiolics by cytochrome P450 KEGG_01.03.2017 1.0E-6 21.0E-6 4.7E+0 9.46 7.00 GO:0005204 Chemical carcinogenesis KEGG_01.03.2017 2.1E-6 40.0E+0 3.7E+0 66.0E-6 4.2E+0 7.87 7.00 GO:0005204 DensidogicalProcess-GOA_2.302.2017_10h01 3.7E+6 60.0E-6 4.2E+0 7.87 7.00 60.00010675 <td>GO:000053</td> <td>Ascorbate and aldarate metabolism</td> <td>KEGG_01.03.2017</td> <td>690.0E-12</td> <td>19.0E-9</td> <td>7.7E+0</td> <td>25.93</td> <td>7.00</td>	GO:000053	Ascorbate and aldarate metabolism	KEGG_01.03.2017	690.0E-12	19.0E-9	7.7E+0	25.93	7.00
GC:0000210 Glucuronidation REACTOME Pathways_01.03.2017 15.0E-9 380.0E-9 6.4E+0 25.00 6.0 GO:0000800 Retinol metabolism KEGG 01.03.2017 19.0E-9 460.0E-9 6.3E+0 12.31 8.00 GO:0000802 Drug metabolism KEGG 01.03.2017 39.0E-8 860.0E-9 6.1E+0 11.4.3 8.00 GO:0000802 Breid hormone biosynthesis KEGG 01.03.2017 19.0E-9 40.0E-4 5.4E+0 12.07 7.00 GO:0000800 Metabolism of xenobiolics by cytochrome P450 KEGG 01.03.2017 1.0E-6 21.0E-6 4.7E+0 9.46 7.00 GO:0000800 Metabolism of xenobiolics by cytochrome P450 KEGG 01.03.2017 2.1E-6 40.0E-6 4.4E+0 8.54 7.00 GO:00045833 negative regulation of lipid metabolic process GO_DiologicalProcess-GOA_23.02.2017_10h01 3.7E+0 5.44 8.64 7.00 GO:0000205 Phase II conjugation REACTOME Pathways 01.03.2017 100.0E-6 1.4E+3 2.9E+0 5.83 6.00 GO:0000205								
GC:0000860 Porphyrin and chlorophyll metabolism KEGG 01.03.2017 19.0E-9 460.0E-9 6.3E+0 16.67 7.00 GO:0000803 Retinol metabolism KEGG 01.03.2017 21.0E-9 490.0E-9 6.3E+0 12.31 8.00 GO:0000800 Steroid hormone biosynthesis KEGG 01.03.2017 190.0E-9 4.0E-6 5.4E+0 11.43 8.00 GO:0000804 Netabolism of xenobiotics by cytochrome P450 KEGG 01.03.2017 1.00.E-6 21.0E-6 4.0E-6 4.7E+0 9.46 7.00 GO:00008204 Chemical carcinogenesis KEGG 01.03.2017 2.1E-6 40.0E-6 4.7E+0 9.46 7.00 GO:0008204 Chemical carcinogenesis KEGG 01.03.2017 2.1E-6 40.0E-6 4.7E+0 7.46 GO:0008205 Plasa II conjugation Cellular carbohydrate metabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 3.7E+6 66.0E-6 4.2E+0 7.87 7.00 GO:0000205 Fleast III conjugation REACTOME_Pathways 01.03.2017 100.0E-6 1.3E+3 2.8E+0 5.83 6.00								
CO.0000982 Drug metabolism KEGG_01.03.2017 39.0E-9 860.0E-9 6.1E-0 11.43 8.00 GO.00009140 Steroid hormone biosynthesis KEGG_01.03.2017 190.0E-9 4.0E-6 5.4E+0 12.07 7.00 GO.00005204 Chemical carcinogenesis KEGG_01.03.2017 1.0E-6 21.0E-6 4.0E-6 4.7E+0 9.46 7.00 GO.0005204 Chemical carcinogenesis KEGG_01.03.2017 2.1E-6 40.0E-6 4.4E+0 8.54 7.00 GO.0005204 Chemical carcinogenesis GO_BiologicalProcess-GOA_23.02.2017_10h01 3.7E+6 66.0E-6 4.2E+0 7.87 7.00 GO.00005205 regulation of cellular carbohydrate metabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 11.0E-6 19.0D-6 3.7E+0 5.44 8.06 GO.0000208 Cytosolic sulfonation of small molecules REACTOME_Pathways_0.10.3.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO.0000208 Cytosolic sulfonation of small molecules REACTOME_Pathways_0.10.3.2017 100.0E-6 1.5E-3 2.8E+0 5.83 </td <td>GO:000860</td> <td>Porphyrin and chlorophyll metabolism</td> <td>KEGG_01.03.2017</td> <td>19.0E-9</td> <td>460.0E-9</td> <td>6.3E+0</td> <td>16.67</td> <td>7 7.00</td>	GO:000860	Porphyrin and chlorophyll metabolism	KEGG_01.03.2017	19.0E-9	460.0E-9	6.3E+0	16.67	7 7.00
GO:0000140 Steroid hormone biosynthesis KEGG: 01.03.2017 190.0E-s 4.0E-6 5.4E+d 12.07 7.00 GO:000080 Metabolism of xenobiotics by cytochrome P450 KEGG: 01.03.2017 1.0E-6 21.0E-6 4.7E+d 9.46 7.00 GO:0005204 Chemical carcinogenesis KEGG: 01.03.2017 2.1E-6 40.0E-6 4.4E+0 8.54 7.00 GO:0005204 Chemical carcinogenesis GO: BiologicalProcess-GOA_23.02.2017_10h01 3.7E-6 66.0E-6 4.2E+0 7.87 7.00 GO:0001675 fegulation of cellular carbohydrate metabolic process GO BiologicalProcess-GOA_23.02.2017_10h01 190.0E-6 3.7E+0 5.44 8.04 GO:0000205 Phase II conjugaton REACTOME_Pathways 01.03.2017 100.0E+6 1.5E-3 2.8E+0 5.83 6.00 GO:0000205 Phase II conjugaton REACTOME_Pathways 01.03.2017 100.0E+6 1.5E-3 2.8E+0 5.83 6.00 GO:0000421 pigment metabolic process GO MolecularFunction-GOA_23.02.2017_10h01 190.0E+6 5.5E-3 2.2E+0 4.76 5.00	GO:000830	Retinol metabolism						
GC:0000980 Metabolism of xenobiotics by cytochrome P450 KEGG. 01.03.2017 1.0E-6 21.0E-6 4.7E+0 9.46 7.00 GO:00005204 Chemical carcinogenesis KEGG. 01.03.2017 2.1E-6 40.0E-6 4.4E+0 8.56 7.00 GO:0005204 Chemical carcinogenesis GO_MolecularFunction-GOA_23.02.2017_10h01 3.7E+6 66.0E-6 4.2E+0 7.87 7.00 GO:00010757 Fegulation of cellular carchohydrate metabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 11.0E-6 190.0E-6 3.7E+0 5.44 8.06 GO:0000205 Phase II conjugation REACTOME Pathways 01.03.2017 100.0E-6 1.4E-3 2.9E+0 5.83 6.00 GO:0000205 Phase II conjugation REACTOME Pathways 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:00001972 petinotic acid binding GO_MolecularFunction-GOA_23.02.2017_10h01 190.0E-6 2.7E-3 2.6E+0 6.76 5.00 GO:0004307 Metabolic process GO_CellularComponent-GOA_23.02.2017_10h01 190.0E-6 5.5E-3 2.3E+0 6.								
GO:0005204 Chemical carcinogenesis KEGG.010.32017 2.1E-6 40.0E-6 4.4E+0 8.54 7.00 GO:0005204 Chemical carcinogenesis GO BiologicalProcess-GOA_23.02.2017_10h01 3.7E-6 66.0E-6 4.2E+0 7.07 GO:000575 fegative regulation of callular carbohydrate metabolic process GO MolecularFunction-GOA_23.02.2017_10h01 11.0E-6 19.00-E4 3.7E+0 5.44 8.00 GO:00001075 fegulation of callular carbohydrate metabolic process GO BiologicalProcess-GOA_23.02.2017_10h01 19.00-E4 1.5E-3 2.8E+0 5.83 6.00 GO:0000208 Cytosolic sulfonation of small molecules REACTOME_Pathways_01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0001027 Letinoic acid binding GO_DiologicalProcess-GOA_23.02.2017_10h01 190.0E-6 3.2E+0 6.67 5.00 GO:0001972 Letinoic acid binding GO_CollouarComponent-GOA_23.02.2017_10h01 190.0E-6 3.2E+10 5.66 4.00 GO:0001972 Letinoic acid binding GO_CollouarComponent-GOA_23.02.2017_10h01 180.0E-6			KEGG_01.03.2017					
GO:0008194 JUDP-glycosyltransferase activity GO. MolecularFunction-GOA_23.02.2017_10h01 11.0E-6 190.0E-6 3.7E+0 5.44 8.00 GO:0001075 regulation of cellular carbohydrate metabolic process GO. BiologicalProcess-GOA_23.02.2017_10h01 92.0E-6 1.4E-3 2.9E+0 4.79 7.00 GO:0000208 Phase II conjugation REACTOME Pathways_0.10.3.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0000208 Cytosolic sulfonation of small molecules REACTOME Pathways_0.10.3.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0002018 Cytosolic sulfonation of small molecules REACTOME Pathways_0.10.3.2017 100.0E-6 1.5E-3 2.8E+0 6.5.8 6.00 GO:0001972 Jetinoic acid binding GO_MolecularComponent-GOA_23.02.2017_10h01 190.0E-6 3.6E-3 2.4E+0 16.67 3.00 GO:0004320 Anherens junction KEGG_0.10.3.2017 1.48-3 1.0E-3 1.9E+0 4.23 3.00 GO:000462 B cell receptor signaling pathway KEGG_0.10.3.2017 1.4E-3 1.0E-3 1.8	GO:0005204	Chemical carcinogenesis	KEGG_01.03.2017	2.1E-6	40.0E-6	4.4E+(4 7.00
GO:0010675 Fegulation of cellular carbohydrate metabolic process GO. BiologicalProcess-GOA_23.02.2017_10h01 92.0E-6 1.4E-3 2.9E+0 4.79 7.00 GO:0000205 Phase II conjugation REACTOME_Pathways 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0000205 Cytosolic sulfonation of small molecules REACTOME_Pathways 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0001202 Evitasolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 190.0E-6 1.5E-3 2.8E+0 6.76 5.00 GO:0001272 Leinoic acid binding GO_CollularComponent-GOA_23.02.2017_10h01 190.0E-6 3.6E-3 2.4E+0 1.66.7 3.00 GO:0004520 Adherritic spine GO_CollularComponent-GOA_23.02.2017_10h01 1400.E-6 5.5E-3 2.3E+0 4.41 6.07 GO:0004520 Adherritic spine GO CollularComponent-GOA_23.02.2017_10h01 1400.E-6 5.5E-3 2.3E+0 4.31 6.07 GO:0004620 Adherritic spine GO CollularComponent-GOA_23.02.2017_10h01 1.8E-3								
GO:000205 Phase II conjugation REACTOME Pathways. 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0000205 Phase II conjugation REACTOME Pathways. 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.83 6.00 GO:0000205 Potositic suffonation of small molecules REACTOME Pathways. 01.03.2017 100.0E-6 1.5E-3 2.8E+0 5.63 6.00 GO:0001972 petinotic acid binding GO_MolecularFunction-GOA 23.02.2017_10h01 190.0E-6 2.7E-3 2.6E+0 6.76 5.00 GO:00045197 petinotic acid binding GO_CellularComponent-GOA 23.02.2017_10h01 270.0E-6 3.6E-3 2.4E+0 16.67 3.00 GO:0004520 Adherins junction KEGG 0.103.2017 1.8E-3 1.40E-3 1.9E+0 5.56 4.00 GO:0004620 Long-term potentiation KEGG 0.103.2017 1.4E-3 1.9E+0 4.23 3.00 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_0.10.3.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00<								
GO:0042440 pigment metabolic process GO: BiologicalProcess-GOA 23.02.2017_10h01 190.0E-6 2.7E-3 2.6E+0 6.76 5.00 GO:0001972 retinoic acid binding GO< MolecularFunction-GOA 23.02.2017_10h01	GO:0000205	Phase II conjugation	REACTOME_Pathways_01.03.2017	100.0E-6	1.5E-3	2.8E+0	5.83	6.00
GO:0001972 Tethnoic acid binding GO MolecularFunction-GA 23.02.2017 10h01 270.0E-6 3.6E-3 2.4E+d 16.67 3.00 GO:000453197 Jendritic spine GO_CellularComponent-GOA_23.02.2017_10h01 460.0E-6 5.5E-3 2.3E+d 4.41 6.00 GO:0004520 Adherens junction KEGG_01.03.2017 1.8E-1 1.0E-3 1.9E+0 4.23 3.00 GO:0004620 B cell receptor signaling pathway KEGG_01.03.2017 1.4E-3 1.5E-3 1.8E+0 5.56 4.00 GO:0001462 B cell receptor signaling pathway KEGG_01.03.2017 1.4E-3 15.0E-3 1.8E+0 5.56 4.00 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001073 Inblocking of NIDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 <t< td=""><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></t<>								
GO:003197 Generation GO_CellularComponent-GOA_23.02.2017_10h01 460.0E-6 5.5E-3 2.3E+0 4.41 6.00 GO:0004520 Acternas junction KEGG_01.03.2017 1.8E-3 14.0E-3 1.9E+0 5.56 4.00 GO:0004620 Bcell receptor signaling pathway KEGG_01.03.2017 14.0E-3 14.0E-3 1.9E+0 4.23 3.00 GO:0004720 Long-term potentiation KEGG_01.03.2017 14.4E-3 15.0E-3 1.8E+0 6.5.97 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 6.5.8 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 6.63 4.00 GO:0001073 Unblocking of NMDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001072 Unblocking of NMDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0								
GO:0004662 B cell receptor signaling pathway KEGG_01.03.2017 14.0E-3 14.0E-3 19.E+0 4.23 3.0 GO:0004662 B cell receptor signaling pathway KEGG_01.03.2017 1.4E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 5.63 4.00 GO:0001073 Unblocking of NIDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001073 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:000173 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3	GO:0043197	dendritic spine	GO_CellularComponent-GOA_23.02.2017_10h01	460.0E-6	5.5E-3	2.3E+0	4.41	6.00
GO:0004720 Long-term potentiation KEGG.010.32017 1.4E-3 15.0E-3 1.8E+0 5.57 4.00 GO:0001464 Interactions of neuroxins and neuroligins at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001464 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001073 Unblocking of NMDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001073 Unblocking of NMDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001073 CREB phosphorylation through the activation of CaMKII REACTOME_Pathways 0.10.3.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways 0.10.3.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways 0.1								
GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 5.63 4.00 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 1.7E-3 15.0E-3 1.8E+0 9.09 3.00 GO:0001073 Johlooking of NMDA receptoring dutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses GO_BiologicalProcess-GOA_23.02.2017_10h01 6.2E-3 18.8E+0 9.09 3.00 GO:00051310 metaphase plate congression GO_BiologicalProcess-GOA_23.02.2017_10h01 6.2E-3 18.0E-3 1.7E+0								
GO:0001073 Unblocking of NMDA receptor, glutamate binding and activation REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001082 CREB phosphorylation through the activation of CaMKII REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001082 CREB phosphorylation through the activation of CaMKII REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0051710 Interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0051710 Interactions with VEGF and VEGFR GO_BiologicalProcess-GOA_2.3.02.2017_10h01 6.2E-3 18.0E-3 1.7E+0 5.77 3.00 GO:0005211 Neurophilin interactions with VEGF and VEGFR REACTOME_Pathways_01.03.2017 3.2E-3 2.0E-3 1.7E+0 7.32 3.00	GO:0001464	Interactions of neurexins and neuroligins at synapses	REACTOME_Pathways_01.03.2017	1.7E-3	15.0E-3	1.8E+0	5.63	4.00
GO:0001082 CREB phosphorylation through the activation of CaMKII REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001082 CREB phosphorylation through the activation of CaMKII REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001733 SALM protein interactions at the synapses REACTOME_Pathways_01.03.2017 1.7E-3 17.0E-3 1.8E+0 9.09 3.00 GO:0001733 Detail to compression GO_BiologicalProcess-GOA_23.02.2017_10h01 6.2E-3 18.0E-3 1.7E+0 5.77 3.00 GO:000521 Neurophilin interactions with VEGF and VEGFR REACTOME_Pathways_01.03.2017 3.2E-3 2.2E-3 1.7E+0 7.32 3.00								
GO:0001733 SALM protein interactions at the synapses REACTOME Pathways 0.1.03.2017 1.7E-3 1.7.8E-4 0.09 3.00 GO:0001733 SALM protein interactions at the synapses GO								
GO:0051310 metaphase plate congression GO_BiologicalProcess-GOA_23.02.2017_10h01 6.2E-3 18.0E-3 1.7E+0 5.77 3.00 GO:0000521 Neurophilin interactions with VEGF and VEGFR REACTOME_Pathways_01.03.2017 3.2E-3 22.0E-3 1.7E+0 7.32 3.00			REACTOME_Pathways_01.03.2017					3.00
							5.77	

	4 SEMA3A-Plexin repulsion signaling by inhibiting Integrin adhesion	REACTOME_Pathways_01.03.2017	3.2E-3	22.0E-3	1.7E+0	7.32 3.0
	3 Signal transduction by L1	REACTOME_Pathways_01.03.2017	3.2E-3	22.0E-3	1.7E+0	7.32 3.0
	9 forebrain neuron differentiation	GO_BiologicalProcess-GOA_23.02.2017_10h01	11.0E-3	23.0E-3	1.6E+0	4.62 3.0
	1 Renal cell carcinoma	KEGG_01.03.2017	11.0E-3	23.0E-3	1.6E+0	4.62 3.0
	6 Nuclear Events (kinase and transcription factor activation) 8 ERK/MAPK targets	REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	5.9E-3 5.9E-3	23.0E-3 23.0E-3	1.6E+0	5.88 3.0 5.88 3.0
	4 CREB phosphorylation	REACTOME_Pathways_01.03.2017	5.9E-3	23.0E-3	1.6E+0	5.88 3.0
	6 MAPK targets/ Nuclear events mediated by MAP kinases	REACTOME_Pathways_01.03.2017	5.9E-3	23.0E-3	1.6E+0	5.88 3.0
GO:000129	1 CD209 (DC-SIGN) signaling	REACTOME_Pathways_01.03.2017	5.9E-3	23.0E-3	1.6E+0	5.88 3.0
	3 Pre-NOTCH Processing in Golgi	REACTOME_Pathways_01.03.2017	4.1E-3	25.0E-3	1.6E+0	6.67 3.0
	4 Pre-NOTCH Expression and Processing	REACTOME_Pathways_01.03.2017	4.1E-3	25.0E-3	1.6E+0	6.67 3.0
	1 regulation of mitotic metaphase/anaphase transition	GO_BiologicalProcess-GOA_23.02.2017_10h01	5.3E-3	26.0E-3	1.6E+0	6.12 3.0
NPC (PGC2	2 + CLOZUK)					
				Term		
				PValue Corrected		
				with		%
			Term	Benjamini-	Neg log	Associated Nr.
GOID	GOTerm	Ontology Source	PValue	Hochberg	(FDR)	Genes Genes
	4 protein transport within lipid bilayer	GO_BiologicalProcess-GOA_23.02.2017_10h01	59.0E-9	8.6E-6		62.50 5.0
	2 flavone metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	82.0E-6 120.0E-6	6.0E-3	2.2E+0	50.00 3.0
	Adrenergic signaling in cardiomyocytes Adrenergic signaling in cardiomyocytes	KEGG_01.03.2017 KEGG_01.03.2017	120.0E-6 120.0E-6	6.1E-3 6.1E-3	2.2E+0 2.2E+0	6.94 10.0 6.94 10.0
	1 Adrenergic signaling in cardiomyocytes	KEGG_01.03.2017	120.0E-6		2.2E+0	6.94 10.0
	7 xenobiotic glucuronidation	GO_BiologicalProcess-GOA_23.02.2017_10h01	330.0E-6	6.9E-3	2.2E+0	33.33 3.0
	2 sister chromatid cohesion	GO BiologicalProcess-GOA 23.02.2017 10h01	290.0E-6	7.2E-3	2.1E+0	6.87 9.0
GO:000589	1 voltage-gated calcium channel complex	GO_CellularComponent-GOA_23.02.2017_10h01	510.0E-6	7.4E-3	2.1E+0	12.20 5.0
GO:0004024	4 cAMP signaling pathway	KEGG_01.03.2017	410.0E-6	7.5E-3	2.1E+0	5.56 11.0
	4 cAMP signaling pathway	KEGG_01.03.2017	410.0E-6	7.5E-3	2.1E+0	5.56 11.0
	8 striated muscle adaptation	GO_BiologicalProcess-GOA_23.02.2017_10h01	570.0E-6	7.6E-3	2.1E+0	11.90 5.0
	8 striated muscle adaptation	GO_BiologicalProcess-GOA_23.02.2017_10h01	570.0E-6	7.6E-3	2.1E+0	11.90 5.0
	0 microtubule organizing center part 8 Dopaminergic synapse	GO_CellularComponent-GOA_23.02.2017_10h01 KEGG 01.03.2017	480.0E-6 280.0E-6	7.8E-3 8.1E-3	2.1E+0 2.1E+0	5.88 10.0 6.92 9.0
	8 Dopaminergic synapse	KEGG_01.03.2017 KEGG_01.03.2017	280.0E-6	8.1E-3	2.1E+0	6.92 9.0
	8 Dopaminergic synapse	KEGG_01.03.2017	280.0E-6	8.1E-3	2.1E+0	6.92 9.0
	0 Long-term potentiation	KEGG_01.03.2017	760.0E-6		2.0E+0	8.96 6.0
	1 Amphetamine addiction	KEGG_01.03.2017	820.0E-6	9.3E-3	2.0E+0	8.82 6.0
GO:000472	0 Long-term potentiation	KEGG_01.03.2017	760.0E-6	9.3E-3	2.0E+0	8.96 6.0
	1 Amphetamine addiction	KEGG_01.03.2017	820.0E-6	9.3E-3	2.0E+0	8.82 6.0
GO:000581		GO_CellularComponent-GOA_23.02.2017_10h01	960.0E-6	10.0E-3	2.0E+0	6.45 8.0
	3 alpha-actinin binding	GO_MolecularFunction-GOA_23.02.2017_10h01	270.0E-6	10.0E-3	2.0E+0	13.89 5.0
GO:003564		GO_BiologicalProcess-GOA_23.02.2017_10h01	1.6E-3	14.0E-3	1.9E+0	20.00 3.0
	1 Ino80 complex 9 sister chromatid segregation	GO_CellularComponent-GOA_23.02.2017_10h01 GO BiologicalProcess-GOA 23.02.2017 10h01	1.6E-3 1.5E-3	14.0E-3 14.0E-3	1.9E+0	20.00 3.0 4.74 11.0
	5 chromosome, centromeric region	GO CellularComponent-GOA 23.02.2017 10h01	1.6E-3	14.0E-3	1.9E+0	5.03 10.0
	1 dermatan sulfate proteoglycan biosynthetic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	1.6E-3	14.0E-3	1.9E+0	20.00 3.0
	3 SWI/SNF superfamily-type complex	GO_CellularComponent-GOA_23.02.2017_10h01	2.0E-3	15.0E-3	1.8E+0	7.41 6.0
	6 regulation of translational initiation	GO_BiologicalProcess-GOA_23.02.2017_10h01	2.1E-3	15.0E-3	1.8E+0	7.32 6.0
GO:0005410	0 Hypertrophic cardiomyopathy (HCM)	KEGG_01.03.2017	2.3E-3	15.0E-3	1.8E+0	7.23 6.0
	0 glucuronosyltransferase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	2.1E-3	15.0E-3	1.8E+0	11.76 4.0
	0 Hypertrophic cardiomyopathy (HCM)	KEGG_01.03.2017	2.3E-3	15.0E-3	1.8E+0	7.23 6.0
	3 purine-containing compound catabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.5E-3 2.0E-3	15.0E-3	1.8E+0	8.62 5.0
	cap-independent translational initiation mucopolysaccharide metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10101 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.0E-3 3.5E-3	16.0E-3 17.0E-3	1.8E+0	18.75 3.0 5.83 7.0
	0 platelet formation	GO_BiologicalProcess-GOA_23.02.2017_10h01	2.9E-3	17.0E-3	1.8E+0	16.67 3.0
	2 second-messenger-mediated signaling	GO_BiologicalProcess-GOA_23.02.2017_10h01	3.6E-3	17.0E-3	1.8E+0	4.23 11.0
	6 aspartate family amino acid metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	3.3E-3	18.0E-3	1.7E+0	8.06 5.0
GO:0005414	4 Dilated cardiomyopathy	KEGG_01.03.2017	3.5E-3	18.0E-3	1.7E+0	6.67 6.0
GO:001489		GO_BiologicalProcess-GOA_23.02.2017_10h01	3.9E-3	18.0E-3	1.7E+0	15.00 3.0
	0 Retinol metabolism	KEGG_01.03.2017	4.1E-3	18.0E-3	1.7E+0	7.69 5.0
	9 divalent inorganic cation transmembrane transporter activity	GO_MolecularFunction-GOA_23.02.2017_10h01	3.1E-3	18.0E-3	1.7E+0	4.92 9.0
	4 Dilated cardiomyopathy 9 relaxation of cardiac muscle	KEGG_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01	3.5E-3 3.4E-3	18.0E-3 18.0E-3	1.7E+0	6.67 6.0 15.79 3.0
GO:0055113 GO:007187		GO BiologicalProcess-GOA 23.02.2017_10101 GO BiologicalProcess-GOA 23.02.2017 10h01	3.4E-3 3.4E-3	18.0E-3	1.7E+0	15.79 3.0
	9 divalent inorganic cation transmembrane transporter activity	GO_MolecularFunction-GOA_23.02.2017_10h01	3.1E-3	18.0E-3	1.7E+0	4.92 9.0
	2 glutathione peroxidase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	4.5E-3	19.0E-3	1.7E+0	14.29 3.0
GO:0048872	2 homeostasis of number of cells	GO_BiologicalProcess-GOA_23.02.2017_10h01	4.8E-3	19.0E-3	1.7E+0	4.07 11.0
	1 regulation of cellular pH	GO_BiologicalProcess-GOA_23.02.2017_10h01	4.5E-3	19.0E-3	1.7E+0	6.32 6.0
	6 response to topologically incorrect protein	GO_BiologicalProcess-GOA_23.02.2017_10h01	4.3E-3	19.0E-3	1.7E+0	4.69 9.0
	0 RORA activates gene expression	REACTOME_Pathways_01.03.2017	5.3E-3	19.0E-3	1.7E+0	7.25 5.0
	1 BMAL1:CLOCK,NPAS2 activates circadian gene expression 9 Circadian Clock	REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	5.3E-3 5.3E-3	19.0E-3 19.0E-3	1.7E+0	7.25 5.0
	0 Porphyrin and chlorophyll metabolism	REACTOME_Pathways_01.03.2017 KEGG_01.03.2017	4.7E-3	19.0E-3	1.7E+0	9.52 4.0
	2 cGMP-PKG signaling pathway	KEGG 01.03.2017	5.3E-3	19.0E-3	1.7E+0	4.91 8.0
	2 cGMP-PKG signaling pathway	KEGG_01.03.2017	5.3E-3	19.0E-3	1.7E+0	4.91 8.0
	5 methionine metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	5.2E-3	20.0E-3	1.7E+0	13.64 3.0
	1 skeletal muscle adaptation	GO_BiologicalProcess-GOA_23.02.2017_10h01	6.7E-3	20.0E-3	1.7E+0	12.50 3.0
	1 skeletal muscle adaptation	GO_BiologicalProcess-GOA_23.02.2017_10h01	6.7E-3	20.0E-3	1.7E+0	12.50 3.0
	4 Interactions of neurexins and neuroligins at synapses	REACTOME_Pathways_01.03.2017	5.9E-3	20.0E-3	1.7E+0	7.04 5.0
	5 Protein-protein interactions at synapses 7 multicellular organismal signaling	REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01	5.9E-3 6.5E-3	20.0E-3 20.0E-3	1.7E+0	7.04 5.0
	1 skeletal muscle adaptation	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	6.7E-3	20.0E-3	1.7E+0	12.50 3.0
	2 cAMP binding	GO_MolecularFunction-GOA_23.02.2017_10h01	6.7E-3	20.0E-3	1.7E+0	12.50 3.0
GO:004319	7 dendritic spine	GO_CellularComponent-GOA_23.02.2017_10h01	6.9E-3	21.0E-3	1.7E+0	5.15 7.0
	2 chondrocyte differentiation	GO_BiologicalProcess-GOA_23.02.2017_10h01	7.7E-3	21.0E-3	1.7E+0	5.66 6.0
	8 positive regulation of translational initiation	GO_BiologicalProcess-GOA_23.02.2017_10h01	5.9E-3	21.0E-3	1.7E+0	13.04 3.0
	6 kinetochore	GO_CellularComponent-GOA_23.02.2017_10h01	7.4E-3	21.0E-3	1.7E+0	5.07 7.0
	8 response to misfolded protein 6 chondroitin sulfate biosynthetic process	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	5.9E-3 7.5E-3	21.0E-3 21.0E-3	1.7E+0	13.04 3.0 12.00 3.0
	0 pigment metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	7.5E-3 7.1E-3	21.0E-3 21.0E-3	1.7E+0	6.76 5.0
	0 cellular response to retinoic acid	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	7.1E-3	21.0E-3	1.7E+0	6.76 5.0
	3 Drug metabolism	KEGG_01.03.2017	6.5E-3	21.0E-3	1.7E+0	8.70 4.0
	0 pigment metabolic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	7.1E-3	21.0E-3	1.7E+0	6.76 5.0
	2 Arrhythmogenic right ventricular cardiomyopathy (ARVC)	KEGG_01.03.2017	6.3E-3	21.0E-3	1.7E+0	6.94 5.0
GO:0005412					4 7	4.76 8.0
GO:0005412 GO:001972	2 calcium-mediated signaling	GO_BiologicalProcess-GOA_23.02.2017_10h01	6.3E-3	21.0E-3	1.7E+0	
GO:0005412 GO:001972 GO:000493		GO_BiologicalProcess-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01	6.3E-3 8.1E-3 8.3E-3	21.0E-3 22.0E-3 22.0E-3	1.7E+0	4.70 8.0 5.61 6.0 4.55 8.0

GO:0004260 C	Cardiac muscle contraction	KEGG_01.03.2017	8.8E-3	23.0E-3	1.6E+0	6.41 5.00
GO:0008287 p	protein serine/threonine phosphatase complex	GO_CellularComponent-GOA_23.02.2017_10h01	9.4E-3	23.0E-3	1.6E+0	7.84 4.00
	Ascorbate and aldarate metabolism	KEGG_01.03.2017	9.3E-3	23.0E-3	1.6E+0	11.11 3.00
	regulation of postsynaptic membrane potential	GO_BiologicalProcess-GOA_23.02.2017_10h01	9.0E-3	23.0E-3	1.6E+0	4.90 7.00
	Cardiac muscle contraction	KEGG_01.03.2017	8.8E-3	23.0E-3	1.6E+0	6.41 5.00
	protein serine/threonine phosphatase complex	GO_CellularComponent-GOA_23.02.2017_10h01	9.4E-3	23.0E-3	1.6E+0	7.84 4.00
	cardiac conduction	GO_BiologicalProcess-GOA_23.02.2017_10h01	9.0E-3	23.0E-3	1.6E+0	4.90 7.00
	regulation of cardiac muscle contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_MolecularFunction-GOA_23.02.2017_10h01	8.8E-3 10.0E-3	23.0E-3 24.0E-3	1.6E+0 1.6E+0	6.41 5.00 7.69 4.00
	excitatory extracellular ligand-gated ion channel activity Mitochondrial biogenesis	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3 24.0E-3	1.6E+0	7.69 4.00
	Vitochondrial transcription initiation	REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3 24.0E-3	1.6E+0	7.69 4.00
	Transcriptional activation of mitochondrial biogenesis	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
	Transcription from mitochondrial promoters	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
	Mitochondrial biogenesis	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
	Mitochondrial transcription initiation	REACTOME Pathways 01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
GO:0000673 T	Transcriptional activation of mitochondrial biogenesis	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
GO:0001680 T	Transcription from mitochondrial promoters	REACTOME_Pathways_01.03.2017	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
GO:0008194 L	JDP-glycosyltransferase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	10.0E-3	24.0E-3	1.6E+0	4.76 7.00
	igand-gated ion channel activity	GO_MolecularFunction-GOA_23.02.2017_10h01	10.0E-3	24.0E-3	1.6E+0	4.79 7.00
	excitatory extracellular ligand-gated ion channel activity	GO_MolecularFunction-GOA_23.02.2017_10h01	10.0E-3	24.0E-3	1.6E+0	7.69 4.00
	Glutathione metabolism	KEGG_01.03.2017	11.0E-3	26.0E-3	1.6E+0	7.41 4.00
	actin-mediated cell contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01	11.0E-3	26.0E-3	1.6E+0	5.17 6.00
	actin-mediated cell contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01	11.0E-3 12.0E-3	26.0E-3	1.6E+0	5.17 6.00 4.23 8.00
	protein methylation Regulation of TP53 Activity through Acetylation	GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017	12.0E-3 12.0E-3	27.0E-3 27.0E-3	1.6E+0 1.6E+0	4.23 8.00
	cellular response to topologically incorrect protein	GO_BiologicalProcess-GOA_23.02.2017_10h01	12.0E-3	27.0E-3 27.0E-3	1.6E+0	4.58 7.00
	Chondroitin sulfate biosynthesis	REACTOME Pathways 01.03.2017	12.0E-3	27.0E-3 27.0E-3	1.6E+0	10.00 3.00
	Dermatan sulfate biosynthesis	REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	12.0E-3	27.0E-3 27.0E-3	1.6E+0	10.00 3.00
	CS/DS degradation	REACTOME_Pathways_01.03.2017	12.0E-3	27.0E-3	1.6E+0	10.00 3.00
	Defective CHST3 causes SEDCJD	REACTOME_Pathways_01.03.2017	12.0E-3	27.0E-3	1.6E+0	10.00 3.00
	Defective CHST14 causes EDS, musculocontractural type	REACTOME_Pathways_01.03.2017	12.0E-3	27.0E-3	1.6E+0	10.00 3.00
	Defective CHSY1 causes TPBS	REACTOME_Pathways_01.03.2017	12.0E-3	27.0E-3	1.6E+0	10.00 3.00
GO:0001964 s	startle response	GO_BiologicalProcess-GOA_23.02.2017_10h01	12.0E-3	27.0E-3	1.6E+0	10.00 3.00
	cell communication involved in cardiac conduction	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	27.0E-3	1.6E+0	7.14 4.00
	cell communication involved in cardiac conduction	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	27.0E-3	1.6E+0	7.14 4.00
	positive regulation of interleukin-10 production	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	28.0E-3	1.6E+0	9.68 3.00
	positive regulation of interleukin-10 production	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	28.0E-3	1.6E+0	9.68 3.00
	positive regulation of interleukin-10 production	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	28.0E-3	1.6E+0	9.68 3.00
	regulation of muscle contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01	13.0E-3	28.0E-3	1.6E+0	4.52 7.00
	cartilage development	GO_BiologicalProcess-GOA_23.02.2017_10h01	15.0E-3	30.0E-3	1.5E+0	4.08 8.00
	B cell homeostasis	GO_BiologicalProcess-GOA_23.02.2017_10h01	14.0E-3	30.0E-3	1.5E+0	9.38 3.00
	Golgi Associated Vesicle Biogenesis	REACTOME_Pathways_01.03.2017	16.0E-3	30.0E-3	1.5E+0	4.80 6.00 4.80 6.00
	Signaling by BRAF and RAF fusions	REACTOME_Pathways_01.03.2017	16.0E-3 16.0E-3	30.0E-3 30.0E-3	1.5E+0 1.5E+0	
	Dncogenic MAPK signaling ntegral component of Golgi membrane	REACTOME_Pathways_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01	14.0E-3	30.0E-3	1.5E+0	4.80 6.00 6.90 4.00
	positive regulation of interleukin-2 production	GO_BiologicalProcess-GOA_23.02.2017_10h01	14.0E-3	30.0E-3	1.5E+0	9.38 3.00
GO:0004110		KEGG 01.03.2017	14.0E-3 15.0E-3	31.0E-3	1.5E+0	4.84 6.00
	7-methylguanosine mRNA capping	GO_BiologicalProcess-GOA_23.02.2017_10h01	16.0E-3	31.0E-3	1.5E+0	9.09 3.00
	Rev-mediated nuclear export of HIV RNA	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	Export of Viral Ribonucleoproteins from Nucleus	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	NEP/NS2 Interacts with the Cellular Export Machinery	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
GO:0000417 li	nteractions of Rev with host cellular proteins	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	Downregulation of TGF-beta receptor signaling	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	TGF-beta receptor signaling activates SMADs	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	HuR (ELAVL1) binds and stabilizes mRNA	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	Cyclin A/B1 associated events during G2/M transition	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	5.49 5.00
	Unblocking of NMDA receptor, glutamate binding and activation	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	9.09 3.00
	CREB phosphorylation through the activation of CaMKII	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	9.09 3.00
	SALM protein interactions at the synapses	REACTOME_Pathways_01.03.2017	16.0E-3	31.0E-3	1.5E+0	9.09 3.00 4.02 8.00
	voltage-gated ion channel activity voltage-gated ion channel activity	GO_MolecularFunction-GOA_23.02.2017_10h01	16.0E-3 16.0E-3	31.0E-3 31.0E-3	1.5E+0 1.5E+0	4.02 8.00
	cellular response to interleukin-6	GO_MolecularFunction-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	17.0E-3	32.0E-3	1.5E+0	8.82 3.00
	mport across plasma membrane	GO_BiologicalProcess-GOA_23.02.2017_10101 GO_BiologicalProcess-GOA_23.02.2017_10101	17.0E-3	32.0E-3	1.5E+0	8.82 3.00
	pehavioral fear response	GO_BiologicalProcess-GOA_23.02.2017_10h01	17.0E-3	32.0E-3	1.5E+0	8.82 3.00
	B cell proliferation	GO_BiologicalProcess-GOA_23.02.2017_10h01	17.0E-3	32.0E-3	1.5E+0	5.38 5.00
	Pentose and glucuronate interconversions	KEGG_01.03.2017	17.0E-3	32.0E-3	1.5E+0	8.82 3.00
GO:0043620 r	regulation of DNA-templated transcription in response to stress	GO_BiologicalProcess-GOA_23.02.2017_10h01	18.0E-3	33.0E-3	1.5E+0	4.69 6.00
n	negative regulation of adaptive immune response based on somatic					
	recombination of immune receptors built from immunoglobulin		10.07	00.07		
GO:0002823 s	superfamily domains	GO_BiologicalProcess-GOA_23.02.2017_10h01	19.0E-3	33.0E-3	1.5E+0	8.57 3.00
GU:0008376 a	aceiyigaiactosaminyitransterase activity	GO_iviolecular-unction-GOA_23.02.2017_10h01	19.0E-3	33.0E-3	1.5E+0	8.57 3.00
	negative regulation of immune response	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	19.0E-3 19.0E-3	34.0E-3 34.0E-3	1.5E+0 1.5E+0	4.62 6.00 4.62 6.00
	negative regulation of immune response Circadian entrainment	GO_BiologicalProcess-GOA_23.02.2017_10n01 KEGG_01.03.2017	19.0E-3 20.0E-3	34.0E-3 35.0E-3	1.5E+0 1.5E+0	4.62 6.00 5.21 5.00
	Circadian entrainment	KEGG_01.03.2017 KEGG_01.03.2017	20.0E-3 20.0E-3	35.0E-3	1.5E+0	5.21 5.00
GO:0006901 v		GO_BiologicalProcess-GOA_23.02.2017_10h01	20.0E-3 21.0E-3	36.0E-3	1.4E+0	6.15 4.00
	Renal cell carcinoma	KEGG 01.03.2017	21.0E-3	36.0E-3	1.4E+0	6.15 4.00
	positive regulation of interferon-gamma production	GO_BiologicalProcess-GOA_23.02.2017_10h01	21.0E-3	36.0E-3	1.4E+0	6.15 4.00
GO:0015698 ir	norganic anion transport	GO_BiologicalProcess-GOA_23.02.2017_10h01	21.0E-3	36.0E-3	1.4E+0	4.12 7.00
GO:0030968 e	endoplasmic reticulum unfolded protein response	GO_BiologicalProcess-GOA_23.02.2017_10h01	21.0E-3	36.0E-3	1.4E+0	4.55 6.00
	positive regulation of interferon-gamma production	GO_BiologicalProcess-GOA_23.02.2017_10h01	21.0E-3	36.0E-3	1.4E+0	6.15 4.00
GO:0001756 s		GO_BiologicalProcess-GOA_23.02.2017_10h01	24.0E-3	39.0E-3	1.4E+0	5.88 4.00
	vesicle targeting, to, from or within Golgi	GO_BiologicalProcess-GOA_23.02.2017_10h01	24.0E-3	39.0E-3	1.4E+0	5.88 4.00
	response to unfolded protein	GO_BiologicalProcess-GOA_23.02.2017_10h01	24.0E-3	39.0E-3	1.4E+0	4.02 7.00
	positive regulation of muscle tissue development	GO_BiologicalProcess-GOA_23.02.2017_10h01	24.0E-3	39.0E-3	1.4E+0	5.88 4.00
GO:0004360 A	Axon guidance norganic anion transmembrane transporter activity	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01	24.0E-3 24.0E-3	40.0E-3 40.0E-3	1.4E+0 1.4E+0	4.00 7.00 4.38 6.00
	norganic anion transmembrane transporter activity	GO_MolecularFunction-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	24.0E-3 27.0E-3	40.0E-3 42.0E-3	1.4E+0 1.4E+0	4.38 6.00
	Drug metabolism	GO_BiologicalProcess-GOA_23.02.2017_10n01 KEGG 01.03.2017	27.0E-3 27.0E-3	42.0E-3 42.0E-3	1.4E+0 1.4E+0	4.81 5.00 5.71 4.00
	Nicotine addiction	KEGG_01.03.2017 KEGG_01.03.2017	27.0E-3 27.0E-3	42.0E-3 42.0E-3	1.4E+0 1.4E+0	7.50 3.00
	chloride transport	GO BiologicalProcess-GOA 23.02.2017 10h01	27.0E-3 27.0E-3	42.0E-3 42.0E-3	1.4E+0	4.81 5.00
	peptidyl-lysine trimethylation	GO_BiologicalProcess-GOA_23.02.2017_10h01	28.0E-3	44.0E-3	1.4E+0	7.32 3.00
	Post NMDA receptor activation events	REACTOME_Pathways_01.03.2017	28.0E-3	44.0E-3	1.4E+0	7.32 3.00
	CREB phosphorylation through the activation of Ras	REACTOME_Pathways_01.03.2017	28.0E-3	44.0E-3	1.4E+0	7.32 3.00
	Activation of NMDA receptor upon glutamate binding and postsynaptic				1	
GO:0001085 e	events	REACTOME_Pathways_01.03.2017	28.0E-3	44.0E-3	1.4E+0	7.32 3.00
GO:0001085 e GO:0001086 F	events Ras activation uopn Ca2+ infux through NMDA receptor	REACTOME_Pathways_01.03.2017	28.0E-3	44.0E-3	1.4E+0	7.32 3.00
GO:0001085 e GO:0001086 F	events					

GO:0000777 condensed chromosome kinetochore						
	GO_CellularComponent-GOA_23.02.2017_10h01	30.0E-3	46.0E-3	1.3E+0	4.67	5.00
GO:0000218 Signaling by NOTCH	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000549 Signaling by NOTCH1	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000550 Signaling by NOTCH2	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000654 Activated NOTCH1 Transmits Signal to the Nucleus	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000743 Signaling by NOTCH1 PEST Domain Mutants in Cancer	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000744 Signaling by NOTCH1 in Cancer	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000746 Constitutive Signaling by NOTCH1 PEST Domain Mutants	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000756 Signaling by NOTCH1 HD Domain Mutants in Cancer	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000757 Constitutive Signaling by NOTCH1 HD Domain Mutants	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000765 Signaling by NOTCH1 HD+PEST Domain Mutants in Cancer	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000766 Constitutive Signaling by NOTCH1 HD+PEST Domain Mutants	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:0000768 NOTCH2 Activation and Transmission of Signal to the Nucleus	REACTOME_Pathways_01.03.2017	31.0E-3	46.0E-3	1.3E+0	4.63	5.00
GO:1903779 regulation of cardiac conduction	GO_BiologicalProcess-GOA_23.02.2017_10h01	31.0E-3	46.0E-3	1.3E+0	5.48	4.00
GO:0051489 regulation of filopodium assembly	GO_BiologicalProcess-GOA_23.02.2017_10h01	32.0E-3	47.0E-3	1.3E+0	6.98	3.00
GO:0006904 vesicle docking involved in exocytosis	GO_BiologicalProcess-GOA_23.02.2017_10h01	32.0E-3	47.0E-3	1.3E+0	6.98	3.00
GO:0008135 translation factor activity, RNA binding	GO MolecularFunction-GOA 23.02.2017 10h01	32.0E-3	47.0E-3	1.3E+0	4.59	5.00
GO:0006661 phosphatidylinositol biosynthetic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	34.0E-3	48.0E-3	1.3E+0	4.05	6.00
GO:0021549 cerebellum development	GO_BiologicalProcess-GOA_23.02.2017_10h01	35.0E-3	48.0E-3	1.3E+0	4.50	5.00
GO:0021545 Cerebendin development GO:0031571 mitotic G1 DNA damage checkpoint	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	35.0E-3	48.0E-3	1.3E+0	5.26	4.00
GO:0048678 response to axon injury	GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	35.0E-3	48.0E-3	1.3E+0	5.26	4.00
	GO Biological Process-GOA 23.02.2017 10h01	33.0E-3		1.3E+0		
GO:0051304 chromosome separation		33.0E-3 34.0E-3	48.0E-3 48.0E-3	1.3E+0	5.33	4.00
GO:0004973 Carbohydrate digestion and absorption	KEGG_01.03.2017				6.82	
GO:0000309 Complement cascade	REACTOME_Pathways_01.03.2017	34.0E-3	48.0E-3	1.3E+0	6.82	3.00
GO:0000310 Lectin pathway of complement activation	REACTOME_Pathways_01.03.2017	34.0E-3	48.0E-3	1.3E+0	6.82	3.00
GO:0000311 Initial triggering of complement	REACTOME_Pathways_01.03.2017	34.0E-3	48.0E-3	1.3E+0	6.82	3.00
GO:0000313 Creation of C4 and C2 activators	REACTOME_Pathways_01.03.2017	34.0E-3	48.0E-3	1.3E+0	6.82	3.00
Ficolins bind to repetitive carbohydrate structures on the target cell						
GO:0000759 surface	REACTOME_Pathways_01.03.2017	34.0E-3	48.0E-3	1.3E+0	6.82	3.00
GO:0003022 Basal transcription factors	KEGG_01.03.2017	36.0E-3	49.0E-3	1.3E+0	6.67	3.00
GO:0033555 multicellular organismal response to stress	GO_BiologicalProcess-GOA_23.02.2017_10h01	36.0E-3	49.0E-3	1.3E+0	5.19	4.00
GO:0032580 Golgi cisterna membrane	GO_CellularComponent-GOA_23.02.2017_10h01	36.0E-3	49.0E-3	1.3E+0	5.19	4.0
GO:0000493 Pre-NOTCH Processing in Golgi	REACTOME_Pathways_01.03.2017	36.0E-3	49.0E-3	1.3E+0	6.67	3.0
GO:0000494 Pre-NOTCH Expression and Processing	REACTOME_Pathways_01.03.2017	36.0E-3	49.0E-3	1.3E+0	6.67	3.00
NEURON (PGC2 + CLOZUK)						
NEOKON (FGCZ + CEOZOK)			Term			
			PValue			
			Corrected			
			with		%	
		Term	Benjamini-	Nea loa	Associated	Nr.
GOID GOTerm	Ontology Source	PValue	Hochberg	(FĎR)		Genes
GO:0097346 INO80-type complex	GO_CellularComponent-GOA_23.02.2017_10h01	830.0E-9	52.0E-6	4.3E+0	27.27	6.00
GO:0070603 SWI/SNF superfamily-type complex	GO CellularComponent-GOA 23.02.2017 10h01	520.0E-9	66.0E-6	4.2E+0	12.35	10.00
GO:0044297 cell body	GO_CellularComponent-GOA_23.02.2017_10h01	5.2E-6	220.0E-6	3.7E+0	4.41	24.00
GO:0006338 chromatin remodeling	GO_BiologicalProcess-GOA_23.02.2017_10h01	8.2E-6	260.0E-6	3.6E+0	7.50	12.00
GO:0098794 postsynapse	GO_CellularComponent-GOA_23.02.2017_10h01	21.0E-6	380.0E-6	3.4E+0	4.55	20.00
GO:0043025 neuronal cell body	GO_CellularComponent-GOA_23.02.2017_10h01	21.0E-6	450.0E-6	3.3E+0	4.40	21.00
				3.3E+0		
GO:0030425 dendrite	GO_CellularComponent-GOA_23.02.2017_10h01	19.0E-6	490.0E-6		4.30	22.00
					7.00	
GO:0004728 Dopaminergic synapse	KEGG_01.03.2017	37.0E-6	600.0E-6	3.2E+0	7.69	10.00
GO:0004728 Dopaminergic synapse	KEGG_01.03.2017	37.0E-6	600.0E-6	3.2E+0 3.2E+0	7.69	10.00
GO:0004728 Dopaminergic synapse GO:0042578 phosphoric ester hydrolase activity	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01	37.0E-6 45.0E-6	600.0E-6 630.0E-6	3.2E+0 3.2E+0 3.2E+0	7.69 4.62	10.00
GC:0004728 Dopaminergic synapse GC:0042578 phosphoric ester hydrolase activity GC:0031011 [no80 complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6	600.0E-6 630.0E-6 880.0E-6	3.2E+0 3.2E+0 3.2E+0 3.1E+0	7.69 4.62 26.67	10.00 18.00 4.00
GC:0004728 Dopaminergic synapse GO:0042578 phosphoric ester hydrolase activity GO:0031011 Ino80 complex GO:00332526 response to retinoic acid	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6 92.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0	7.69 4.62 26.67 7.69	10.00 18.00 4.00 9.00
GO:0004728 Dopaminergic synapse GO:0042578 phosphoric ester hydrolase activity GO:0031011 lino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6 92.0E-6 150.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0	7.69 4.62 26.67 7.69 9.46	10.00 18.00 4.00 9.00 7.00
GC:0004728 Dopaminergic synapse GO:0042578 phosphoric ester hydrolase activity GO:0031011 Ino80 complex GO:00332526 response to retinoic acid	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6 92.0E-6 150.0E-6 290.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0	7.69 4.62 26.67 7.69 9.46 13.16	10.00 18.00 4.00 9.00 7.00
GO:0004728 Dopaminergic synapse GO:0042578 phosphoric ester hydrolase activity GO:0031011 lino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6 92.0E-6 150.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0	7.69 4.62 26.67 7.69 9.46	10.00 18.00 4.00 9.00 7.00 5.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 69.0E-6 92.0E-6 150.0E-6 290.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0 2.6E+0	7.69 4.62 26.67 7.69 9.46 13.16	10.00 18.00 9.00 7.00 5.00 5.00
GC:0004728 Dopaminergic synapse GO:00472578 phosphoric ester hydrolase activity GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 92.0E-6 150.0E-6 290.0E-6 290.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 2.8E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0 2.6E+0 2.6E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16	10.00 18.00 9.00 7.00 5.00 5.00 11.00
GC:0004728 Dopaminergic synapse GO:004728 phosphoric ester hydrolase activity GO:003101 Ino80 complex GO:00332526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:009122 cellular response to acid chemical	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 92.0E-6 150.0E-6 290.0E-6 290.0E-6 370.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 2.8E-3 3.4E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0 2.6E+0 2.6E+0 2.5E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39	10.00 18.00 9.00 7.00 5.00 5.00 11.00 5.00
GC:0004728 Dopaminergic synapse GO:004728 phosphoric ester hydrolase activity GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0071229 cellular response to acid chemical GO:0005691 voltage-gated calcium channel complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 KEGG_010.30.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 150.0E-6 290.0E-6 370.0E-6 420.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 2.8E-3 3.4E-3 3.5E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20	10.00 18.00 9.00 7.00 5.00 5.00 11.00 5.00 5.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:007302 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:007122 cellular response to acid chemical GO:0005891 voltage-gated calcium channel complex GO:000581 voltage-gated calcium channel complex GO:000581 Maphetamine addiction	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 GO_CellularComponent-GOA_3.02.2017_10h01 KEGG_010.30.2017	37.0E-6 45.0E-6 69.0E-6 92.0E-6 150.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 420.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 3.4E-3 3.4E-3 3.5E-3 3.5E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.6E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20 12.20	10.00 18.00 9.00 7.00 5.00 11.00 5.00 5.00 6.00
GC:0004728 Dopaminergic synapse GO:004728 phosphoric ester hydrolase activity GO:0031011 Ino80 complex GO:0031011 Ino80 complex GO:00371300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0071229 cellular response to acid chemical GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.20.22017_10h01 GO_CellularComponent-GOA_23.20.22017_10h01 GO_CellularComponent-GOA_23.20.22017_10h01 GO_CellularComponent-GOA_23.20.22017_10h01	37.0E-6 45.0E-6 99.0E-6 99.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 420.0E-6 660.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 3.4E-3 3.4E-3 3.5E-3 3.5E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.6E+0 2.6E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0 2.4E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20 12.20 12.20 8.82	10.00 18.00 4.00 9.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:003101 Ino80 complex GO:007300 cellular response to retinoic acid GO:007300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:007122 cellular response to acid chemical GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017	37.0E-6 45.0E-6 99.0E-6 92.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 420.0E-6 660.0E-6 630.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 3.4E-3 3.5E-3 3.5E-3 3.5E-3 4.2E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.0E+0 2.0E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0 2.5E+0 2.4E+0 2.4E+0	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20 12.20 8.82 5.07	10.00 18.00 9.00 5.00 5.00 11.00 5.00 6.00 11.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:003101 Ino80 complex GO:0032526 response to retinoic acid GO:00932526 ruclear transcriptional repressor complex GO:0093058 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0005691 voltage-gated calcium channel complex GO:0005691 voltage-gated calcium channel complex GO:0005691 voltage-gated calcium channel complex GO:0005691 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0014069 postsynaptic density	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 660.0E-6 660.0E-6 660.0E-6 630.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 3.4E-3 3.5E-3 3.5E-3 3.5E-3 4.2E-3 4.2E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.8E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0 2.4E+0 2.4E+0	7.69 4.62 26.67 7.69 9.46 13.16 5.39 12.20 12.20 12.20 8.82 5.07 8.82 5.07	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 6.00 11.00
GC:0004728 Dopaminergic synapse GO:004728 phosphoric ester hydrolase activity GC:0031011 Ino80 complex GC:0031012 Ino80 complex GC:0032526 response to retinoic acid GC:0090568 nuclear transcriptional repressor complex GC:0090568 nuclear transcriptional repressor complex GC:0070568 nuclear transcriptional repressor complex GC:0071229 cellular response to acid chemical GC:0005891 voltage-gated calcium channel complex GC:0005031 voltage-gated calcium channel complex GC:0005031 voltage-gated calcium channel complex GC:0005031 voltage-gated calcium channel complex GC:0005031 Amphetamine addiction GC:0014069 postsynaptic density GC:0004052 AMPK signaling pathway	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 99.0E-6 150.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 420.0E-6 660.0E-6 660.0E-6 630.0E-6 630.0E-6 630.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 2.8E-3 3.4E-3 3.5E-3 3.5E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0 2.4E+0 2.4E+0 2.4E+0 2.4E+0 2.4E+0 2.4E+0	7.69 4.62 26.67 7.69 9.46 13.16 5.39 12.20 12.20 12.20 8.82 5.07 8.82 5.07 6.61	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 6.00 11.00 8.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:00332526 response to retinoic acid GO:007300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:007129 cellular response to acid chemical GO:0005681 voltage-gated calcium channel complex GO:0005681 voltage-gated calcium channel complex GO:0005631 Amphetamine addiction GO:0005631 Amphetamine addiction GO:0005631 Amphetamine addiction GO:0005631 Amphetamine addiction GO:0005631 Amphetamine addiction GO:0005632 Amphetamine addiction GO:0005632 Amphetamine addiction GO:0005632 Amphetamine addiction GO:0005632 Amphetamine addiction	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 KEGG_01.03.2017	37.0E-6 45.0E-6 99.0E-6 150.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 660.0E-6 660.0E-6 660.0E-6 660.0E-6 630.0E-6 620.0E-6 620.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 3.4E-3 3.5E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.4E-3	3.2E+C 3.2E+C 3.2E+C 3.1E+C 2.8E+C 2.6E+C 2.6E+C 2.6E+C 2.5E+C 2.5E+C 2.4E+C 2.4E+C 2.4E+C 2.4E+C 2.4E+C 2.4E+C	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20 12.20 8.82 5.07 8.82 5.07 6.61 6.61	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 6.00 11.00 8.00 8.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:0031011 Ino80 complex GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0014069 postsynaptic density GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 660.0E-6 660.0E-6 660.0E-6 630.0E-6 620.0E-6 620.0E-6 590.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 3.4E-3 3.5E-3 3.5E-3 4.2E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 2.8E+0 2.6E+0 2.6E+0 2.5E+0 2.5E+0 2.5E+0 2.4E+0 2.	7.69 4.62 26.67 7.69 9.46 13.16 13.16 5.39 12.20 12.20 8.82 5.07 8.82 5.07 6.61 11.36	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 6.00 11.00 8.00 8.00 5.00
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:003101 Ino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:007129 cellular response to acid chemical GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0004169 postsynaptic density GO:0004162 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:00041720 Long-term potentiation	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 370.0E-6 420.0E-6 420.0E-6 660.0E-6 630.0E-6 630.0E-6 620.0E-	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.0E-3 1.0E-3 2.8E-3 2.8E-3 3.3.EE-3 3.3.EE-3 3.3.EE-3 3.3.EE-3 4.2E-3 4.2E-3 4.2E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3	3.2E+0 3.2E+(-0 3.2E+(-0 3.2E+(-0 3.1E+(-0 2.8E+(-0 2.6E+(-0 2.6E+(-0 2.6E+(-0 2.6E+(-0 2.6E+(-0 2.6E+(-0 2.6E+(-0 2.4E+(-0 2.4E+(-0 2.4E+(-0 2.4E+(-0))))))))))))))))))))))))))))))))))))	7.69 4.62 26.67 7.69 9.46 13.16 5.39 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 6.61 6.61 6.61 11.36 8.82	10.00 18.00 9.00 7.00 5.00 5.00 6.00 11.00 6.00 11.00 8.00 8.00 6.00 6.00 6.00 11.00 8.00 8.00 6
GC:0004728 Dopaminergic synapse GC:004728 Dopaminergic synapse GC:003101 Ino80 complex GC:0032526 response to retinoic acid GC:0032526 response to retinoic acid GC:009768 nuclear transcriptional repressor complex GC:009768 nuclear transcriptional repressor complex GC:009768 nuclear transcriptional repressor complex GC:007122 cellular response to acid chemical GC:0005891 voltage-gated calcium channel complex GC:0005891 voltage-gated calcium channel complex GC:0005891 voltage-gated calcium channel complex GC:0005031 Amphetamine addiction GC:0014069 postsynaptic density GC:0004152 AmPk signaling pathway GC:0004152 AMPK signaling pathway GC:0004720 Long-term potentiation	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 420.0E-6 660.0E-6 660.0E-6 660.0E-6 630.0E-6 630.0E-6 620.0E-6 620.0E-6 620.0E-6 610.0E-6 610.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.0E-3 2.8E-3 3.4E-3 3.4E-3 3.5E-3 4.2E-3 4.2E-3 4.2E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3	3.2E+0 3.2E+0 3.2E+0 3.1E+0 3.0E+0 2.8E+0 2.6E+0 2.6E+0 2.4E+0 2.	7.69 4.62 26.67 7.69 9.46 13.16 13.16 13.16 13.16 13.10 12.20 12.20 12.20 12.20 12.20 12.20 12.20 6.61 11.36 6.61 11.36 8.896 8.896	10.00 18.00 9.00 7.00 5.00 5.00 6.00 11.00 6.00 11.00 8.00 8.00 6.
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:0031011 Ino80 complex GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0014069 postsynaptic density GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 Carbohydrate digestion and absorption GO:004720 Long-term potentiation GO:0004720 Long-term potentiation GO:0004730 Carbohydrate digestion and absorption	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_GellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_DiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 290.0E-6 420.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 610.0E-6 610.0E-6 610.0E-6 610.0E-6 610.0E-6	600.0E-6 630.0E-6 880.0E-6 1.0E-3 1.6E-3 2.8E-3 3.4E-3 3.5E-3 4.2E-3 4.2E-3 4.2E-3 4.4E-3 4.4E-3 4.4E-3 4.6E-3 4.6E-3 4.6E-3	3.2E+0 3.2E+0 3.2E+0 3.2E+0 3.0E+0 2.8E+0 2.6E+0 2.5E+0 2.5E+0 2.4E+0 2.3E+0 2.	7,69 4,62 26,67 7,69 9,46 13,16 13,16 13,16 13,16 13,16 13,16 13,16 12,20 12,20 12,20 12,20 12,20 12,20 12,20 12,20 12,20 6,61 11,36 8,96 8,96 8,96 11,36 11,36 11,37 11	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 8.00 8.00 5.00 6.00 5.00 5.00 6.00 5.
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:003101 Ino80 complex GO:003101 Ino80 complex GO:0032526 response to retinoic acid GO:0071300 cellular response to retinoic acid GO:0090568 nuclear transcriptional repressor complex GO:0071220 cellular response to racid chemical GO:0071220 cellular response to racid chemical GO:0005891 voltage-gated calcium channel complex GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0005031 Amphetamine addiction GO:0004150 Amphetamine addiction GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:000473 Carbohydrate digestion and absorption GO:004702 Long-term potentiation GO:0004702 Long-term potentiation GO:0004703 Carbohydrate digestion and absorption GO:0004703 Carbohydrate digestion and absorption GO:0004703 Long-term potentiation	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017	37.0E-6 45.0E-6 92.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 420.0E-6 660.0E-6 660.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 610.0E-6 590.0E-6	600.0E-6 630.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 2.8E-3 2.8E-3 3.3.EE-3 3.3.EE-3 4.2E-3 4.2E-3 4.2E-3 4.2E-3 4.4E-3 4.4E-3 4.4E-3 4.4E-3 4.6E-3 4.6E-3 4.6E-3	3.2E+0 3.2E+0 3.2E+0 3.2E+0 3.0E+0 3.0E+0 2.8E+0 2.6E+0 2.5E+0 2.5E+0 2.4E+0 2.	7.69 4.62 26.67 7.69 9.464 13.16 13.16 13.16 5.39 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 12.20 6.61 11.36 6.61 11.36 8.96 8.96 8.96 8.96 8.96 8.96 8.96 8.9	10.00 18.00 9.00 7.00 5.00 11.00 5.00 6.00 11.00 8.00 8.00 6.
GC:0004728 Dopaminergic synapse GO:004728 Dopaminergic synapse GO:0031011 Ino80 complex GO:0031011 Ino80 complex GO:0032526 response to retinoic acid GO:00932526 response to retinoic acid GO:00932526 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:0090568 nuclear transcriptional repressor complex GO:007122 cellular response to acid chemical GO:0005891 voltage-gated calcium channel complex GO:0005031 Amphetamine addiction GO:0014069 postsynaptic density GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 AMPK signaling pathway GO:0004152 Competerm potentiation GO:0004720 Long-term potentiation GO:0004720 Long-term potentiation GO:0004737 Carbohydrate digestion and absorption GO:0004737 Carbohydrate digestion and absorption GO:0004737 Carbohydrate digestion and absorption GO:0004737 Carbohydrate digestion and absorption GO:0004731 histone deacetylase complex	KEGG_01.03.2017 GO_MolecularFunction-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_EllularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 GO_CellularComponent-GOA_23.02.2017_10h01 KEGG_01.03.2017 CellularComponent-GOA_23.02.2017_10h01 GO_CellularComponent-GOA_23.02.2017_10h01	37.0E-6 45.0E-6 92.0E-6 92.0E-6 290.0E-6 290.0E-6 290.0E-6 420.0E-6 420.0E-6 630.0E-6 630.0E-6 630.0E-6 630.0E-6 590.0E-6 500.0E-	600.0E-6 630.0E-6 880.0E-6 1.0E-3 2.8E-3 2.8E-3 3.5E-3 3.5E-3 3.5E-3 4.2	3.2E+6 3.2E+6 3.2E+6 3.1E+0 3.0E+6 2.6E+6 2.6E+6 2.6E+6 2.6E+6 2.6E+6 2.5E+6 2.4E+6 2.	7,69 4,62 26,67 7,69 9,46 (13,16 (13,16 (13,16 (13,16 (13,16 (13,16 (13,16) 8,82 5,07 (6,61 (11,36) 8,82 5,07 (6,61) (11,36) 8,82 (6,61) (11,36) 8,82 (6,61) (11,36) (10.00 18.00 4.00 9.00 5.00 5.00 5.00 5.00 6.00 11.00 6.00 11.00 8.00 6.
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GO:1903169 regulation of calcium ion transmembrane transport GO_BiologicalProcess-GOA_23.02.2017_10h01 17.0E-3 30.0E-3 1.5E+0 4.55 GO:0005143 African trypanosomiasis KEGG 01.03.2017 17.0E-3 30.0E-3 1.5E+0 4.65 GO:0001472 hormone receptor binding GO. MolecularFunction-GOA_23.02.2017_10h01 19.0E-3 31.0E-3 1.5E+0 4.05 GO:0000970 ArninoacyI-tRNA biosynthesis KEGG 01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.05 GO:0001425 DNA Damage Recognition in GG-NER REACTOME Pathways 01.03.2017 21.0E-3 32.0E-3 1.5E+0 4.00 GO:0000230 Purine metabolism KEGG 01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0000230 Purine metabolism KEGG 01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.90 GO:0004603 cardiac muscle cell contraction GO_DioigicalProcess-GOA_23.02.2017_10h01 21.0E-3 33.0E-3								3.00
GC:0005143 African trypanosomiasis KEGG_0103.2017 17.0E-3 30.0E-3 1.5E+0 8.57 GO:0005143 Aminacot/HRNA biosynthesis KEGG_01.03.2017 19.0E-3 31.0E-3 1.5E+0 4.05 GO:0000200 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0000200 DNA branage Recognition in GG-NER REACTOME_Pathways_01.03.2017 21.0E-3 32.0E-3 1.5E+0 4.00 GO:0000200 DNA methylation GO_BiologicalProcess-GOA_23.02.2017_10h01 19.0E-3 32.0E-3 1.5E+0 6.06 GO:0000230 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0000230 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723								4.00
GO:0051427 hormone receptor binding GO. MolecularFunction-GOA_23.02.2017_10h01 19.0E-3 31.0E-3 15.E+0 4.05 GO:000070 AminoacyI-IRNA biosynthesis KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 6.06 GO:0000230 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0001425 DNA methylation GO_BiologicalProcess-GOA_23.02.2017_101 19.0E-3 32.0E-3 1.5E+0 4.00 GO:0000230 Purine metabolism KEGG_01.03.2017 21.0E-3 32.0E-3 1.5E+0 6.00 GO:0000302 Purine metabolism KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004721 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 5.88 GO								3.00
GO:0000970 AminoacyI-RNA biosynthesis KEGG_01.03.2017 19.0E-3 32.0E-3 1.5E+0 6.06 GO:0000200 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 7.09 GO:0000306 DNA Damage Recognition in GG-NER REACTOME_Pathways_01.03.2017 21.0E-3 32.0E-3 1.5E+0 7.09 GO:0000306 DNA methylation GO_BiologicalProcess-GOA_23.02.2017_10h01 19.0E-3 32.0E-3 1.5E+0 4.00 GO:0000305 Purine metabolism KEGG_0.10.3.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG_0.10.3.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_0.10.3.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_0.10.3.2017 21.0E-3 33.0E-3 1.5E+0 5.88 GO:0004704 RORA activates gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.88 <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td>7.00</td>								7.00
GO:000230 Purine metabolism KEGG_010.3.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0001425 DNA Damage Recognition in GG-NER REACTOME_Pathways_01.03.2017 21.0E-3 32.0E-3 1.5E+0 7.88 GO:0000306 DNA methylation GO_BiologicalProcess-GOA_23.02.2017_10h01 19.0E-3 32.0E-3 1.5E+0 6.06 GO:0000307 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:00004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 5.86 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 5.86 GO:00040723 Retrograde endocannabinoid signaling KEGG_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.86 <td></td> <td></td> <td></td> <td></td> <td></td> <td>1 = 5 0</td> <td></td> <td>4.00</td>						1 = 5 0		4.00
GO:0006306 DNA methylation GO_BiologicalProcess-GOA_23.02.2017_10h01 19.0E-3 32.0E-3 1.5E+0 6.06 GO:0000300 Purine metabolism KEGG_01.03.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0003024 heurotransmitter receptor activity GO_MolecularFunction-GOA_23.02.2017_10h01 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004073 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004014 BMAL1:CLOCK.NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 3.40E-3 1.5E+0 5.80 GO:0000404 BMAL1:CLOCK.NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 3.40E-3 1.5E+0 5.80 GO:00000304 DNA modification GO_BiologicalProcess-GOA_2.30.2.2017_10h01	GO:0000230	Purine metabolism			32.0E-3			7.00
GO:0000230 Purine metabolism KEGG 0103.2017 20.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 32.0E-3 1.5E+0 4.00 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004073 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004073 Retrograde expression REACTOME Pathways 01.03.2017 22.0E-3 3.4.0E-3 1.5E+0 5.88 GO:0000414 BMAL1:CLOCK,NPAS2 activates circadian gene expression REACTOME Pathways 01.03.2017 22.0E-3 3.4.0E-3 1.5E+0 5.80 GO:0000414 BMAL1:CLOCK,NPAS2 activates circadian gene expression REACTOME Pathways 01.03.2017 22.0E-3 3.4.0E-3 1.5E+0 5.80 GO:0000404 ATP-dependent chromatin remodeling GO BiologicalProcess-GOA_23.02.2017								3.00
GO:0030594 neurotransmitter receptor activity GO. MolecularFunction-GOA 23.02.2017_10h01 20.0E-3 32.0E-3 1.5E+0 5.00 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:000410 RORA activates gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.88 GO:0000141 BMAL1:2LDCK, NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000404 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000404 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000405 GO:0004060 QLBandogicalProcess-GOA_23.02.2017_10h01								4.00
GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:00086003 cardiac muscle cell contraction GO BiologicalProcess-GOA_23.02.2017_10h01 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0004723 Retrograde endocannabinoid signaling KEGG_01.03.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:000411 BMAL1:CLOCK, NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000979 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000397 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000397 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000490 GO:0004904 ATP-dependent chromatin remodeling GO_BiologicalProcess-GOA_23.02								7.00
GO:0086003 gardiac muscle cell contraction GO_BiologicalProcess-GOA_23.02.2017_10h01 21.0E-3 33.0E-3 1.5E+0 5.88 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 5.88 GO:0004723 Retrograde endocannabinoid signaling KEGG 01.03.2017 21.0E-3 33.0E-3 1.5E+0 5.88 GO:000140 RORA activates gene expression REACTOME Pathways_0.10.3.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000979 Circadian Clock REACTOME Pathways_0.10.3.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000979 Circadian Clock REACTOME Pathways_0.10.3.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0003040 DNA modification GO_BiologicalProcess-GOA_23.02.2017.10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0004304 APT-dependent chromatin remodeling GO_BiologicalProcess-GOA_23.02.2017.10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0004306 Giycosyl compound cataboblic process GO_BiologicalProcess-GO								5.00
GO:0004723 Retrograde endocannabinoid signaling KEGG .010.3.2017 21.0E-3 33.0E-3 1.5E+0 4.95 GO:0008003 cardiac muscle cell contraction GO BiologicalProcess-GOA_23.02.2017 10B-3 33.0E-3 1.5E+0 5.88 GO:000140 RORA activates gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000141 BMAL1:CLOCK.NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000404 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0004304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0004304 ATP-dependent chromatin remodeling GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0004305 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0004656 glycosyl compound catabolic			GO BiologicalProcess-GOA 23.02.2017 10601					5.00 4.00
GO:0086003 cardiac muscle cell contraction GO_BiologicalProcess-GOA_23.02.2017_10h01 21.0E-3 33.0E-3 1.5E+0 5.88 GO:0000140 RORA activates gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000140 BMAL1:CLOCK,NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000379 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000397 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0004056 Glycine, serine and threonine metabolism KEGG_01.03.2017 24.0E-3 3.0E-3 1.5E+0 7.50 GO:0004056 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.5E+0 7.50 GO:0004056 Scienteroptory GO_BiologicalProces-GOA_23.02.2017_10h01								5.00
GC:0000140 RORA activates gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000141 BMAL1:CLOCK,NPAS2 activates circadian gene expression REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000301 Dixadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0003040 ATP-dependent chromatin remodeling GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000260 GO,BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000460 Glycine, series and throonine metabolism KEGG_01.03.2017 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0004605 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0004605 B cell receptor signaling pathway KEGG_01.03.2017 24.0E-3								4.00
GO:0000141 BMAL1:SLDCK.NPAS2 activates circadian gene expression REACTOME Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000379 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0006304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0006304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0006304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0006305 Glycine, series and threonine metabolism KEGG_0.10.32017 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0004658 glocosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0004662 Scill receptor signaling pathway KEGG_0.10.32017 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0001465 Interactions of neurotisin and neuroligins at synapses GO_BiologicalProce								4.00
GC:0000979 Circadian Clock REACTOME_Pathways_01.03.2017 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0006304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0006304 DNA modification GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 4.85 GO:0002600 Glycine, serine and threonine metabolism KEGG_01.03.2017 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0003260 Glycine, serine and threonine metabolism KEGG_01.03.2017 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0003040 CologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.4E+0 5.65 GO:0004662 B cell receptor signaling pathway KEGG_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001464 Interactions of neurexins and neuroligins at synapses GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-	GO:0000141	BMAL1:CLOCK,NPAS2 activates circadian gene expression	REACTOME_Pathways_01.03.2017	22.0E-3	34.0E-3		5.80	4.00
GO:0043044 ATP-dependent chromatin remodeling GO_BiologicalProcess-GOA_23.02.2017_10h01 22.0E-3 34.0E-3 1.5E+0 5.80 GO:0000260 Glycine, serine and threonine metabolism KEGG_01.03.2017 24.0E-3 35.0E-3 1.5E+0 7.50 GO:1901658 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0003000 cardiac muscle hypertrophy GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 35.0E-3 1.5E+0 7.50 GO:0004625 Dell receptor signaling pathway KEGG_0.10.3.2017 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0004666 nucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0004666 nucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0001464 Interactions of neuroligins at synapses REACTOME_Pathways 01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions at synaps	GO:000979	Circadian Clock	REACTOME_Pathways_01.03.2017	22.0E-3		1.5E+0	5.80	4.00
GO:0002200 Givene, serine and threonine metabolism KEGG_01.03.2017 24.0E-3 35.0E-3 1.5E+0 7.5G GO:101658 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 35.0E-3 1.5E+0 7.5G GO:0001300 cardiac muscle hypertrophy GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0001300 cardiac muscle hypertrophy GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001466 bucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63								5.00
GO:1901658 glycosyl compound catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 24.0E-3 35.0E-3 1.5E+0 7.5G GO:000300 cardiac muscle hypertrophy GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0004665 B cell receptors signaling pathway KEGG_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0004666 hucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0004666 hucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0004664 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 3.0E-3 1.4E+0 5.63								4.00
GO:0003300 cardiac muscle hypertrophy GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0004662 B cell receptor signaling pathway KEGG_10.03.2017 24.0E-3 36.0E-3 1.4E+0 5.56 GO:0004662 B cell receptor signaling pathway KEGG_10.03.2017 24.0E-3 36.0E-3 1.4E+0 5.53 GO:000166 nucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 3.6.0E-3 1.4E+0 5.56 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions of as synapses REACTOME_Pathways_01.03.2017 24.0E-3 3.6.0E-3 1.4E+0 5.63								3.00
GO:0004662 B cell receptor signaling pathway KEGG_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0009166 hucleotide catabolic process GO_BiologicalProcess-GOA_23.02.2017 1.0E-3 36.0E-3 1.4E+0 5.65 GO:0001646 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001645 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63								3.00
GO:0009166 nucleotide catabolic process GO.BiologicalProcess-GOA_23.02.2017_10h01 26.0E-3 36.0E-3 1.4E+0 5.56 GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63								4.00
GO:0001464 Interactions of neurexins and neuroligins at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63 GO:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63								4.00
G0:0001465 Protein-protein interactions at synapses REACTOME_Pathways_01.03.2017 24.0E-3 36.0E-3 1.4E+0 5.63								4.00
								4.00
	GO:0004662	B cell receptor signaling pathway	KEGG_01.03.2017	24.0E-3	36.0E-3	1.4E+0	5.63	4.00
GO:0005161 Hepatitis B KEGG_01.03.2017 25.0E-3 37.0E-3 1.4E+0 4.17	GO:0005161	Hepatitis B						6.00
G0:0001072 Post NMDA receptor activation events REACTOME_Pathways_01.03.2017 25.0E-3 37.0E-3 1.4E+0 7.32								3.00
GO:0001083 CREB phosphorylation through the activation of Ras REACTOME_Pathways_01.03.2017 25.0E-3 37.0E-3 1.4E+0 7.32	GO:0001083	CREB phosphorylation through the activation of Ras	REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3.00

Activation o	NMDA receptor upon glutamate binding and postsynaptic						
GO:0001085 events		REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
	on uopn Ca2+ infux through NMDA receptor	REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
O:0005161 Hepatitis B		KEGG_01.03.2017	25.0E-3	37.0E-3	1.4E+0	4.17	6
O:0001072 Post NMDA	receptor activation events	REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
	phorylation through the activation of Ras	REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
Activation of	NMDA receptor upon glutamate binding and postsynaptic						
GO:0001085 events		REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
	on uopn Ca2+ infux through NMDA receptor	REACTOME_Pathways_01.03.2017	25.0E-3	37.0E-3	1.4E+0	7.32	3
	cetylase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	27.0E-3	38.0E-3	1.4E+0	7.14	3
	gulation of neuron apoptotic process	GO_BiologicalProcess-GOA_23.02.2017_10h01	28.0E-3	39.0E-3	1.4E+0	4.05	6
GO:0004918 Thyroid hor		KEGG_01.03.2017	28.0E-3	39.0E-3	1.4E+0	5.41	4
GO:0004918 Thyroid hor	none synthesis	KEGG_01.03.2017	28.0E-3	39.0E-3	1.4E+0	5.41	4
GO:0006497 protein lipid		GO_BiologicalProcess-GOA_23.02.2017_10h01	30.0E-3	41.0E-3	1.4E+0	4.00	6
O:0055117 regulation o	f cardiac muscle contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01	33.0E-3	45.0E-3	1.3E+0	5.13	4
O:0055117 regulation o	f cardiac muscle contraction	GO_BiologicalProcess-GOA_23.02.2017_10h01	33.0E-3	45.0E-3	1.3E+0	5.13	4
GO:0032204 regulation o	f telomere maintenance	GO_BiologicalProcess-GOA_23.02.2017_10h01	35.0E-3	46.0E-3	1.3E+0	5.06	4
O:0004919 Thyroid hor		KEGG_01.03.2017	35.0E-3	46.0E-3	1.3E+0	4.31	5
O:0004919 Thyroid hor	mone signaling pathway	KEGG_01.03.2017	35.0E-3	46.0E-3	1.3E+0	4.31	5
O:0004919 Thyroid hor	none signaling pathway	KEGG_01.03.2017	35.0E-3	46.0E-3	1.3E+0	4.31	5
O:0000197 Inositol pho	sphate metabolism	REACTOME_Pathways_01.03.2017	36.0E-3	47.0E-3	1.3E+0	6.38	3
	IP2, IP, and Ins in the cytosol	REACTOME_Pathways_01.03.2017	36.0E-3	47.0E-3	1.3E+0	6.38	3
	signaling pathway	KEGG_01.03.2017	37.0E-3	47.0E-3	1.3E+0	4.24	5
O:1901019 regulation o	f calcium ion transmembrane transporter activity	GO_BiologicalProcess-GOA_23.02.2017_10h01	36.0E-3	47.0E-3	1.3E+0	5.00	4
O:0004071 Sphingolipic		KEGG_01.03.2017	37.0E-3	47.0E-3	1.3E+0	4.24	5
	ceptor cell differentiation	GO_BiologicalProcess-GOA_23.02.2017_10h01	38.0E-3	48.0E-3	1.3E+0	6.25	3
O:0043204 perikaryon		GO_CellularComponent-GOA_23.02.2017_10h01	38.0E-3	48.0E-3	1.3E+0	4.20	5
LIA (PGC2 + CLOZUK)			1				
,			_	Term			
				PValue			
				Corrected			
				with		%	
			Term	Benjamini-		Associated	
OID GOTerm		Ontology Source	PValue				Gene
O:0052697 xenobiotic g	lucuronidation	GO_BiologicalProcess-GOA_23.02.2017_10h01	48.0E-6		2.9E+0	33.33	
	diester hydrolase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	180.0E-6		2.7E+0	6.12	6
O:0043197 dendritic spi		GO_CellularComponent-GOA_23.02.2017_10h01	160.0E-6		2.6E+0	5.15	7
	activation involved in immune response	GO_BiologicalProcess-GOA_23.02.2017_10h01	400.0E-6	3.0E-3	2.5E+0	4.43	7
O:0000983 Drug metab		KEGG_01.03.2017	610.0E-6	3.7E-3	2.4E+0	8.70	4
	ucleotide phosphodiesterase activity	GO_MolecularFunction-GOA_23.02.2017_10h01	1.5E-3	5.6E-3	2.3E+0	11.11	3
	linositol phospholipase C activity	GO_MolecularFunction-GOA_23.02.2017_10h01	1.6E-3	5.6E-3	2.3E+0	10.71	3
O:0000053 Ascorbate a		KEGG_01.03.2017	1.5E-3	5.6E-3	2.3E+0	11.11	3
O:0004520 Adherens ju		KEGG_01.03.2017	3.2E-3	5.8E-3	2.2E+0	5.56	4
	of neurexins and neuroligins at synapses	REACTOME_Pathways_01.03.2017	3.1E-3	5.8E-3	2.2E+0	5.63	4
	ein interactions at synapses	REACTOME_Pathways_01.03.2017	3.1E-3	5.8E-3	2.2E+0	5.63	4
O:0000982 Drug metab		KEGG_01.03.2017	2.9E-3	5.9E-3	2.2E+0	5.71	4
O:0001073 Unblocking	of NMDA receptor, glutamate binding and activation	REACTOME_Pathways_01.03.2017	2.7E-3	6.2E-3	2.2E+0	9.09	3
	phorylation through the activation of CaMKII	REACTOME_Pathways_01.03.2017	2.7E-3	6.2E-3	2.2E+0	9.09	3
O:0001733 SALM prote	in interactions at the synapses	REACTOME_Pathways_01.03.2017	2.7E-3	6.2E-3	2.2E+0	9.09	3
O:0002292 T cell differe	entiation involved in immune response	GO_BiologicalProcess-GOA_23.02.2017_10h01	1.4E-3	6.3E-3	2.2E+0	6.90	4
O:0000040 Pentose and	d glucuronate interconversions	KEGG_01.03.2017	2.9E-3	6.3E-3	2.2E+0	8.82	3
O:0004720 Long-term p	otentiation	KEGG_01.03.2017	2.5E-3	6.3E-3	2.2E+0	5.07	4
	ulation of telomere maintenance	GO_BiologicalProcess-GOA_23.02.2017_10h01	1.3E-3		2.2010	5.97	
				6.5E-3	2.2E+0	5.97	4
O:0001782 B cell home	ostasis	GO_BiologicalProcess-GOA_23.02.2017_10h01	2.4E-3	6.5E-3 6.7E-3			4
		GO_BiologicalProcess-GOA_23.02.2017_10h01 KEGG_01.03.2017			2.2E+0	7.14	4 (3
O:0000830 Retinol met			2.4E-3	6.7E-3	2.2E+0 2.2E+0	7.14 9.38	4 3 4
O:0000830 Retinol meta O:0000521 Neurophilin	abolism interactions with VEGF and VEGFR	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3	6.7E-3 6.8E-3	2.2E+0 2.2E+0 2.2E+0	7.14 9.38 6.15	4 3 4 3
D:0000830 Retinol meta D:0000521 Neurophilin D:0000973 Sema3A PA	abolism	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3	6.7E-3 6.8E-3 8.4E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0	7.14 9.38 6.15 7.32	4 00 4 00 00
O:0000830 Retinol meta O:0000521 Neurophilin O:0000973 Sema3A PA O:0000974 SEMA3A-P	abolism interactions with VEGF and VEGFR K.K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0	7.14 9.38 6.15 7.32 7.32	
0:0000830 Retinol met: 0:0000521 Neurophilin 0:0000973 Sema3A PA 0:0000974 SEMA3A-PI 0:0001093 Signal trans 0:0000860 Porphyrin a	abolism Interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorobhvll metabolism	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32	
O:0000830 Retinol met: O:0000521 Neurophilin O:0000973 Sema3A PA O:0000974 SEMA3A-PI O:0001093 Signal trans O:0000860 Porphyrin a	abolism Interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorobhvll metabolism	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 KEGG_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0	7.14 9.38 6.15 7.32 7.32 7.32	
O:0000830 Retinol met. O:0000521 Neurophilin O:0000973 Sema3A PA O:0000974 SEMA3A-PI O:0000973 Signal trans O:0000974 Signal trans O:0000800 Porphyrin a O:0000493 Pre-NOTCH	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 nd chlorophyll metabolism I Processing in Golgi	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.4E-3 6.5E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.5E-3 9.8E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.14 6.67	
O:0000830 Retinol met. O:0000521 Neurophilin O:0000973 Sema3A PA O:0000974 SEMA3A-PI O:0001093 Signal trans O:0000860 Porphyrin a O:0000493 Pre-NOTCH O:0000493 Pre-NOTCH	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 ad chlorophyll metabolism Processing in Golgi Expression and Processing	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 6.5E-3 6.5E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.5E-3 9.8E-3 9.8E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.14 6.67 6.67	
C:0000830 Retinol met C:0000521 Neurophilin C:0000973 Sema3A P.A C:000973 SEM33A P.A C:00001093 Signal trans C:0000409 Porphyrin a C:0000494 Pre-NOTCH C:0000494 Pre-NOTCH C:00030071 regulation 0	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism I Processing in Golgi I Expression and Processing mitotic metaphase/anaphase transition	KEGG_01(03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 G. BiologicalProcess-GOA_23.02.2017_10h01	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 6.5E-3 6.5E-3 8.3E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.14 6.67 6.67 6.12	
D:0000830 Retinol meti D:0000521 Neurophilin D:0000973 Sema3A PP D:0001093 Signal trans D:0000904 SemA3A PP D:0001093 Signal trans D:0000403 Pre-NOTCH D:0000494 Pre-NOTCH D:00030971 regulation o D:00030071 regulation o	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 a chlorophyll metabolism I Processing in Golgi Expression and Processing finitotic metaphase/anaphase transition differentiation	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.4E-3 6.5E-3 6.5E-3 8.3E-3 8.3E-3	6.7E-3 6.8E-3 8.4E-3 8.5E-3 8.3 8.10E-3 8.35 8.31100000000000000000000000000000000000	2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.34 6.67 6.67 6.12 6.12	
D:0000830 Retinol meter D:0000521 Neurophilin D:0000973 Sem3A P P D:0000974 SEMA3A-Pi D:0000974 Signal trans D:0000974 Signal trans D:0000974 Signal trans D:0000493 Pre-NOTCH D:0000493 Pre-NOTCH D:0000494 Pre-NOTCH D:0000497 Pre-NOTCH D:0000497 Pre-NOTCH D:0004078 Pre-NOTCH D:0004079 Pre-NOTCH D:0004070 Feelyter cel D:0004208 Pre-NOTCH	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 nd chlorophyll metabolism Processing in Golgi Expression and Processing f mitotic metaphase/anaphase transition I differentiation meostasis	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 6.5E-3 6.5E-3 8.3E-3 8.3E-3 9.0E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.5E-3 9.8E-3 9.8E-3 9.11.0E-3 11.0E-3	2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinal meti C:0000830 Retinal meti D:0000621 Neurophilin D:0000731 SEma3A PP D:0000741 SEMA3A.PP D:0000741 SEMA3A.PP D:0000743 SEMA3A.PP D:0000749 SemaA PP D:0000749 Pre-NOTCH- D:0000493 Pre-NOTCH- D:0000494 Pre-NOTCH- D:0000493 Pre-NOTCH- D:0000494 Pre-NOTCH- D:0000493 Pre-NOTCH- D:0000494 Pre-NOTCH- D:0000493 Pre-NOTCH- D:0000493 Pre-NOTCH- D:0000494 Pre-NOTCH- D:0000495 Pre-NOTCH- D:0000497 Image: Pre-NOTCH- D:0000497 Image: Pre-NOTCH- D:0000497 Image: Pre-NOTCH- D:0000497 Image: Pre-NOTCH- D:0001776 Image: Pre-NOTCH- D:0001776 Image: Pre-NOTCH-	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism I Processing in Golgi I Expression and Processing mitotic metaphase/anaphase transition I differentiation pomeostasis plate congression	KEGG_01(03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 5.4E-3 6.5E-3 6.5E-3 8.3E-3 8.3E-3 9.0E-3 9.7E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 1.0E-3 8.11.0E-3 8.12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol meter C:0000830 Retinol meter C:0000973 Sema3A PF C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000494 Pre-NOTCH C:00004097 T-helper ce C:0001776 leukocyte hi C:00051310 metaphase C:0000556 Nuclear Eve	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 ad chlorophyll metabolism Processing in Golgi Expression and Processing finitotic metaphase/anaphase transition differentiation omeostasis plate congression ints (kinase and transcription factor activation)	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 6.5E-3 6.5E-3 8.3E-3 9.0E-3 9.0E-3 9.0E-3 9.0E-3 9.0E-3 9.0E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 11.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 6.67 6.67 6.67 6.12 6.12 6.12 6.12 6.12 6.12 5.77 5.88	
C:0000830 Retinol met C:0000931 Neurophilin C:0000973 Sema3A PF C:0000974 SEMA3A-Pi C:0000806 Porphyrin a C:0000409 Pre-NOTCH C:0004093 Pre-NOTCH C:0030071 regulation o C:0042093 T-helper cel C:000176 leukocyte h C:00015110 metaphase C:0000556 Nuclear Eva C:0000556 ERK/MAPK	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism Processing in Golg Expression and Processing f mitotic metaphase/anaphase transition d differentiation meostasis plate congression ints (kinase and transcription factor activation) targets	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 2.2E-3 5.0E-3 5.0E-3 5.0E-3 6.5E-3 6.5E-3 8.3E-3 9.0E-3 9.0E-3 9.2E-3 9.2E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol meti C:0000521 Neurophilin C:0000973 Sem3A P P C:0000974 SEMA3A-PI C:0000974 SEMA3A-PI C:0000974 SEMA3A-PI C:0000974 SEMA3A-PI C:0000806 Porphyrin a C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000493 T-helper cel C:0000776 eukocyte h C:00051310 metaphase C:0000556 Nuclear Eve C:0000556 CREMAPK C:0000564 CREB phos	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism I Processing in Golg I Expression and Processing mitotic metaphase/anaphase transition I differentiation omeostasis plate congression ents (kinase and transcription factor activation) targets phorylation	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.0E-3 5.4E-3 6.5E-3 8.3E-3 8.3E-3 9.0E-3 9.7E-3 9.7E-3 9.2E-3 9.2E-3	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.10E-3 12.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol met C:0000831 Neurophilin C:0000973 Sema3A PF C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000497 Pre-NOTCH C:0000497 Pre-NOTCH C:0000470 Pre-NOTCH C:00001776 eukocyte h C:0001776 eukocyte h C:00001776 eukocyte h C:0000156 Nuclear Eve C:0000558 ERK/MAPK C:0000564 CREB phos C:0001116 MAPK targe	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 nd chlorophyll metabolism Processing in Golgi Expression and Processing f mitotic metaphase/anaphase transition I differentiation meostals plate congression nets (kinase and transcription factor activation) targets phorylation ts /Nuclear events mediated by MAP kinases	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO BiologicalProcess-GOA_23.02.2017_10h01 GO BiologicalProcess-GOA_23.02.2017_10h01 GO BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-2 2.2E-2 5.0E-2 5.0E-2 5.0E-2 6.5E-2 6.5E-2 8.3E-2 9.7E-2 9.2E-2 9.2E-2 9.2E-2 9.2E-2 9.2E-2	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.10E-3 12.0E-3 12.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol met C:0000831 Neurophilin C:0000973 Sema3A PF C:0000974 SEMA3A-P C:0000974 SEMA3A-P C:0000984 Signal trans C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000497 Pre-NOTCH C:0000497 Pre-NOTCH C:0000497 Pre-NOTCH C:000176 leukocyte h C:000176 leukocyte h C:00051310 metaphase C:0000556 Nuclear Eve C:0000556 Nuclear Eve C:0000564 CREB phos C:0001116 MAPK targe C:000129 CD209 (DC2)	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism I Processing in Golgi I Expression and Processing f mitotic metaphase/anaphase transition d differentiation meostasis plate congression Ints (kinase and transcription factor activation) targets phorylation ts/ Nuclear events mediated by MAP kinases SIGN) signaling	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017	2.4E-2 2.2E-3 5.0E-2 5.0E-2 5.0E-2 5.0E-2 6.5E-2 6.5E-2 6.5E-2 9.0E-2 9.0E-2 9.2E-2 9.2E-2 9.2E-2 9.2E-2 9.2E-2	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 11.0E-3 12.0E-3 12.0E-3 12.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol mete C:0000831 Neurophilin C:0000973 Sema3A PF C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000494 Pre-NOTCH C:0000409 T-helper ce C:0001776 leukocyte hi C:00051310 metaphase C:0000558 ERK/MAPK C:0000558 ERK/MAPK C:0000558 CREB phos C:0000514 CREB phos C:0001116 MAPK targe C:0001291 CD290 [DC C:0000640 Glutathione	abolism interactions with VEGF and VEGFR KK dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 ad chlorophyll metabolism Processing in Golgi Expression and Processing fmitotic metaphase/anaphase transition differentiation omeostasis plate congression ints (kinase and transcription factor activation) targets phorylation ts/ Nuclear events mediated by MAP kinases SIGN) signaling metabolism	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017 REACTOME_Pathways_01.03.2017	2.4E-5 2.2E-5 5.0E-5 5.0E-5 5.4E-5 6.5E-5 8.3E-5 9.0E-5 9.0E-5 9.2E-5 9.	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.5E-3 9.9E-3 9.9E-3 9.9E-3 11.0E-3 11.0E-3 12.0E-3 12.0E-3 12.0E-3 12.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	$\begin{array}{c} 7.14\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.44\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 6.588\\ 5.88$	
0:0000830 Retinol met 0:0000831 Reurophilin 0:0000973 Sema3A PF 0:0000974 SEMA3A-Pi 0:0000974 SEMA3A-Pi 0:0000974 SEMA3A-Pi 0:0000974 SEMA3A-Pi 0:0000493 Signal trans 0:0000493 Pre-NOTCH 0:0000494 Pre-NOTCH 0:0000497 Pre-NOTCH 0:000176 leukocyte h 0:000176 leukocyte h 0:00005056 RK/MAPK 0:0000516 Nuclear Eve 0:0000176 MAPK targe 0:0001121 CD209 (DC 0:0001210 CD209 (DC 0:0000400 Steroid horin	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 nd chlorophyll metabolism Processing in Golgi Expression and Processing f mitotic metaphase/anaphase transition I differentiation meostasis plate congression ints (kinase and transcription factor activation) targets bhorylation ts/ Nuclear events mediated by MAP kinases SIGN) signaling metabolism none biosynthesis	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017	2.4E-5 2.2E-5 5.0E-5 5.0E-5 5.0E-5 5.0E-5 5.0E-5 6.6E-5 8.3E-5 9.0E-5 9.0E-5 9.2E-5 9.2E-5 9.2E-5 9.2E-5 9.2E-5 9.2E-5 10.0E-5	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0 1.9E+0	7.14 9.38 6.15 7.32 7.32 7.32 7.32 7.32 7.32 7.32 7.32	
C:0000830 Retinol met 0:0000521 Neurophilin 0:0000973 Sema3A P4 0:0000974 SEMA3A-P1 0:0000973 Signal trans 0:0000974 SEMA3A-P1 0:0000974 SEMA3A-P1 0:0000403 Signal trans 0:0000403 Pre-NOTCH 0:0000404 Pre-NOTCH 0:0000407 regulation o 0:000176 leukozyte h 0:0000556 Nuclear Eve 0:0000564 CREB phos 0:0001116 MAPK targe 0:000120 CD209 (DC 20) 0:0001216 Steroid horn 0:000140 Steroid horn	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism I Processing in Golgi I Expression and Processing mitotic metaphase/anaphase transition d differentiation meostasis plate congression Ints (kinase and transcription factor activation) targets phorylation ts/ Nuclear events mediated by MAP kinases SIGN) signaling metabolism none biosynthesis uron differentiation	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017 REACTOME_Pathways_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 KEGG_01.03.2017	2.4E-5 2.2E-5 5.0E-5 5.0E-5 5.0E-5 5.0E-5 5.4E-5 8.3E-5 9.2E-5 9.	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.5E-3 9.8E-3 9.8E-3 11.0E-3 11.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.	$\begin{array}{c} 7.14\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.34\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.88\\$	
C:000830 Retinol met C:000831 Neurophilin C:0000973 Sem3A P/ C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0004093 T-helper ce C:00001776 eukocyte h C:0042093 T-helper ce C:0000576 Nuclear Eve C:0000558 ERK/MAPK C:0000516 CREB phos C:0000516 CREB phos C:0000140 Steroid hort C:0002479 Grebrain ne C:0002479 Grebrain ne	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 and chlorophyll metabolism Processing in Golgi Expression and Processing finitotic metaphase/anaphase transition I differentiation omeostasis plate congression ints (kinase and transcription factor activation) targets phorylation str Nuclear events mediated by MAP kinases SIGN) signaling metabolism none biosynthesis uron differentiation tion involved in immune response	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 <td>2.4E- 2.2E- 5.0E-5</td> <td>6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3</td> <td>2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.</td> <td>$\begin{array}{r} 7.14\\ 9.38\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.14\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\$</td> <td></td>	2.4E- 2.2E- 5.0E-5	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.	$\begin{array}{r} 7.14\\ 9.38\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.14\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\$	
C:0000830 Retinol met C:0000831 Neurophilin C:0000973 Sema3A P4 C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000498 Pre-NOTCH C:0000499 Pre-NOTCH C:0000499 Pre-NOTCH C:00042093 T-helper cel C:00042093 T-helper cel C:0001776 eukocyte h C:0001761 eukocyte h C:000556 Nuclear Eva C:0000564 CREB phos C:0001716 MAPK targe C:0001291 CD209 (DC C:0000480 Glutathione C:00012179 forebrain ne C:0002121 Renal cell cetva	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism Processing in Golgi Expression and Processing f mitotic metaphase/anaphase transition d differentiation meostasis plate congression ints (kinase and transcription factor activation) targets phorylation ts' Nuclear events mediated by MAP kinases SIGN) signaling metabolism none biosynthesis uron differentiation tion involved in immune response arcinoma	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REG_01.03.2017 KEGG_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01	2.4E- 2.2E- 5.0E- 5.0E- 5.0E- 5.0E- 6.5E- 6.5E- 9.0E- 9.0E- 9.2E- 9.2E- 9.2E- 9.2E- 9.2E- 10.0E- 13.0E- 14.	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 11.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.7E+0 1.	$\begin{array}{c} 7.14\\ 9.38\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.34\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.77\\ 5.77\\ 5.78\\ 5.88\\$	
C:0000830 Retinol mete C:0000830 Retinol mete C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000974 SEMA3A-Pi C:0000493 Pre-NOTCH C:0000493 Pre-NOTCH C:0000494 Pre-NOTCH C:0000494 Pre-NOTCH C:0000491 T-helper ce C:0001776 leukocyte hi C:00051310 metaphase C:0000558 ERK/MAPK C:0000558 ERK/MAPK C:0000558 CREB phos C:00001116 MAPK targe C:0000140 Steroid hort C:000149 Grebrain ne C:0001421 Steroid hort C:0001212 S cell activa C:0002312 8 cell activa C:0006211 Renal cell C	abolism interactions with VEGF and VEGFR K dependent Axon repulsion exin repulsion signaling by inhibiting Integrin adhesion duction by L1 d chlorophyll metabolism Processing in Golgi Expression and Processing f mitotic metaphase/anaphase transition d differentiation meostasis plate congression ints (kinase and transcription factor activation) targets phorylation ts' Nuclear events mediated by MAP kinases SIGN) signaling metabolism none biosynthesis uron differentiation tion involved in immune response arcinoma	KEGG_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 GO_BiologicalProcess-GOA_23.02.2017_10h01 REACTOME_Pathways_01.03.2017 REACTOME_Pathways_01.03.2017 <td>2.4E- 2.2E- 5.0E-5</td> <td>6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3</td> <td>2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.</td> <td>$\begin{array}{r} 7.14\\ 9.38\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.14\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\$</td> <td></td>	2.4E- 2.2E- 5.0E-5	6.7E-3 6.8E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 8.4E-3 9.8E-3 9.8E-3 9.8E-3 11.0E-3 12.0E-3	2.2E+0 2.2E+0 2.2E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.1E+0 2.0E+0 2.0E+0 2.0E+0 2.0E+0 1.9E+0 1.	$\begin{array}{r} 7.14\\ 9.38\\ 9.38\\ 6.15\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.32\\ 7.14\\ 6.67\\ 6.12\\ 6.12\\ 6.12\\ 6.12\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\ 5.88\\ 5.88\\ 5.48\\ 5.88\\$	

Table 3.16 | GO for genes in SZ risk-associated chromosomal contacts.GO enrichment of genes in Table 3.15 See description of Table 3.2-3.6 forlegend; Benjamini Hochberg was used as correction for multiple comparisons.

(P	NPC GC+CLOZUK)	(P	NEURON GC+CLOZUK)	(PG	GLIA iC+CLOZUK)		NPC (PGC)	NE	URON (PGC)	G	GLIA (PGC)	NPC	-String (PGC)	N	EURON-String (PGC)	Gl	IA-String (PGC)
1	EIF2B1	1	PCDHA1	1	ANKRD44	1	PHF21A	1	EPC2	1	SNAP91	1	ITIH4	1	BAHD1	1	ADSS
2	CUL3	2	FSIP2	2	SNAP91	2	ZBTB18	2	DIS3L2	2	ANKRD44	2	SLC2A5	2	CUL3	2	MOB4
3	MARS2	3	PCDHA2	3	BRINP3	3	FANCL	3	SFMBT1	3	GRAMD1B	3	ITIH3	3	BAZ2A	3	CUL3
4	DNAJC19	4	CSMD1	4	ZNF804A	4	MAD1L1	4	NLGN4X	4	ZNF804A	4	SEC16B	4	TAF5	4	NFATC3
5	ORC5	5	PCDHA11	5	RABGAP1L	5	KDM4A	5	KMT2E	5	SLC4A10	5	CACNA1D	5	SF3B1	5	SF3B1
6	ZEB2	6	CHRNA3	6	LINC01122	6	KDM3B	6	SEMA6C	6	LINC01122	6	HECW2	6	ACTR5	6	ADSL
7	PHF21A	7	ATPAF2	7	SLC4A10	7	SOX2-OT	7	NT5M	7	TCF4	7	PPP2R3A	7	POLDIP3	7	EP300
	707040		DODUOAAA								ANKHD1-				ANIDOOF		DNEGOO
8	ZBTB18	8	PCDHGA10	8	MIR137HG	8	SAP30L-AS1	8	PLCL2	8	EIF4EBP3	8	SH3RF1	8	ANP32E	8	RNF220
9	ITGA9	g	IL17RB	9	GRAMD1B	9	ARNT	9	PLPPR5	g	C1orf100	9	TCTN2	g	CDC20	9	ANKHD1
10	ALMS1	10	PSMG3-AS1	10	OPCML	10	FXR1	10	PBRM1	10	CSMD1	10	CLCN3	10	CDC25C	10	UTS2
11	TET3	11	C3orf49	11	CSMD1	11	CD46	11	RAI1	11		11	CLU	11	DNAJC19	11	HECW2
12	CCDC150	12	PPP2R3A	12	HECW2	12	EPB41	12	RNF220	12		12	CACNA1C	12	THOC7	12	MEF2C
13	MPHOSPH9	13	BRINP3	13	PLD5	13	RAD54L2	13	PLEKHO1	13	UTS2	13	PDE4D	13	SUFU	13	TCF4
											PTCHD1-						
14	KNTC1	14	FBXO41	14	DOCK10	14	RPS6KA3	14	RERE	14	AS	14	GPM6A	14	MAD1L1	14	GRIN2A
15	POC1A	15		15	CHRM3	15	RSRC2	15	INPP5D	15	CCDC39	15	DNAJC18	15	RNF220	15	PLXNA4
16	ANP32E	16		16	GPM6A	16		16		16	ZNF165	16	NT5C2	16	PHF21A	16	MAPK3
17		17		17	TRANK1	17		17			PITPNM2	17	GRIN2A	17		17	
18	SGO2	18	PCDHA6	18	GALNT15	18	TAF5	18	FAM53C	18	RILPL1	18	OXNAD1	18	SEMA6C	18	JUND
19	CENPL	19	PCDHA8	19	PDE4B	19	PPP1CC	19	PTPRF	19	COQ10B	19	HSPA9	19	NT5M	19	SH3RF1
									FPGT-								
20	PPIH	20	PCDHA7	20	PTCHD1-AS	20	SF3B1	20	TNNI3K	20	JUND	20	CYP26B1	20	STAG1	20	AOX1
21	BRD8	21	PCDHA9	21	UTS2	21	DESI2	21	AKT3	21	ARTN	21	UGT1A7	21	CEP162	21	GPER1
22	SUGP1	22	PCDHAC1	22	C1orf100	22	NFATC3	22	RRP7BP	22	SH3RF1	22	UGT1A10	22	H6PD	22	NMUR2
23	CD46	23	PLD5	23	EMX1	23	PCGF6	23	SERHL2	23	NEK1	23	UGT1A9	23	PSKH1	23	DPP4
									SAP30L-								
24	EPB41	24	KCNH7	24	ALMS1P1	24	NOSIP	24	AS1	24	GRIN2A	24	UGT1A8	24	CTNND1	24	CNKSR2
25	RSRC2	25		25	NAT8	25	PPIH	25	SATB2	25		25	UGT1A6	25	SREBF1	25	UGT1A9
26	GTF3C3	26	CA8	26	HYDIN	26	CETN3	26	ZBTB18	26		26	SDCCAG8	26		26	
27	SBNO1	27	FTCDNL1	27	SLC9C2	27	SMS	27	PHF21A	27		27	PPARGC1A	27	GATAD2A	27	UGT1A8
28	PPP1CC	28	NTM	28	NEK1	28	KNTC1	28	CBR4	28	DOCK10	28	CA6	28	HARS	28	UGT1A4
29	SF3B1	29	OPCML	29	GRIN2A	29	ANP32E	29	COX20	29		29	AOX1	29	ZMAT2	29	UGT1A6
30	TAF5	30	ARHGAP40	30	PLXNA4	30	CDC25C	30	ZSCAN23	30		30	CLIP1	30	AMBRA1	30	UGT1A10
31	ZSWIM6	31		31	MEF2C	31		31	SMG1P2	31		31	ASB5	31	PLCB2	31	
32	RAD54L2	32		32	SDCCAG8	32		32		32		32	ST3GAL3	32	MED8		
33	RPS6KA3	33	SERPINE2	33	ARTN	33		33	SLC35G2	33		33	CREB3L2	33	ADSS		
34	RANGAP1	34		34	SH3RF1	34		34	ARL6IP4	34		34	PCCB	34	MARS		
35	SUGP2	35	PCDHGA2	35	PITPNM2	35		35	PLXNA2	35		35	GRIA1	35	PSMG3		
36	ARNT	36		36	STAB2	36		36	TOX	36		36	HDAC3	36	SH3GL3		
	FXR1	37		37	KCNJ13	37		37				37	PPIH		HECW2		
37	FARI	31	NDUFA4L2	31	KCINJ13	31	ZSWIM6	31	RASAL2	37	TMX2-	31	FFIN	37	HEGWZ		
20	MDK	38	STAC3	38	NDUFA13	20	DANCAD1	38	PCDHGA6	20		38	SUGP1	38	CA8		
38	NCK1	30	IL19	30	SLC4A5	38	RANGAP1 SUGP2	30	PCDHGA6 PCDHGB3	38			GATAD2A				
39						39				39		39		39			
40	TKT	40	ITIH1	40	YJEFN3	40	EIF2B1	40	PCDHGA4	40		40	UTP4	40	PPP2R3A		
41	NUBP1	41	BOLL	41	JUND	41	CUL3	41	PCDHGA5	41		41	PSMD14	41	GRM3		
42	HDAC3	42	EGR4	42	COQ10B	42	MARS2	42	SERPINF1	42		42	EP300	42	WDR81		
43	PIH1D1	43	SDCCAG8	43	RILPL1	43	MOB4	43	TMEM110	43		43	MAD1L1	43	NT5C2		
44	PSMD14	44		44	PGM3	44	AMBRA1	44	DPYD	44		44	CUL3	44	GRIN2A		
45	EIF3B	45	PCDHA5	45	LSMEM1	45	ZKSCAN4	45	P2RX3	45	NUGGC	45	DNAJC19	45	ITIH4		
					TMX2-												
46	GATAD2A	46		46	CTNND1	46		46	PTBP2	46		46	ARNT	46			
47	SNX7	47		47	KLHL20	47		47		47		47	SNTB2	47			
48	CETN3	48		48	ERMAP	48	INPP5D	48	PGAP1	48		48	RANGAP1	48			
49	FGFR1	49		49	SFXN5	49	RASAL2	49	TYW5	49		49	EPB41	49	SNAP91		
50	PPP2R2A	50	MIR548AI	50	TBC1D10B	50	VSIG2	50	C2orf69	50		50	RPS6KA3	50	PFKFB2		
51	DESI2	51	NT5C2	51	USP40	51		51	ZSCAN12	51		51	TAF5	51	CACNA1C		
52	NFATC3	52	ALMS1P1	52	VRK2	52	C2orf69	52	CD46	52		52	PPP1CC	52			
53	SMS	53	NAT8	53	EXOC6B	53	PGAP1	53	MSL2	53		53	SF3B1	53	DGKD		
54	NOSIP	54	PHEX	54	MBTPS2	54	ADSS	54	SBNO1	54		54	BRD8	54	MPL		
55	PCGF6	55	TOM1L2	55	MAPK3	55	ETF1	55	BAZ2A	55		55	KNTC1	55	PPARGC1A		
56	FAM53C	56		56	GRIA1	56	HSPD1	56	ZSWIM6	56		56	ANP32E	56	HSPA9		
57	DUS2	57		57	MAN2A1	57		57	TAF5	57		57	SGO2	57	HARS2		
58	RFT1	58		58	IMMP2L	58		58	ACTR5	58		58	CDC25C	58	ATG13		
59	PRDX6	59	ANKRD45	59	ST3GAL3	59		59	RPS6KA3	59		59	AKT3	59	SLC12A4		
60	ADSS	60	DCDC1	60	AUP1	60	DUS2	60	KMT2E-AS1	60		60	PHF21A	60	CTNNA1		
61	ETF1	61	PTCHD1-AS	61	GALNT10	61	SNX7	61	GID4	61		61	KMT2E	61	PLXNA2		
62	HSPD1	62	FAM124B	62	GPER1	62	ORC5	62	FXR1	62	CNBD1	62	PLCL2	62	PDE6D		
63	FANCL	63	ITIH4	63	MKL1	63	DNAJC19	63	WDFY1	63	TNNI3K	63	NCAN	63	DGKZ		
64	MAD1L1	64	C1orf100	64	SLC6A9	64	HPF1	64	BAHD1	64	MPP6	64	EPHB1	64	GRIA1		
65	KDM4A	65	CR1L	65	TNRC6B	65	ATP6V0A2	65	CUL3	65	DNAH10	65	CHST12	65	CA6		
66		66		66	TCF4	66		66	DESI2	66	C12orf65	66	RPRD2	66			
67	SOX2-OT	67	DOCK10	67	CCDC39	67		67	POLDIP3	67		67	MAU2	67	MBTPS2		
68	STAG1	68		68	ZNF165	68		68	NFATC3	68		68	B4GALT2	68			
69		69		69			UTP4	69	SF3B1	69		69		69			
		50	.=	20	-				MPHOSPH								
70	ASH2L	70	CPEB1	70	NUGGC	70	EIF3B	70		70	PSKH1	70	DPYD	70	DYSF		
71		71		70			GATAD2A	71	CCDC150	71		71	NCK1	71			
72		72			MIR4304		NUBP1		ANP32E		TBC1D5	72		72			
73		73		73	NMUR2	73		73	NEMP1	73		73	HSPD1	73			
13		13	. 0240	13	ANKHD1-	13	. 12/100	13	A DE IVIT 1	13	. LENIOT	13	. 101 0 1	13			
	TBC1D5	74	PDE4D	74	EIF4EBP3	74	PIH1D1	74	G3BP1	74	RERE	74	EBNA1BP2				
7/				74				. 74	30011	14		74	CDIM/ODF Z				
74 75		75	HIVEP2	75	HCN1	75	MAU2	75	CDC25C	75	NLGN4X	75	GTF2H3				

76	MAU2	76	CACNA1C	76	PAPPA2	76	B4GALT2	76	CDC20	76	TAOK2	76	EPC2	1	1		
70	WIA02	70	CACNA1C-	70	TATTA2	70	D4GAL12	70	CDC20	70	TAON2	70	LFOZ				
77	ADGRG6	77	AS4	77		77		77		77	GALNT10	77	STAG1				
78	SOX2	78	CACNA1C-IT3	78	CNBD1	78		78		78	MKL1						
79 80	CMTR2 RANBP10	79 80	GALNT15 PRUNE1	79 80	STK31 FCAMR	79	AADAT EPC2	79 80	IPO11 SOX2-OT	79 80	GPER1 MAPK3						
00	KAINDE IU	00	ADAMTS9-	00	FCAINK	00	EFG2	00	3072-01	00	MAFKS						
81	ATP6V0A2	81	AS2	81	UGT1A7	81	STAG1	81	MAD1L1	81	GRIA1						
82	EP300	82	EPN2	82		82	DENR	82		82	MAN2A1						
83	FPGT-TNNI3K	83	GPM6A	83	UGT1A4	83	VPS33A	83	DNAJC19	83	IMMP2L						
84 85	PTPRF OXNAD1	84 85	TMEFF2 EMX1	84 85	OR1S2 UGT1A10	84 85	DPYD TMEM110	84 85	KDM4A SUFU	84 85	ST3GAL3 UNCX						
86	PODXL	86	PCDHGA12	86		86		86		86							
	1 OBAL	00	1 OBIIO/ILE	00	001110	00	TMEM110-	00	174102	00	111111010						
87	AADAT	87	MEF2C	87		87	MUSTN1	87		87							
88	EPC2	88		88		88		88			NRN1L						
89	SLC39A8 CHST12	89	DGKD SLC2A5	89 90		89		89		89							
90 91	GFRA3	90 91	ITIH3	90		90 91		90 91		90	VRK2 USP40						
92	LYPD6	92		92	AOX2P	92		92			LRRIQ3						
93	NLGN4X	93	CDHR3	93	MYLPF	93	PCDHGA4	93	MARS	93	BOLA2						
94	SATB1	94	LINC00634	94	UNCX	94	PCDHGA5	94		94	OGFOD2						
95	PLEKHO1	95	PCDHAC2	95	TNFRSF9	95	SLC39A8	95		95	ERMAP						
96 97	RERE RPRD2	96 97	BRINP2 CACNA1D	96 97	AOX1 NRN1L	96 97	CHST12 GFRA3	96 97	SLC25A33 DPH3	96 97	TBC1D10B SLC6A9						
98	SAP30L-AS1	98	KLHL29	98	RFTN2	98		98	ZMAT2	98	TNRC6B						
99	DPYD	99	PCDHA4	99	LINC00461	99		99	PARK7	99	NMUR2	1		1	İ		
					TMEM161B-												
100	SATB2	100	SH3PXD2A	100	AS1	100		100		100	HCN1			L			
101	M1AP	101		101		101	PLEKHO1 RERE	101	C7orf50	101	PAPPA2 C2orf69			I			
102	SLC35G2 EMB	102	MRAP2 INO80B	102 103	RERE NLGN4X	102	RERE RPRD2	102		102	C2orf69 CUL3						
103	RASAL2	103	SLC2A7	103	TAOK2	103	DPP4	103		103	SBNO1			1			
105	VSIG2	104	TLR9	105	TNNI3K	105	PLCL2	105	CTNND1	105	WDFY1			L	İ		
106	TYW5	106	PPARGC1A	106	CNKSR2	106	CEP170	106		106	SMS						
107	C2orf69	107	CCDC62	107	MPP6	107	KMT2E	107	VPS45	107	CD46						
108	PGAP1	108	MPL ADAMTEL 2	108	DNAH10	108	CBR4	108		108	ZSCAN16-A	51		<u> </u>		$\left - \right $	
109	GIGYF2 INPP5D	109	ADAMTSL3 PCDHA13	109 110	FAT2 ABCB9	109	ZSCAN23 AKT3	109 110	TBC1D5 ADGRA3	109	CCDC150 FAM83D						
110	INFF3D	110	FODRATS	110	ABCB9	110	ARTS	110	ADGRAS	110	MPHOSPH						
111	AMBRA1	111	HECW2	111	DQX1	111	PAIP2B	111	STAG1	111	9			L			
							FPGT-										
112	ZKSCAN4	112	PEX5L	112	DPP4	112	TNNI3K	112		112	DHX35						
113	LRRIQ3 TMEM110-	113	ASTN1	113	PLCL2	113	PTPRF	113	ZDHHC5	113	ZSWIM6						
114	MUSTN1	114	PCDHA12	114	PPP1R16B	114	FAM53C	114	GATAD2A	114	SF3B1						
115	MYO1A	115	ANKRD44	115	CPEB1-AS1	115		115		115							
116	OR2B2	116		116		116	IMMP2L	116		116	KMT5A						
117	PLXNA2	117		117		117		117	TMEM161A	117	ITGA9						
118	PCDHGA6	118		118		118		118		118							
119	PCDHGB3	119	SNAP91	119	ADSS	119	SLC25A42	119	DUS2	119	ZNF691						
119 120	PCDHGB3 PCDHGA4	119 120	SNAP91 LYPD6B	119 120	ADSS DPYD	119 120	SLC25A42 AOX2P	119 120	DUS2 ENO1	119 120	ZNF691 ZNF48						
119	PCDHGB3 PCDHGA4 PCDHGA5	119	SNAP91 LYPD6B DNAJC6	119	ADSS DPYD HTRA2	119	SLC25A42	119 120 121	DUS2 ENO1 TMEFF2	119	ZNF691						
119 120 121	PCDHGB3 PCDHGA4	119 120 121	SNAP91 LYPD6B	119 120 121	ADSS DPYD	119 120 121	SLC25A42 AOX2P UGT1A6	119 120	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12	119 120 121	ZNF691 ZNF48 FANCL						
119 120 121 122 123 124	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E	119 120 121 122 123 124	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34	119 120 121 122 123 124	ADSS DPYD HTRA2 MOB4 IBTK PDE6D	119 120 121 122 123 124	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3	119 120 121 122 123 124	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A	119 120 121 122 123 124	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL						
119 120 121 122 123 124 125	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1	119 120 121 122 123 124 125	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2	119 120 121 122 123 124 125	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2	119 120 121 122 123 124 125	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A	119 120 121 122 123 124 125	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5	119 120 121 122 123 124 125	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300						
119 120 121 122 123 124 125 126	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3	119 120 121 122 123 124 125 126	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3	119 120 121 122 123 124 125 126	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6	119 120 121 122 123 124 125 126	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2	119 120 121 122 123 124 125 126	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2	119 120 121 122 123 124 125 126	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A						
119 120 121 122 123 124 125 126 127	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B	119 120 121 122 123 124 125 126 127	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4	119 120 121 122 123 124 125 126 127	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3	119 120 121 122 123 124 125 126 127	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2 EBNA1BP2	119 120 121 122 123 124 125 126 127	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1	119 120 121 122 123 124 125 126 127	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT						
119 120 121 122 123 124 125 126	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3	119 120 121 122 123 124 125 126	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4	119 120 121 122 123 124 125 126	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3	119 120 121 122 123 124 125 126	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2 EBNA1BP2	119 120 121 122 123 124 125 126	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2	119 120 121 122 123 124 125 126 127	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A						
119 120 121 122 123 124 125 126 127	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B	119 120 121 122 123 124 125 126 127	SNAP91 LYP06B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT	119 120 121 122 123 124 125 126 127	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2	119 120 121 122 123 124 125 126 127	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2 EBNA1BP2 DPH3	119 120 121 122 123 124 125 126 127	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4	119 120 121 122 123 124 125 126 127	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1						
119 120 121 122 123 124 125 126 127 128 129	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4	119 120 121 122 123 124 125 126 127 128 129	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR	119 120 121 122 123 124 125 126 127 128 129	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2	119 120 121 122 123 124 125 126 127 128 129	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYQ1A OR2B2 EBNA1BP2 DPH3 IK	119 120 121 122 123 124 125 126 127 128 129	DUS2 EN01 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C-	119 120 121 122 123 124 125 126 127 128 129	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2						
119 120 121 122 123 124 125 126 127 128 129 130	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1	119 120 121 122 123 124 125 126 127 128 129 130	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IIL34 SRPK2 ERI3 NEK4 PEMT SRR USP40	119 120 121 122 123 124 125 126 127 128 129 129	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTNND1	119 120 121 123 124 125 126 127 128 129 129	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2 EBNA1BP2 DPH3 IK FAM114A2	119 120 121 122 123 124 125 126 127 128 129 129	DUS2 EN01 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- IT3	119 120 121 122 123 124 125 126 127 128 129 130	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A						
119 120 121 122 123 124 125 126 127 128 129 130 131	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR	119 120 121 122 123 124 125 126 127 128 129 129 130 131	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8	119 120 121 122 123 124 125 126 127 128 129 130 131	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRI03 BOLA2 OGFOD2 CTINID1 TTYH3	119 120 121 123 124 125 126 127 128 129 129 130 131	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3	119 120 121 122 123 124 125 126 127 128 129 129 130 131	DUS2 EN01 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- IT3 PDE4D	119 120 121 122 123 124 125 126 127 128 129 129 130 131	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4						
119 120 121 122 123 124 125 126 127 128 129 130	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1	119 120 121 122 123 124 125 126 127 128 129 130	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8	119 120 121 122 123 124 125 126 127 128 129 129	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTNND1	119 120 121 123 124 125 126 127 128 129 129	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MYO1A OR2B2 EBNA1BP2 DPH3 IK FAM114A2	119 120 121 122 123 124 125 126 127 128 129 129	DUS2 EN01 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- IT3 PDE4D	119 120 121 122 123 124 125 126 127 128 129 129 130 131	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A						
119 120 121 122 123 124 125 126 127 128 129 130 131 132 133	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7orf50	119 120 121 123 124 125 126 127 128 129 130 131 132 133	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9	119 120 121 122 123 124 125 126 127 128 129 130 131 132	DUS2 EN01 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- IT3 PDE4D CACNA1C ADAMTS9- AS2	119 120 121 122 123 124 125 126 127 128 129 130 131 132	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-0T MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF						
119 120 121 122 123 124 125 126 127 128 129 130 131 131 132 133	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGF0D2 CTNND1 TTYH3 C120rf65 C7orf50 PSKH1	119 120 121 123 124 125 126 127 128 129 130 131 132 133 134	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- IT3 PDE4D CACNA1C- IT3 PDE4D CACNA1C ADAMTS9- AS2 PRUNE1	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI	3K					
119 120 121 122 123 124 125 126 127 128 129 129 130 131 132 133 134 135	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3	119 120 121 122 123 124 125 126 127 128 129 129 130 131 132 133 134 135	ADSS DPYD DPYD HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRI03 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7arf50 PSKH1 TBC1D5	119 120 121 122 123 124 125 126 126 127 128 127 128 130 131 132 133 134 135	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7of31	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NTSC2 NTSC2 NTSC2 NTSC2 NTSC2 TOM1L2 CACNA1C- IT3 PDE4D CACNA1C ADAMTS9- AS2 PRUNE1 CDHR3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A ADSL EP300 KDM4A ADSL SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI ARL6IP4						
119 120 121 122 123 124 125 126 127 128 129 130 131 131 132 133	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTNND1 TTYH3 C120rf65 C7orf50 PSKH1 TBC1D5 VPS45	119 120 121 123 124 125 126 127 128 129 130 131 132 133 134	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NTSC2 NTSC2 NTSC2 NTSC2 NTSC2 TOM1L2 CACNA1C- IT3 PDE4D CACNA1C ADAMTS9- AS2 PRUNE1 CDHR3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI						
119 120 121 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK RTKN FAM114A2	119 120 121 123 124 125 126 127 128 129 130 131 132 133 134 135 136 136 137	SNAP91 LYPD6B DNALC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6	119 120 121 123 124 125 126 126 127 128 129 129 130 131 132 133 134 135 136 136 137	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGF0D2 CTNND1 TTYH3 C1207f65 C707f50 PSKH1 TBC1D5 VPS45 ITGA9	119 120 121 122 123 124 125 126 127 128 129 130 131 131 132 133 134 135 136 137	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7or31 EFHD1 TMTC1	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137	DUS2 ENO1 TMEFF2 GPM6A PCDH6A12 GRIN2A SLC2A5 NT5C2 ONEK1 TOM1L2 CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CACNA1C CDHR3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-0T MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI ARLEIP4 ANKHD1 SMG1P2						
119 120 121 122 123 124 125 126 127 128 129 129 130 131 132 133 134 135 136	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK RTKN	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13	119 120 121 122 123 124 125 126 127 128 129 129 130 131 132 133 134 135 136	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRI03 BOLA2 OGFOD2 CTNND1 TTYH3 C120rf65 C7orf50 PSKH1 TBCD5 VPS45	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC36G2 HSPA9 WDR55 C7orf31 EFHD1	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- IT3 PDE4D CACNA1C- IT3 PDE4D CACNA1C- CACNA1C- CACNA1C- DHR3 PCDHAC2 GABRAT	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136	ZNF691 ZNF48 FANCL HIRIP3 INO80E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI ARL6IP4 ANKHD1	3K					
119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK RTKN FAM114A2 GTF2H3 SFMBT1	119 120 121 122 123 124 125 126 126 127 128 129 129 130 131 131 132 133 134 135 136 137 138 139	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6 AP153 NGEF	119 1200 1221 1222 123 124 1255 126 127 128 1300 1311 1322 1333 1344 1355 1366 1377 1388 1399	ADSS DPYD DPYD DPYD HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRI03 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7orf50 PSKH1 TBC1D5 VPS45 ITGA9 RNF220 ATF7IP2	119 120 121 122 123 124 125 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7orf31 EFHD1 TMTC1 PTCHD1-AS TSPAN9	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- AS4 CACNA1C- ADAMTS9- AS2 PRUNE1 CDHR3 PCDHAC2 GABRT1 LINC00634 SPATS2L	119 120 121 122 123 124 125 126 127 128 129 120 131 131 132 133 134 135 136 137 138 139	2NF691 2NF48 FANCL HIRIP3 IN080E ADSL EP300 KDMAA KOMAA SOX2-OT MAD111 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI ARL6IP4 ANKHD1 SMG1P2 SATB2 SATB2 SATB2 SATB2 SAP30-	3K					
119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SFMBT1 ERI3	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6 AP153 NGEF DGKZ	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140	ADSS DPYD HTRA2 MOB4 IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTNND1 CTYPH3 C120rf65 C7orf50 PSKH1 TBC1D5 VPS45 ATF7IP2 GIGYF2	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7orf31 EFHD1 TMTC1-AS TSPAN9 PCDHA12	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140	DUS2 ENO1 TMEFF2 GPM6A PCDH6A12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- AS4 CACNA1C- ADAMTS9- AS2 PRUNE1 CDHR3 SPATS2L WDR81	119 120 121 123 124 125 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139 140	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNN1 ARL6IP4 ANKHD1 SMG1P2 SAT82 ATF7IP2 SAT820L- AS1	3K					
119 120 121 122 123 124 125 126 127 128 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139 140 141	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SFMBT1 ERI3 SLC6A9	1199 1200 121 1222 123 124 125 126 127 128 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139 139 139 140 141	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6 AP153 NGEF DGKZ EXOC6B	119 1200 121 122 123 124 126 126 127 128 126 127 128 130 131 131 132 133 134 135 136 137 138 138 139 139 139 140 141	ADSS DPYD DPYD DPYD HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRIG3 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7orf50 PSKH1 TBC1D5 VPS45 ITGA9 RNF220 ATF7IP2 GIGYF2 CCT7	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR2B2 EBNA1BP2 EBNA1BP2 FAM114A2 GTF2H3 JK FAM114A2 GTF2H3 SLC3S62 HSPA9 WDR55 C7ori31 EFHD1 TMTC1 PTCHD1-AS TSPAN9 PCDHA12 PCDHA12	119 120 121 122 123 124 125 126 127 128 127 128 129 130 131 132 134 135 136 137 138 139 140 141	DUS2 ENO1 TMEFF2 GPM6A PCDH6A12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- HT3 PDE4D CACNA1C- ADAMTS9- AS2 PRUNE1 CDHR3 PCDHAC2 GABBR1 LINC00634 SPATS2L WDR81 SNX8	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141	2NF691 2NF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A KDM4A SOX2-0T MAD1L1 MAD1L1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TINI ARL6IP4 ANKHD1 SATB2 ATF7IP2 SATB2 ATF7IP2 SATB2 ATF7IP2	3K					
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119 120 121 122 123 124 125 126 127 128 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139 140 141	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SFMBT1 ERI3 SLC6A9	1199 1200 121 1222 123 124 125 126 127 128 126 127 128 129 130 131 131 132 133 134 135 136 137 138 139 139 139 140 141	SNAP91 LYPD6B DNALC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRP514 FRMD8 GALNT10 PCNX3 AKAP13 SM66 AP153 NGEF DGKZ EXOC68 DPYD-AS11 PCDHGA3	119 1200 121 122 123 124 126 126 127 128 126 127 128 130 131 131 132 133 134 135 136 137 138 138 139 139 139 140 141	ADSS DPYD DPYD DPYD HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRIG3 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7orf50 PSKH1 TBC1D5 VPS45 ITGA9 RNF220 ATF7IP2 GIGYF2 CCT7	119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR2B2 EBNA1BP2 EBNA1BP2 FAM114A2 GTF2H3 JK FAM114A2 GTF2H3 SLC3S62 HSPA9 WDR55 C7ori31 EFHD1 TMTC1 PTCHD1-AS TSPAN9 PCDHA12 PCDHA12	119 120 121 122 123 124 125 126 127 128 129 130 131 132 134 135 136 137 138 139 140 141	DUS2 ENO1 TMEFF2 GPM6A PCDH6A12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- T3 PDE4D CACNA1C- CACNA1C- T3 PDE4D CACNA1C- AS2 PRUNE1 CDHR3 PCDHAC2 GABBR1 LINC00634 SPATS2L WDR81 SNX8 CTS8 DOCK10	119 120 121 122 123 124 125 126 127 128 130 131 131 132 133 134 135 136 137 138 139 140 141	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-0T MADIL1 RFTN2 SLX1A CNTN4 AKT3 PTPRF FPGT-TNNI ARL61P4 ANKHD1 SMG1P2 SAT82 ATF71P2 SAP30L- AS1 MIR3188 PDE6D TOX	ЗК					
119 120 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141 142 143	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK EBNA1BP2 DPH3 IK EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SFMBT1 ERI3 SLC6A9 ACTR8 EXOC6B	119 120 121 122 123 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141 141 143 1445	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6 AP153 NGEF DGKZ EXOC68 DPYD-AS1 PCOHGA3 CREB3L1 DYSF	119 120 121 122 123 124 125 126 127 128 128 129 129 131 131 132 133 134 135 136 137 138 139 139 140 141 141 142 143 144	ADSS DPYD HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRIQ3 BOLA2 OGFOD2 CTINND1 CTTYH3 C120rf65 C7orf50 PSKH1 TBC1D5 VPS45 ITGA9 RNF220 ATF7IP2 GIGYF2 CCT7 ADSL INO80E ALMS1 ZNF48	119 120 121 122 122 123 124 126 127 128 126 127 128 130 131 131 132 133 134 135 136 137 138 138 139 140 141 142 143 144 145	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRIQ3 MY01A OR2B2 EBNA1BP2 DPH3 IK FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7orf31 EFHD1 TMTC1 PTCHD1-AS TSPAN9 PCDHA12 PCDHA12 CCDC24 DAZL CCDC39 CFAP57	119 1200 121 122 123 124 125 126 127 128 126 127 128 130 131 132 133 134 135 136 137 138 139 140 141 142 143	DUS2 ENO1 TMEFF2 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 PDE40 CACNA1C- AS4 PDE40 CACNA1C- AS2 PRUNE1 CDHR3 SPATS2L WDR81 SNX8 CTSS DOCK10 LRP1	119 120 121 122 123 124 126 126 127 128 130 131 131 132 133 134 135 136 137 138 138 139 140 141 142 143 144 145	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-0T MAD1L1 RFTN2 SLX1A CNTN4 AKT3 AKT3 AKT3 AKT3 AKT3 AKT3 AKT4 SATB2 ATF7P2 SA7930L- AS1 MIR3188 PDE6D TOX ADSS DPYD	3K					
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119 120 121 121 122 123 124 125 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 142 143 144 145 144 145 144 145 144 145 146 147 148 147 150 152 156 157 158 159 159	PCDHGB3 PCDHGA4 PCDHGA5 CBR4 ZSCAN23 KMT2E RC3H1 AKT3 PAIP2B BOLA3 MOB4 DCTN1 DENR VPS33A EBNA1BP2 DPH3 IK RTKN FAM114A2 GTF2H3 SFMBT1 ERI3 SLC6A9 ACTR8 EXOC6B C11orf49 FER SFXN5 WBP1L PCDHG81 PCDHG82 PCDHG82 PCDHG82 PCDHG82 PCDHG7 AOX2P ARL14 UGT1A7 DGKZ AOX1 ESAM	119 121 121 121 122 121 122 122 122 122 122 122 122 122 122 122 122 122 122 122 122 122 122 122 131 132 133 133 133 133 134 133 133 133 133 133 133 133 133 134 135 137 138 139 141 142 144 144 144 155 158	SNAP91 LYPD6B DNAJC6 SH3GL3 R3HDM2 IL34 SRPK2 ERI3 NEK4 PEMT SRR USP40 ACTR8 MRPS14 FRMD8 GALNT10 PCNX3 AKAP13 SMG6 AP153 NGEF DGKZ EXOC68 DPYD-AS1 DYSF MBTP52 HCN1 INPP4B ST3GAL3 DDK60L PSMB10 RWDD2A CHDH PCDHG84 PCDHG84 PCDHG84 PCDHG84 PCDHG84 PCDHG84 PCDHG84 PCDHG84 SNORD63	119 121 121 122 123 124 125 126 127 128 120 121 123 124 125 127 128 130 131 132 133 134 135 138 139 141 142 143 144 145 144 145 144 145 152 153 154 155 156 157 158 159	ADSS DPYD HTRA2 HTRA2 MOBA IBTK PDE6D SATB2 TSSK6 LRRI03 BOLA2 OGFOD2 CTNND1 TTYH3 C12orf65 C7orf50 PSKH1 TTYH3 C12orf65 C7orf50 PSKH1 TTYH3 C12orf65 C7orf50 PSKH1 TBC1D5 VPS45 ITGA9 RNF220 ATF7IP2 GIGYF2 CCT7 ADSL ING80E ALMS1 ZNF48 FANCL HIRIP3 ZNF691 EF300 KDM4A MAD1L1 SOX2-0T SMS SF3B1 KMT5A NFATC3 MOB1A WDFY1 CCDC150	119 121 1221 1233 1244 1255 1277 1289 1311 1322 1333 1344 1355 1366 1377 1389 1411 1442 1447 1441 1445 1447 1441 1455 1566 1577 158 1566 1577 158 159 159	SLC25A42 AOX2P UGT1A6 MIR3160-1 UGT1A8 LRRI03 MY01A OR282 EBNA18P2 EBNA18P2 BNA18P2 EBNA18P2 FAM114A2 GTF2H3 SLC35G2 HSPA9 WDR55 C7orf31 EFHD1 TMTC1 PTCHD1-AS TSPAN9 PCDHA42 CCDC24 DAKRD44 CCDC239 CFAP57 MIR137HG PCDHGA1 NYAP2 SLC4A10 ANKRD44 CACNA1D LINC01122 ZINF804A PCDHA6 PCDHA6 <td>119 120 121 121 122 121 122 123 124 126 127 128 126 127 128 126 127 128 130 131 133 134 133 134 133 134 139 136 139 137 138 139 140 1411 1422 143 1441 145 151 156 152 1556 157 158 156 1567 157 158 159 159</td> <td>DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- TOM1L2 PDE4D CACNA1C- DADATS9 AS4 CACNA1C- CACNA1C JPE4D CACNA1C- DOHR3 PRUNE1 CDHR3 PCDHAC2 GABBR1 MIR22HG PFKFB2 PHKPS TLCD2 SNORD32B ITH3 CDDAG30 ZSCAN31 TH44 FAM124B CR1L</td> <td>119 120 121 123 124 125 126 127 128 120 121 123 1245 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141 142 144 145 144 145 144 145 144 145</td> <td>ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A AKT3 PTPRF FPGT-TNNI AKT3 ATCSTP2 SAT82 ATCSTP2 ATCSTP2 SAT82 ATCSTP2 ATCSTP2 SAT82 ATCSTP2 ATCST</td> <td>31</td> <td></td> <td></td> <td></td> <td></td> <td></td>	119 120 121 121 122 121 122 123 124 126 127 128 126 127 128 126 127 128 130 131 133 134 133 134 133 134 139 136 139 137 138 139 140 1411 1422 143 1441 145 151 156 152 1556 157 158 156 1567 157 158 159 159	DUS2 ENO1 TMEFF2 GPM6A PCDHGA12 GRIN2A SLC2A5 NT5C2 NEK1 TOM1L2 CACNA1C- AS4 CACNA1C- TOM1L2 PDE4D CACNA1C- DADATS9 AS4 CACNA1C- CACNA1C JPE4D CACNA1C- DOHR3 PRUNE1 CDHR3 PCDHAC2 GABBR1 MIR22HG PFKFB2 PHKPS TLCD2 SNORD32B ITH3 CDDAG30 ZSCAN31 TH44 FAM124B CR1L	119 120 121 123 124 125 126 127 128 120 121 123 1245 126 127 128 129 130 131 132 133 134 135 136 137 138 139 140 141 142 144 145 144 145 144 145 144 145	ZNF691 ZNF48 FANCL HIRIP3 IN080E ADSL EP300 KDM4A SOX2-OT MAD1L1 RFTN2 SLX1A AKT3 PTPRF FPGT-TNNI AKT3 ATCSTP2 SAT82 ATCSTP2 ATCSTP2 SAT82 ATCSTP2 ATCSTP2 SAT82 ATCSTP2 ATCST	31					

							PSMG3-			Т	1		260
163		163 ALAS1 164 HARS2	163 LOXL3 164 ZSWIM6	163 164		163 164	AS1 PPP2R3A		 -				260
165		165 OXNAD1 166 PGM3	165 C2orf69 166 CUL3	165		165 166	C3orf49 PCDHA1						
167	MBTPS2	167 PCDHGC3	167 SBNO1	166 167	FTCDNL1	167	PCDHA2						
168		168 PCDHGB6 169 PITPNM2	168 MIR3188 169 RC3H1	168 169		168 169	FSIP2 PCDHA11		 				
170	USP38	170 ATG13	170 DAAM1	170	GRIN2A	170	SNORD63						
171	AP1S3	171 COQ10B	171 CD46 ZSCAN16-	171	CCDC30	171	CSMD1			_		 	
172		172 SNX8	172 AS1	172		172							
173		173 SPATS2L 174 MIR22HG	173 SAP30L-AS1 174 FOXP1	173		173	ADIG PCDHA10			_			
175	NPY6R	175 TLCD2	175 TOX	175	TFAMP1	175	PCDHA5						
176		176 PALLD 177 PCCB	176 ARL6IP4 177 ANKHD1	176		176 177				_		 	
178	KCNH7	178 SPARC	178 SMG1P2	178	PEX5L	178	ZFP57						
179	MPP6	179 SLC12A4	179 AKT3 FPGT-	179	SRPK2	179	SMPX		 	_		 	
180		180 CTNNA1	180 TNNI3K	180		180				_			
181	STK31 C7orf31	181 RFTN1 182 PCDHGA6	181 PTPRF	181 182		181 182							
183		183 PCDHGB3			ITIH3	183							
184		184 PCDHGA4 185 PCDHGA5			ITIH4 ASB5	184 185							
180	PCDHA7	186 SERPINF1 187 PCDHGB1		186 187	SEC16B TMEFF2	186 187	PCDHA9 HECW2						
187		188 PCDHGB1		187		188							
189		189 ACTG1P17 190 LINC01004			PHEX SLC2A5	189	FTCDNL1 SYNPR			_			
		TMEM110-				190							
191 192	PLD5 FTCDNL1	191 MUSTN1 192 C1orf116		191 192	GALNT15 LRRC43	191 192	PLD5 PCDHAC1					<u> </u>	
193	ZNF391	193 MYO1A		193	CLU	193	ZBED9						
194		194 OGFOD2 195 BOLA2		194 195		194 195	MIR137HG ARHGAP40						
196	SERPINC1	196 CTRL		196	PDE4D	196	LINC01122						
197		197 LRRIQ3 198 MIR33B		197 198	DOCK10 GPM6A	197 198	LYPD6B SH3GL3						
199	PARP8	199 LAPTM5		199	PCDHGA11	199	ZNF804A						
200	PPP2R3A PCDHA10	200 ARHGAP15 201 SLX1A		200		200							
202		202 LINC00698 203 PCDHGA7		202		202	SNAP91 ANKRD44						
203		203 PCDHGA7 204 GFRA3		203		203 204	DGKD						
205	PCDHGA1 DCTN1-AS1	205 SEMA3G 206 KMT2E		205		205 206	SRPK2 MRAP2		 				
207	CACNA2D3	207 SEMA6C		207	PITPNM2	207	C2orf82						
208	MIR137HG	208 NT5M		208	CLIP1	208	SH3PXD2A PTCHD1-			_			
209		209 PLCL2		209		209	AS						
210		210 PLPPR5 211 RNF220			SH3RF1 PCDHGB6	210 211	PCDHA12 PCDHA4			_			
212	PSD3	212 RAI1		212	TVP23A	212	PCDHA13						
213		213 PLEKHO1 214 RERE			CLCN3 TCTN2	213 214	ADAMTSL3 PEX5L			_			
215		215 DIS3L2			RILPL2	215	ASTN1						
216		216 DCTN1 217 EPC2			SNX8 RFTN1	216	BRINP2 R3HDM2						
218		218 KCNJ13 219 MYO15A		218 219		218 219	CCDC62 PPARGC1A			_			
220	CCNH	220 TNNI3K		220	GRIA1	220	MOG						
221	LINC00461 RASA1	221 DPEP3 222 MIR4677		221		221	MPL GPX5			_	1		
223		223 CNNM2		223		223	SLC2A7						
224	SLC25A42	TMX2- 224 CTNND1		224	ST3GAL3	224	TLR9						
225		225 RFTN2		225	CYP26B1	225	PCDHGA11 PCDHGB7						
226	MIR3160-1	226 TTC14 227 ABCB9		227	PCDHGA2 PCDHGA7	227	PCDHGB4						
228	UGT1A8 PKD2L2	228 RTN4RL1 229 CPEB1-AS1			DGKZ MBTPS2		PCDHGA1 GRM3						
230	SEMA3G	230 AP3B2		230	VRK2	230	BOLL						
231		231 GLRA1 232 FAM57B			DDX60L GSDME	231	STAC3 NDUFA4L2				<u> </u>		
233	BRINP2	233 PLCL1		233	AOX1	233	TMX2-CTNN	ID1					
234		234 PCDHA3 235 CNKSR2			ESAM PCDHGB4		CNNM2 RFTN2					<u> </u>	
236	ALMS1P1	236 PSD3		236	PCDHGB7	236	TTC14						
237		237 NLGN4X 238 PRKD1			GALNT10 CA6		GFRA3 VTRNA1-1						
239	DAZL	239 EPHA7		239	HCN1	239	LINC00698						
240 241	PCDHA12	240 SFMBT1 241 G3BP1		241	FCHSD1 RELL2	241	SEMA3G LAPTM5						
	HECW2	242 CDC20		242	KCNJ13 PKD2L2	242 243	MIR33B SNORD63						
244	BRINP3	244 CCDC150		244	PPARGC1A	244	SLX1A						
245		245 MPHOSPH9		245	BNIPL	245				-			
	LINC01122	246 POC1A			IQCF1	246	MUSTN1						
	RABGAP1L GPR52	247 SSRP1 248 CENPL			UGT1A10 UGT1A9	247 248	MYO1A C1orf116						
249	NYAP2	249 ANP32E		249	IL19	249	OGFOD2						
250		250 NEMP1 251 IPO11			UGT1A7 PCDHGA8	250 251	BOLA2 CTRL			_			
252	CACNA1D	252 PCGF6		252	PCDHGA9	252	LINC01004						
253	KLHL29	253 CCT7		253	DPYD-AS1	253	DPEP3			1	I		

255	MEF2C	254	SF3B1		254	PCDHGA3	254									
	EMX1	255	NFATC3		255	PCDHA10	255	KCNJ13								
	PCDHGA12	256	DESI2		256	PCDHA5	256	TNNI3K								
	DOCK10	257	POLDIP3		257	C4BPA	257	PCDHA3								
258	MSRA	258	RPS6KA3		258	SMPX	258	CNKSR2								
259	GPM6A	259	HDAC2		259	BOLL	259	ZKSCAN3								
260	SEC16B	260	ACTR5		260	SLC17A7	260	ABCB9								
261	GRIN2A	261	TAF5		261	GPX5	261	RTN4RL1								
262	TNFSF4	262	BAZ2A		262	GPX6	262	KCNH7								
263	GALNT15	263	MSL2		263	RFTN2	263	GLRA1								
264	LRRC43	264	CD46		264	SDCCAG8	264	FAM57B								
265	CLU	265	KMT2E-AS1		265	TNNI3K	265	PLCL1								
266	REEP2	266	GNL3		266	PCDHA3	266	PCDHGB1								
267	AIG1	267	PPP2R2A		267	EPHB1	267	PCDHGB2								
268	CACNA1C	268	MAP3K11		268	NCAN	268	PCDHGA2								
269	PDE4B	269	SMS		269	CNKSR2	269	PCDHGA7								
270	PDE4D	270	MDK		270	PLPPR5	270	DPYD-AS1								
271	FAM13B	271	BNIP3L		271	NMUR2	271	PCDHGA3								
	PHEX	272	MOGS		272	MYOT	272	CREB3L1								
	SLC2A5	273	TMEM161A		273	NPY6R	273									
	TMEFF2	274	FXR1		274	FAT2		MBTPS2								
	SCG2	275	GID4		275	STK31	275									
	TRANK1	276	TYW5		276	GLRA1	276									
	DGKD	277	C2orf69		277	PLCL1	277	ERI3								
	BOLA3-AS1	278	CUL3		278	KCNH7	278	NEK4								
	SRPK2	279	BAHD1	-	279	MPP6	279	SRR							1	
	INO80B	280	SBNO1		280	ATP2A2	280	PEMT			-					
	RILPL2	281	STAG1		281	ERI3	281	AKAP13								
	SPATA24	282	ADGRA3		282	SLC6A9	282								1	
	PER3	283	KDM4A		283	PPP2R3A	283	ZNF592								
	TFAMP1	283	SUFU		283	TCF4	283	DDX60L								
	CCDC39	285	PBRM1		285	PCDHA1	285	PSMB10								
	CFAP57	285	ALMS1		285	PCDHA1 PCDHA2	285				-					
	NME5	280	FANCL		280	C11orf49		ATXN7			-					
	NISCH	287	SOX2-OT		287	FER	287									
			MAD1L1			PCDHGA10										
	PCDHGB6	289 290	VPS37B		289 290	WBP1L	289 290	HSPA9 HARS2			-	<u> </u>				
	GAB1 ANKRD45	290 291	DNAJC19		290	WOPIL	290	STAT6			-	<u> </u>				
	TCTN2	292	DARS2		-		292				-					
	NEK1	293	SFXN2 THOC7				293		_		-					
	NT5C2	294					294									
	DNAJC18	295	PGAP1				295						L			
	PCDHAC2	296	RTN4RL2				296								I	l
	CCDC24	297	RASAL2				297									
	HYDIN	298	DDHD2					CTNNA1							L	
	IQCE	299	TOX					RFTN1								
	CCDC30	300	PHF21A				300									
	SLC9C2	301	P2RX3				301	COQ10B								
	MFAP3	302	PTBP2				302	PITPNM2								
	CLCN3	303	AMBRA1				303	AP1S3								
304	SNX8	304	C7orf50				304	CA6								
	IL17RB	305	HARS				305	HCN1								
306	TCF4	306	PRADC1				306	PCDHGA9								
	CHDH	307	EIF4E2				307	PCDHGA8								
308	PCDHGA10	308	KAT5				308	SNORD63								
309	TMTC1	309	PDE6D				309	IL19								
310	PTCHD1-AS	310	PARK7				310	ITIH1								
	TSPAN9	311	TWF2				311									
	CLIP1	312	DPH3				312									
	ITIH3	313	SLC25A33				313	GALNT10								
	ITIH4	314	MARS				314	YY2								
	TVP23A	315	ADSS												1	
	ASB5	316	CACYBP													
	BANK1	317	PSMG3										 		İ —	
	FCHSD1	318	SNX7										 		İ —	
	RELL2	319	DUS2										 		İ —	
	KCNJ13	320	ENO1										 		İ —	
	SLC17A7	321	ELOVL1													
	SLC4A5	322	GATAD2A													
	ZBTB37	323	GLT8D1													
	BNIPL	324									-					
	IQCF1	325	SREBF1												1	
	PPARGC1A	326	CEP162												1	
	TNN	327											 		İ —	
	IMMP2L	328	H6PD	-											1	
	PCCB	329	PSKH1		-						-					
	GBF1	330	ALKBH5		-						-					
	ST3GAL3	331	MED8													
	GRIA1	332	DUSP11													
	MAN2A1	333	SLC35G2													
	CREB3L2	334	ARL6IP4		-						-					
	PALLD	335	GAS5		-											
	PCDHGA8	336	LEPR													
	PCDHGA8 PCDHGA9	330									-					
			SMG1P2													
	CYP26B1 BOLL	338	GIGYF2 INPP5D		-											
		339					-	-		-	-					
	IL19	340	FAM53C		-						-					L
	DDX60L	341	SOX2		-						-					L
	SDCCAG8	342	FPGT-TNNI3K		-						-					L
	TNNC1	343	PTPRF										l	ļ	I	
344	PCDHGA11	344	AKT3										l	ļ	I	
C	PCDHGB4	345	RRP7BP										L		L	L
345	PCDHGB7	346	SERHL2										L			
345 346			CBR4				1					1		1	1	1
345 346 347	HSPA9	347														
345 346 347 348	HSPA9 ALAS1	348	COX20													
345 346 347 348 349	HSPA9															

351	SH3RF1	351	FOXP1								000
352	PITPNM2	352	PLXNA2								262
353	COQ10B	353	DPYD								-
354	CYSTM1	354	PLCB2								

Table 3.17 | List of genes shown in RNA correlation heatmaps.

List of genes (by row) in the order they appear in each cell type's full connectome and STRING RNA Pearson correlation heatmaps (See Figure 3.10 and Figure 3.12, 3.17-19).

		length after	absolute correlation	# of	P-value	P-value (distance	P-value (full v.	P-value (full v. string; string
list name	length	deletions	mean	samplings/permutations	(random)	constraint)	string)	removed from random)
NPC vs COS	308	290	0.2455	1000000, 1000	< 0.000001	< 0.001		
NEU vs COS	358	314	0.2329	1000000, 1000	< 0.000001	< 0.001		
GLIA vs COS	165	151	0.2066	1000000, 1000	0.00001	0.264		
string NPC	77	77	0.2963	100000	< 0.000001		0.007	< 0.001
string NEU	73	73	0.2877	100000	< 0.000001		0.008	
string GLIA	31	31	0.2225	10000	0.02		0.595	
string NPC medium	139	138	0.2725747	100000	< 0.000001		0.012	
string NEU medium	138	138	0.2654931	1000000	< 0.000001		0.003	
string GLIA medium	58	58	0.2183428	10000	0.001		0.434	

Table 3.18 | Summary of results from RNA-seq sampling/permutation anslyses.

Summary of results from RNA-seq sampling/permutation analyses (see Materials and methods, "RNA transcriptomic correlation heatmaps"). List name, list being compared against a background (COS = Childhood Onset Schizophrenia cohort); length, number of genes in the input list; length after deletions, number of genes remaining after filtering out genes with CPM < 1 across 30% of individual RNA-seq experiments; absolute correlation mean, the organization score for each heatmap; # of samplings/permutations, (random analysis, distance-constrained analysis); P-value (random), P-value from randomized sampling analysis without distance constraint; P-value (distance constraint), P-value calculated from distance-constrained randomized sampling (x1,000) analysis (featured in Fig 3.10, D); P-value (full v. string), P-value from the comparison between STRING heatmaps against those from the full connectome background (featured in Fig 3.13, B); P-value (full v. string; string removed from random), P-value from a version of the comparison that removed the subset STRING genes from the full connectome gene list before randomly sampling 1000 times

NPC 1	NPC 2	NEURON 1	NEURON 2	GLIA 1	GLIA 2
EIF2B1	UGT1A6	PCDHA1	SFMBT1	ANKRD44	OGFOD2
CUL3	PEX5L	FSIP2	G3BP1	SNAP91	CTNND1
					TTYH3
MARS2	BRINP2	PCDHA2	CDC20	BRINP3	
DNAJC19	ASTN1	CSMD1	CDC25C	ZNF804A	C12orf65
ORC5	TNR	PCDHA11	CCDC150	RABGAP1L	C7orf50
ZEB2	ALMS1P1	CHRNA3	MPHOSPH9	LINC01122	PSKH1
PHF21A	NAT8	ATPAF2	POC1A	SLC4A10	TBC1D5
ZBTB18	PCDHA4	PCDHGA10	SSRP1	MIR137HG	VPS45
ITGA9	DAZL	IL17RB	CENPL	GRAMD1B	ITGA9
ALMS1	CSGALNACT1		ANP32E	OPCML	RNF220
TET3	PCDHA12	C3orf49	NEMP1	CSMD1	ATF7IP2
CCDC150	HECW2	PPP2R3A	IPO11	HECW2	GIGYF2
MPHOSPH9	PCDHA13	BRINP3	PCGF6	PLD5	
					CCT7
KNTC1	BRINP3	FBXO41	CCT7	DOCK10	ADSL
POC1A	ZNF804A	LINC01122	SF3B1	CHRM3	INO80E
ANP32E	LINC01122	GRM3	NFATC3	GPM6A	ALMS1
CDC25C	RABGAP1L	PCDHGA1	DESI2	TRANK1	ZNF48
SGO2	GPR52	PCDHA6	POLDIP3	GALNT15	FANCL
CENPL	NYAP2	PCDHA8	RPS6KA3	PDE4B	HIRIP3
PPIH	SLC4A10	PCDHA7	HDAC2	PTCHD1-AS	ZNF691
BRD8	ANKRD44	PCDHA9	ACTR5	UTS2	EP300
SUGP1	CACNA1D	PCDHAC1	TAF5	C1orf100	KDM4A
CD46	KLHL29	PLD5	BAZ2A	EMX1	MAD1L1
EPB41	MEF2C	KCNH7	MSL2	ALMS1P1	SOX2-OT
RSRC2	EMX1	SYNPR	CD46	NAT8	SMS
GTF3C3	PCDHGA12	CA8	KMT2E-AS1	HYDIN	SF3B1
SBNO1	DOCK10	FTCDNL1	GNL3	SLC9C2	KMT5A
PPP1CC	MSRA	NTM	PPP2R2A	NEK1	NFATC3
SF3B1	GPM6A	OPCML	MAP3K11	GRIN2A	MOB1A
TAF5	SEC16B	ARHGAP40	SMS	PLXNA4	WDFY1
ZSWIM6	GRIN2A	MIR137HG	MDK		CCDC150
RAD54L2	TNFSF4	EFHD1	BNIP3L		FAM83D
RPS6KA3	GALNT15	SERPINE2	MOGS	ļ	DHX35
RANGAP1	LRRC43	PCDHGA2	TMEM161A	ļ	MPHOSPH9
SUGP2	CLU	PCDHGA8	FXR1		LOXL3
ARNT	REEP2	PCDHGA9	GID4		ZSWIM6
FXR1	AIG1	NDUFA4L2	TYW5		C2orf69
MDK	CACNA1C	STAC3	C2orf69		CUL3
NCK1	PDE4B	IL19	CUL3		SBNO1
TKT	PDE4D	ITIH1	BAHD1		MIR3188
NUBP1	FAM13B	BOLL	SBN01		RC3H1
HDAC3	PHEX	EGR4	STAG1		DAAM1
PIH1D1	SLC2A5	SDCCAG8	ADGRA3		CD46
PSMD14	TMEFF2	PCDHA10	KDM4A		ZSCAN16-AS
EIF3B	SCG2	PCDHA5	SUFU		SAP30L-AS1
GATAD2A	TRANK1	MIR339	PBRM1		FOXP1
SNX7	DGKD	SMPX	ALMS1		тох
CETN3	BOLA3-AS1	CYP26B1	FANCL		ARL6IP4
FGFR1	SRPK2	ADIG	SOX2-OT		ANKHD1
PPP2R2A	INO80B	MIR548AI	MAD1L1		SMG1P2
DESI2	RILPL2	NT5C2	VPS37B		AKT3
NFATC3	SPATA24	ALMS1P1	DNAJC19		FPGT-TNNI3k
					PTPRF
SMS	PER3	NAT8	DARS2		PIPKF
NOSIP	TFAMP1	PHEX	SFXN2		
PCGF6	CCDC39	TOM1L2	THOC7	l	l
FAM53C	CFAP57	MSRA	PGAP1		
DUS2	NME5	NEK1	RTN4RL2		
RFT1	NISCH	CCDC39	RASAL2		
PRDX6	PCDHGB6	ANKRD45	DDHD2		
ADSS	GAB1	DCDC1	тох		i
ETF1	ANKRD45	PTCHD1-AS	PHF21A		1
HSPD1	TCTN2	FAM124B	P2RX3		
	NEK1	ITIH4	PTBP2		
	INCIVI				
	NITECO				
MAD1L1	NT5C2	C1orf100	AMBRA1		
MAD1L1 KDM4A	DNAJC18	CR1L	C7orf50		
MAD1L1 KDM4A KDM3B	DNAJC18 PCDHAC2	CR1L CTSS	C7orf50 HARS		
MAD1L1 KDM4A KDM3B SOX2-OT	DNAJC18 PCDHAC2 CCDC24	CR1L CTSS DOCK10	C7orf50 HARS PRADC1		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1	DNAJC18 PCDHAC2 CCDC24 HYDIN	CR1L CTSS DOCK10 LRP1	C7orf50 HARS PRADC1 EIF4E2		
MAD1L1 KDM4A KDM3B SOX2-OT	DNAJC18 PCDHAC2 CCDC24	CR1L CTSS DOCK10 LRP1 PFKFB2	C7orf50 HARS PRADC1		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE	CR1L CTSS DOCK10 LRP1 PFKFB2	C7orf50 HARS PRADC1 EIF4E2 KAT5		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30	CR1L CTSS DOCK10 LRP1	C7orf50 HARS PRADC1 EIF4E2		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2 MFAP3	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2 MFAP3 CLCN3	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 HPF1 ASH2L DARS2 VPS45 ABCD3 BCD5 B4GALT2	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4B PDE4D HIVEP2	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS		
WAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 HBC1D5 34GALT2 WAU2	DNAJC18 PCDHAC2 CCDC24 HYDIN QCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DAR52 VPS45 ABCD3 TBC1D5 BGGALT2 MAU2	DNAJC18 PCDHAC2 CCDC24 HYDIN QCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CCHDH	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C CACNA1C-AS4	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6	DNAJC18 PCDHAC2 CCDC24 HYDIN QCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2	DNAJC18 PCDHAC2 CCDC24 HYDIN IGCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C CACNA1C-AS4 CACNA1C-AS4 CACNA1C-AT3	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2	DNAJC18 PCDHAC2 CCDC24 HYDIN IQCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 LL17RB TCF4 CHDH PCDHGA10 TMTC1	CR1L CTSS DOCK10 LRP1 PFKFB2 CFEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-IS GALNT15	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7		
MAD1L1 KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DAR52 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2 RANBP10	DNAJC18 PCDHAC2 CCDC24 HYDIN QCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 PTCHD1-AS	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4B PDE4D HIVEP2 CACNA1C CACNA1C-AS4 CACNA1C-IT3 GALNT15 PRUNE1	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2		
MAD1L1 KDM4A KDM4A SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2 RANBP10 ATP6V0A2	DNAJC18 PCDHAC2 CCDC24 HYDIN QCE CCDC30 SLC962 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 TMTC1 PTCHD1-AS TSPAN9	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C CACNA1C-R34 CACNA1C-IT3 GALNT15 PRUNE1 ADAMTS9-AS2	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE60 PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2 ENO1		
MAD1L1 KDM4A KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DAR52 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2 RANBP10 ATP6V0A2 EP300	DNAJC18 PCDHAC2 CCDC24 HYDIN GCE CCDC30 SL09C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 PCDHGA10 PTCHD1-AS TSPAN9 CLIP1	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE40 PDE40 HIVEP2 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-FAS	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE60 PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2 ENO1 ELOVL1		
MAD1L1 KDM4A KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2 RANBP10 ATP6V0A2 EP300 PFGT-TNNI3K	DNAJC18 PCDHAC2 CCDC24 HYDIN OCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 TMTC1 PTCHD1-AS TSPAN9 CLIP1 TIH3	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4B PDE4B PDE4D HIVEP2 CACNA1C-A54 CACNA1C	C7orf50 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2 ENO1 ELOVL1 6ATAD2A		
MAD1L1 KDM4A KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADGRG6 SOX2 CMTR2 RANBP10 ATP6V0A2 EP300 EPG7-TNNI3K FPGF-TNNI3K	DNAJC18 PCDHAC2 CCDC24 HYDIN IGCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 TMTC1 PTCHD1-AS CLIP1 TIH3 TIH4	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-TS PRUNE1 ADAMTS9-AS2 EPN2 GPM6A TMEFF2	C70rt60 HARS PRADC1 EIF4E2 KAT5 PDE60 PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2 ENO1 ELOVL1 GATAD2A GLT8D1		
MAD1L1 KDM4A KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L DARS2 VPS45 ABCD3 TBC1D5 B4GALT2 MAU2 ADCRG6 SOX2 CMTR2 RANBP10 ATP6V0A2 EP300 FPGT-TNNI3K PTPRF DXNAD1	DNAJC18 PCDHAC2 CCDC24 HYDIN IGCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 TMTC1 PTCHD1-AS TSPAN9 CLIP1 TIH4 TVP23A	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE40 PDE40 PDE40 HIVEP2 CACNA1C CACNA1C-AS4	C70rf60 HARS PRADC1 EIF4E2 KAT5 PDE6D PARK7 TWF2 DPH3 SLC25A33 MARS SLC25A33 MARS SLC25A33 MARS SLC25A33 MARS ELC25A33 MARS ELC25A33 MARS ELC25A33 MARS ELC25A33 MARS ELC25A3 MARS ELC3CA3 MARS MARS ELC3CA3 MARS MARS ELC3CA3 MARS MARS ELC3CA3 MARS MARS MARS MARS MARS		
KDM4A KDM3B SOX2-OT STAG1 HPF1 ASH2L	DNAJC18 PCDHAC2 CCDC24 HYDIN IGCE CCDC30 SLC9C2 MFAP3 CLCN3 SNX8 L17RB TCF4 CHDH PCDHGA10 TMTC1 PTCHD1-AS CLIP1 TIH3 TIH4	CR1L CTSS DOCK10 LRP1 PFKFB2 CPEB1 JAK1 AIG1 PDE4B PDE4D HIVEP2 CACNA1C-AS4 CACNA1C-AS4 CACNA1C-TS PRUNE1 ADAMTS9-AS2 EPN2 GPM6A TMEFF2	C70rt60 HARS PRADC1 EIF4E2 KAT5 PDE60 PARK7 TWF2 DPH3 SLC25A33 MARS ADSS CACYBP PSMG3 SNX7 DUS2 ENO1 ELOVL1 GATAD2A GLT8D1		

EPC2	FCHSD1	PCDHGA11	TBC1D5
SLC39A8	RELL2	DGKD	H6PD
CHST12	KCNJ13	SLC2A5	PSKH1
GFRA3	SLC17A7	ITIH3	ALKBH5
LYPD6	SLC4A5	PRKCB	MED8
NLGN4X	ZBTB37	CDHR3	DUSP11
SATB1	BNIPL	LINC00634	SLC35G2
PLEKHO1	IQCF1	PCDHAC2	ARL6IP4
RERE	PPARGC1A	BRINP2	GAS5
RPRD2	TNN	CACNA1D	LEPR
	IMMP2L	KLHL29	SMG1P2
	PCCB	PCDHA4	GIGYF2
	GBF1	SH3PXD2A	INPP5D
	ST3GAL3	GRIN2A	FAM53C
	GRIA1	MRAP2	SOX2
	MAN2A1	INO80B	FPGT-TNNI3K
	CREB3L2	SLC2A7	PTPRF
	PALLD	TLR9	AKT3
	PCDHGA8	PPARGC1A	RRP7BP
	PCDHGA9	CCDC62	SERHL2
	CYP26B1	MPL	CBR4
	BOLL	ADAMTSL3	COX20
	IL19	PCDHA13	SAP30L-AS1
	DDX60L	HECW2	SATB2
	SDCCAG8	PEX5L	FOXP1
	TNNC1	ASTN1	PLXNA2
	PCDHGA11	PCDHA12	DPYD
	PCDHGB4	ANKRD44	PLCB2
	PCDHGB7	NYAP2	
	HSPA9	SLC4A10	
	ALAS1	GPR52	
	WDR55	SNAP91	
	RFTN1	LYPD6B	
	SH3RF1	DNAJC6	
	PITPNM2	SH3GL3	
	COQ10B	R3HDM2	
	CYSTM1	IL34	
		SRPK2	

 Table 3.19 | Neuronal signaling and chromatin regulatory genes clustered
 in RNA heatmap. Lis of genes in co-expression clusters from Figure 3.10, D

Differ Tent Produc Durotes of Academic State Method State	NPC CLUSTER 1					
BL6B.20000 More Metropolis and Angeles 6.661.0 6.001.0 000000 BL6B.200000 Monotine of State Chronits Constants 2.287.0 0.001.0 0.001.0 BL6B.200000 Monotine of State Chronits Constants 2.287.0 0.001.0 <						Total #
E485.0000 Moto. Anglanza 0.00000 Note. Anglanza 0.00000 Note. Source 0.000000 Note. Source 0.0000000 Note. Source 0.000000000000 Note. Source <t< td=""><td></td><td></td><td></td><td></td><td></td><td></td></t<>						
R185.20207 Bestdam of Saue Cronical Conserva 2.265 2 6.51 7.8 7.2 <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>						
R482.02713 Seguration of State Chronization 12.862.02 4.19 8.05 10.00						
B-BS.6827 Micro Promotyping 28.862 4.00 Res B-SS.14224 Micro Promotyping 28.662 6.00 Res BSS.14224 Micro Promotyping 28.662 6.00 Res BSS.14224 Micro Promotyping 28.662 6.00 Res BSS.14224 Micro Promotyping 10.562 6.461 8.00 BSS.14224 Micro Promotyping 10.562 6.461 6.00 10.552 BSS.14224 Micro Promotyping 10.562 6.461 6.00 10.552 DSS.050077 Micro Promotyping 10.562 6.451 6.00 10.552 DSS.050077 Micro Promotyping 10.562 6.451 6.00 10.552 DSS.050077 Micro Promotyping 10.562 6.451 10.552						
B-BS-11412 Aprification of again from the strenctores 2.255.03 6.25 6.25 6.05 6.05 B-BS-114144 Aprification of again from the strenctores was 1AO2 whickury span 2.255.03 6.25 6.05						
60.000070 bitmousene. combinate: again 2.89.00 4.81.80 4.81.80 4.81.80 4.81.80 4.81.80 4.81.80 4.81.80 7.00				6.25		
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GC:0034704 calcium channel complex 1.7EE-03 5.88 4.00 6 GC:0034704 calcium channel complex 1.7EE-03 5.88 4.00 6 GO:0034704 calcium channel complex 1.7EE-03 5.88 4.00 6 GO:0034704 calcium channel activity 1.7EE-03 5.88 4.00 6 GO:0039094 ligand-gated cation channel activity 1.7EE-03 5.88 4.00 6 GO:007100 cellulur channel activity 1.7EE-03 5.88 4.00 7 GO:005262 calcium channel activity 1.82E-03 4.13 5.00 12 GO:0051393 alpha-actinin binding 2.55E-03 8.33 3.00 3 GO:0086004 regulation of cardiac muscle cell contraction 2.97E-03 7.68 3.00 3 GO:0015297 antiporter activity 2.98E-03 4.66 4.00 8 GO:0015297 antiporter activity 3.07E-03 7.32 3.00 3 GO:0015297 response to monamine	GO:0032594 GO:1903044 KEGG:05031 GO:097440 GO:1903115 GO:0005891 GO:0005891 GO:0005891 GO:0005891 GO:0005234 KEGG:04713 GO:00051453 GO:00051453 GO:00051453 GO:00051453 GO:00070252 GO:0070252 GO:0070252	protein transport within lipid bilayer protein localization to membrane raft Amphetamine addiction apical dendrite regulation of actin filament-based movement regulation of actin filament-based movement voltage-gated calcium channel complex voltage-gated calcium channel complex voltage-gated calcium channel complex response to epinephrine extracellularly glutamate-gated ion channel activity Circadian entrainment regulation of intracellular pH regulation of pH actin-mediated cell contraction actin-mediated cell contraction Glutamatergic synapse	1.34E-04 1.55E-04 4.09E-04 7.56E-04 7.76E-04 7.76E-04 7.79E-04 7.79E-04 7.79E-04 9.14E-04 9.14E-04 9.70E-04 1.00E-05 1.04E-05 1.70E-05 1.70E-05 1.70E-05	16.67 33.33 7.35 21.43 8.89 9.09 15.79 15.00 5.21 5.26 5.26 4.35 4.35	4.00 3.00 5.00 4.00 4.00 4.00 3.00 3.00 5.00	24.00 9.00 68.00 14.00 45.00 45.00 44.00 44.00 99.00 96.00 99.00 99.00 99.00 99.00 106.00 115.00 115.00
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GC:0014888 striated muscle adaptation 3.62E-03 6.67 3.00 4 GC:0006195 purine nucleotide catabolic process 4.09E-03 6.25 3.00 4	GC:0032594 GO:1032594 GO:1032594 GO:1032597 GO:103115 GO:1003115 GO:1003115 GO:0005891 GO:0005891 GO:0005891 GO:0005891 GO:00071871 GO:00071871 GO:00071873 GO:00071853 GO:00071853 GO:00070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0032743 GO:0086004 GO:0086004 GO:0015297 GO:0071869 GO:0035249 GO:0071869 GO:0035249 GO:0071869 GO:007255	protein transport within lipid bilayer protein localization to membrane raft Amphetamine addiction apical dendrite regulation of actin filament-based movement regulation of actin filament-based movement voltage-gated calcium channel complex voltage-gated calcium channel complex extracellulary glutamate-gated ion channel activity Circadian entrainment regulation of cellular pH regulation of ofH actin-mediated cell contraction actin-mediated cell contraction Glutamatergic synapse cellular monovalent inorganic cation homeostasis cardiac muscle cell contraction calcium channel complex cardiar enter complex cardiar enter complex cardiar mether complex cardiar mether complex cardiar mether complex cardiar mether complex cardiar mether complex cardiar mether complex cardiar console cell contraction calcium channel complex cardiar complex cardiar complex complex cardiar console cell contraction calcium channel complex cardiar complex cardiar complex cardiar muscle cell contraction calcium channel complex cardiar complex ca	1.34E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.75E-04 1.75E-04 1.79E-04 1.79E-04 1.00E-05 1.00E-05 1.00E-05 1.00E-05 1.70E-05 1.70E-05 1.70E-05 1.70E-05 1.76E-	16.67 33.33 7.35 21.43 8.89 9.09 9.09 15.70 5.21 5.20 5.21 5.20 5.21 5.26 4.75 4.35 4.35 4.35 5.88	4 00 3.00 5.00 4.00 4.00 4.00 5.00	24,00C/ 9,0C/ 45,0C/ 45,0C/ 45,0C/ 45,0C/ 45,0C/ 45,0C/ 45,0C/ 45,0C/ 96
	GC:0032594 GO:1903044 KEGG:05031 GO:0997440 GO:1903115 GO:1903115 GO:1903115 GO:0005891 GO:0005891 GO:0005891 GO:0005891 GO:00051453 GO:00051453 GO:00051453 GO:00051453 GO:00051453 GO:00070252 KEGG:04724 GO:00068603 GO:000526 GO:00004 GO:0086003 GO:0034704 GO:0086003 GO:0034704 GO:0086003 GO:0034704 GO:0005262 GO:0005262 GO:00052743 GO:00051393 GO:0005287 GO:0007215 KEGG:05033 GO:0007215 KEGG:05033 GO:0001659 GO:0001659 GO:00042805	protein transport within lipid bilayer protein localization to membrane raft Amphetamine addiction apical dendrite regulation of actin filament-based movement regulation of actin filament-based movement voltage-gated calcium channel complex voltage-gated calcium channel complex voltage-gated calcium channel complex voltage-gated calcium channel complex vertacellulary gutamate-gated ion channel activity Circadian entrainment regulation of intracellular pH regulation of pH actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex calcium channel complex cardiac muscle cell contraction calcium channel complex calcium channel activity cellular response to reterioic acid calcium channel activity response to monoamine response to cardiac muscle cell contraction response to cardiac muscle cell contraction calcium channel activity response to cardiac muscle cell contraction calcium channel complex calcium channel activity response to cardiac muscle cell contraction calcium channel activity calcium channel complex calcium channel complex calcium channel complex calcium channel complex calcium cha	1.34E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.75E-04 1.76E-04 1.79E-04 1.00E-05 1.33E-05 1.70E-	16.67 33.33 7.35 21.43 8.89 8.89 9.09 15.70 5.21 5.20 5.21 5.20 5.21 5.20 5.21 5.26 4.35 4.35 4.39 4.39 5.88	4 00 3.00 5.00 4.00 4.00 4.00 5.00 5.00 5.00 5.00 5.00 5.00 5.00 5.00 5.00 5.00 4.00 4.00 4.00 4.00 4.00 4.00 5.00 3.00	24,000 9,000 44,000 45,000 45,000 9,000 9,000 9,000 105,0000 105,0000 105,0000 105,0000 105,0000 105,0000 105,0000 105,0000000000
	GC:0032594 GO:0032594 GO:1903044 KEGC:05031 GO:0907440 GO:1903115 GO:1903115 GO:0005891 GO:0005891 GO:0005891 GO:00071871 GO:00071871 GO:00071871 GO:00071873 GO:0051453 GO:0051453 GO:0051453 GO:0051453 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0070252 GO:0030044 GO:0086003 GO:0032743 GO:0032743 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0051393 GO:0071867 GO:0071867 GO:007159 GO:0007215 KEGG:05033 GO:0042805 GO:0042805 GO:0042805 RD:0042805 GO:0042805	protein transport within lipid bilayer protein localization to membrane raft Amphetamine addiction apical dendrite regulation of actin filament-based movement regulation of actin filament-based movement voltage-gated calcium channel complex voltage-gated calcium channel complex extracellulary glutamate-gated ion channel activity Circadian entrainment regulation of intracellular pH regulation of cellular pH regulation of cellular pH regulation of cellular pH regulation of oft actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction calcium channel complex eardiar muscle cell contraction calcium channel complex iguand-gated calcion channel activity cellular response to relinoic acid calcium channel complex iguand-gated calcion channel activity cellular response to relinoic acid calcium channel complex iguand-gated calcion channel activity cellular response to relinoic acid calcium channel activity cellular newscle cell contraction calcium channel activity cellular response to celli contraction calcium channel activity cellular response to celli contraction antiporter activity regulation of interfeukin-2 production antiporter activity response to cardiac muscle cell contraction response to c	1.34E-04 1.56E-04 1.56E-04 1.56E-04 7.56E-04 7.76E-04 7.79E-04 8.56E-04 9.70E-04 8.56E-04 9.70E-04 9.70E-04 9.70E-04 9.70E-04 1.00E-05 1.70E-	16.67 33.33 7.35 21.43 8.89 9.09 9.09 15.79 15.70 5.26 4.75 4.39 4.39 4.32 4.33 5.88 5.89 7.69 7.50	$\begin{array}{c} 4.00\\ 3.00\\ 5.00\\ 4.00\\ 4.00\\ 4.00\\ 5.00\\$	24 0000 9 00000 45 00000 9 000000 9 000000 9 000000 9 000000 9 000000 9 000000 9 00000000
GC:0005231 excitatory extracellular ligand-gated ion channel activity 4.47E-03 6.00 3.00 5 GC:0032663 regulation of interleukin-2 production 5.68E-03 5.45 3.00 5	GC:0032594 GO:1903044 KEGG:05031 GO:0907440 GO:1903115 GO:1903115 GO:0005891 GO:0005891 GO:0005891 GO:0005891 GO:0005234 KEGG:04713 GO:00051453 GO:00051453 GO:00051453 GO:00051453 GO:00051453 GO:00070252 KEGG:04724 GO:00068803 GO:00068003 GO:0086004 GO:0086003 GO:0034704 GO:00851933 GO:0071869 GO:0071869 GO:0071869 GO:0071869 GO:0071869 GO:0007215 KEGG:0053249 GO:0007215 KEGG:0053249 GO:0007215 KEGG:00533 GO:0014888 GO:00014888 GO:006195	protein transport within lipid bilayer protein localization to membrane raft Amphetamine addiction apical dendrite regulation of actin filament-based movement regulation of actin filament-based movement voltage-gated calcium channel complex voltage-gated calcium channel complex voltage-gated calcium channel complex response to epinephrine extracellularly glutamate-gated ion channel activity Circadian entrainment regulation of intracellular pH regulation of pH actin-mediated cell contraction actin-mediated cell contraction actin-mediated cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex cardiac muscle cell contraction calcium channel complex calcium channel activity copsitive regulation of cardiac muscle cell contraction calcium channel activity copsitive regulation of cardiac muscle cell contraction calcium channel activity esponse to retincic acid calcium channel activity response to retincic acid calcium channel activity response to monoamine response to monoamin	1.34E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.55E-04 1.76E-04 1.76E-04 1.70E-03 1.70E-	16.67 33.33 7.35 21.43 8.89 8.89 9.09 15.79 15.00 5.21 5.26 4.35 4.35 4.35 4.35 4.38 5.89 6.82 6.82 6.82	$\begin{array}{c} 4.00\\ 3.00\\ 5.00\\ 4.00\\ 4.00\\ 4.00\\ 3.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 5.00\\ 4.00\\ 4.00\\ 4.00\\ 4.00\\ 4.00\\ 4.00\\ 3.00\\$	$\begin{array}{c} 24000\\ 9000\\ 9000\\ 14000\\ 14000\\ 14000\\ 14000\\ 14000\\ 14000\\ 15000\\ 11000\\ 1$

GO:0072523 R-HSA:2151201 GO:0015296 GO:0086065 GO:0071398	purine-containing compound catabolic process	5.81E-03	5.36	3.00	56.00
R-HSA:2151201 GO:0015296 GO:0086065 GO:0071398					
GO:0015296 GO:0086065 GO:0071398	Transcriptional activation of mitochondrial biogenesis	5.81E-03	5.36	3.00	56.00
GO:0086065 GO:0071398	anion:cation symporter activity	5.94E-03	5.26	3.00	57.00
GO:0071398	cell communication involved in cardiac conduction	6.08E-03		3.00	58.00
	cellular response to fatty acid	6.08E-03		3.00	58.00
GO:0086065	cell communication involved in cardiac conduction	6.08E-03	5.17	3.00	58.00
GO:0098739	import across plasma membrane	7.46E-03	4.76	3.00	63.00
GO:0099516	ion antiporter activity	7.46E-03	4.76	3.00	63.00
GO:0032623	interleukin-2 production	7.46E-03	4.76	3.00	63.00
GO:0051966	regulation of synaptic transmission, glutamatergic	7.93E-03	4.62	3.00	65.00
KEGG:04720	Long-term potentiation	8.41E-03	4.48	3.00	67.00
GO:0003341	cilium movement	8.55E-03	4.41	3.00	68.00
GO:0006901	vesicle coating	8.55E-03	4.41	3.00	68.00
GO:0032729	positive regulation of interferon-gamma production	8.55E-03	4.41	3.00	68.00
GO:0009166	nucleotide catabolic process	8.55E-03	4.41	3.00	68.00
GO:1901863	positive regulation of muscle tissue development	8.70E-03	4.35	3.00	69.00
GO:0048199	vesicle targeting, to, from or within Golgi	9.55E-03	4.17	3.00	72.00
GO:0050795	regulation of behavior	9.70E-03	4.11	3.00	73.00
GO:1901292	nucleoside phosphate catabolic process	9.85E-03	4.05	3.00	74.00
NEURON CLUSTE					
		Term PValue Corrected	% Associated		Total #
GOID	GOTerm	with Benjamini-Hochberg		Nr. Genes	
GO:0007156	homophilic cell adhesion via plasma membrane adhesion molecules	5.18E-23	3 13.84	22.00	159.00
GO:0098742	cell-cell adhesion via plasma-membrane adhesion molecules	4.52E-19	8.94	22.00	246.00
GO:0051001	negative regulation of nitric-oxide synthase activity	4.06E-04	25.00	3.00	12.00
GO:0005891	voltage-gated calcium channel complex	7.33E-04	9.09	4.00	44.00
GO:0032769	negative regulation of monooxygenase activity	7.63E-04	18.75	3.00	16.00
GO:0032769 GO:0071871		8.69E-04	15.79	3.00	19.00
	response to epinephrine				
GO:0051966	regulation of synaptic transmission, glutamatergic	2.08E-03	6.15	4.00	65.00
KEGG:05031	Amphetamine addiction	2.20E-03	5.88	4.00	68.00
GO:0086003	cardiac muscle cell contraction	2.20E-03		4.00	68.00
GO:0034704	calcium channel complex	2.20E-03	5.88	4.00	68.00
KEGG:04724	Glutamatergic synapse	2.28E-03	4.39	5.00	114.00
GO:0051354	negative regulation of oxidoreductase activity	2.77E-03	9.09	3.00	33.00
GO:0008038	neuron recognition	3.25E-03	8.33	3.00	36.00
GO:0086004		3.48E-03	7.69	3.00	39.00
	regulation of cardiac muscle cell contraction				
GO:0007215	glutamate receptor signaling pathway	3.64E-03	4.76	4.00	84.00
GO:0035249	synaptic transmission, glutamatergic	3.64E-03	4.76	4.00	84.00
GO:0071867	response to monoamine	3.74E-03	7.32	3.00	41.00
GO:0071869	response to catecholamine	3.74E-03	7.32	3.00	41.00
KEGG:04713	Circadian entrainment	4.20E-03	4.17	4.00	96.00
GO:0004867	serine-type endopeptidase inhibitor activity	4.27E-03		4.00	98.00
GO:0030212	hyaluronan metabolic process	4.28E-03		3.00	44.00
	Carbohydrate digestion and absorption				
KEGG:04973		4.28E-03	6.82	3.00	44.00
GO:0099601	regulation of neurotransmitter receptor activity	4.28E-03	6.67	3.00	45.00
GO:1903115	regulation of actin filament-based movement	4.28E-03	6.67	3.00	45.00
GO:0006195	purine nucleotide catabolic process	4.34E-03	6.25	3.00	48.00
GO:0050999	regulation of nitric-oxide synthase activity	5.47E-03	5.66	3.00	53.00
GO:0098900	regulation of action potential	5.78E-03	5.45	3.00	55.00
	purine-containing compound catabolic process	5.81E-03	5.36	3.00	56.00
SOUD072523					
GO:0072523		C 11E 0			
GO:0086065	cell communication involved in cardiac conduction	6.14E-03	5.17	3.00	58.00
GO:0086065 GO:0030374	ligand-dependent nuclear receptor transcription coactivator activity	6.17E-03	5.17 5.08	3.00 3.00	59.00
GO:0086065 GO:0030374 GO:0030276		6.17E-03 7.43E-03	5.17 5.08 4.69	3.00 3.00 3.00	59.00 64.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768	ligand-dependent nuclear receptor transcription coactivator activity	6.17E-03	5.17 5.08	3.00 3.00 3.00 3.00	59.00
GO:0086065 GO:0030374 GO:0030276	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding	6.17E-03 7.43E-03	5.17 5.08 4.69 4.55	3.00 3.00 3.00	59.00 64.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation	6.17E-0 7.43E-0 7.79E-0	5.17 5.08 4.69 4.55	3.00 3.00 3.00 3.00	59.00 64.00 66.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production	6.17E-0: 7.43E-0: 7.79E-0: 7.82E-0:	5.17 5.08 4.69 4.55 4.48	3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process	6.17E-0; 7.43E-0; 7.79E-0; 7.82E-0; 7.85E-0; 7.85E-0; 7.85E-0;	5.17 5.08 4.69 4.55 4.48 4.41 4.41	3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development	6.17E-0; 7.43E-0; 7.79E-0; 7.82E-0; 7.85E-0; 7.85E-0; 7.85E-0; 7.89E-0;	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 68.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863 GO:0071300	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid	6.17E-0; 7.43E-0; 7.79E-0; 7.82E-0; 7.85E-0; 7.85E-0; 7.89E-0; 7.89E-0; 7.99E-0; 7.94E-0;	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 69.00 70.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development	6.17E-0; 7.43E-0; 7.79E-0; 7.82E-0; 7.85E-0; 7.85E-0; 7.85E-0; 7.89E-0;	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 68.00
GO:0086065 GO:0030374 GO:0030276 BO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863 GO:0071300 GO:1901292	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation opsitive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process	6.17E-0; 7.43E-0; 7.79E-0; 7.82E-0; 7.85E-0; 7.85E-0; 7.89E-0; 7.89E-0; 7.99E-0; 7.94E-0;	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 69.00 70.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863 GO:0071300	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation opsitive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process	6.17E-0: 7.43E-0: 7.79E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.94E-0: 8.95E-0:	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29 4.05	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 68.00 70.00 74.00
GC:0086065 GC:0030374 GC:0030276 GC:0032768 KEGG:04720 GC:0032729 GC:0032729 GC:009166 GC:1901863 GC:0071300 GC:01901292 NEURON CLUSTE	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2	6.17E-0: 7.43E-0: 7.79E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.89E-0: 8.95E-0: Term PValue Corrected	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.45 4.41 4.42 9 4.29 4.29 4.05	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 69.00 70.00 74.00
GC:0086065 GC:0030374 GC:0030376 GC:0032768 KEGG:04720 GC:0009166 GC:1901863 GC:1901863 GC:0071300 GC:1901292 NEURON CLUSTE GOID	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.98E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.98E-0: 7.9	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.35 4.29 4.05 % Associated Genes	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 74.00 Total # Genes
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0091663 GO:091863 GO:091292 NEURON CLUSTER GOID GO:0070603	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex	6.17E-0: 7.43E-0: 7.79E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.89E-0: 7.94E-0: 8.95E-0: Term PValue Corrected with Benjamini-Hochberg 1.11E-0:	5.17 5.08 4.69 4.55 4.44 4.41 4.41 4.41 4.35 4.29 4.05 4.05 6enes 7.78	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 74.00 Total # Genes 77.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:0009166 GO:1901863 GO:1901863 GO:1901863 GO:1901292 NEURON CLUSTE GOID GO:0070603 GO:1904949	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTem SWUSNF superfamily-type complex ATPase complex	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.98E-0: 7.9	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.35 4.29 4.05 4.20 6 4.85 6 4.20 7.79 5.88	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 74.00 74.00 74.00 74.00 74.00 74.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 GO:0032729 GO:0032729 GO:0032729 GO:0032729 GO:001863 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:1901292 NEURON CLUSTE GOID GO:00076603 GO:1904949 GO:009568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process ? 2 COTerm SWUSNF superfamily-type complex ATPase complex nuclear itanscriptional repressor complex	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.88E-0: 7.88E-0: 7.98E-0: 8.95E-0: 8.95E-0: Term PValue Corrected with Benjamini-Hochberg tht Benjamini-Hochberg 2.81E-0: 2.81E-0: 2.81E-0:	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.41 4.42 4.05 4.29 4.05 6 6 6 6 6 7.79 5.88 11.11	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 70.00 74.00 74.00 74.00 70.00 74.00 70.00 74.00 70.00 74.00 70.00 74.00 70.00 74.00 70.00 74.00 70.00 74.00 70.000
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGC:04720 GO:0091663 GO:1001863 GO:0071300 GO:1001863 GO:001292 NEURON CLUSTE GOID GO:0070603 GO:0090568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity cogulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 COTem SWVSNF superfamily-type complex ATP ase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex	6.17E-0: 7.43E-0: 7.43E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.89E-0: 8.95E-0: 8.95E-0: Term PValue Corrected with Benjamini-Hochberg 1.11E-0- 2.81E-0- 2.87E-0- 2.87E-0- 2.87E-0-	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.41 4.42 4.05 4.29 4.05 6.05 8.05 8.05 9.05 9.05 9.05 9.05 9.05 9.05 9.05 9	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00	59.00 64.00 66.00 67.00 68.00 68.00 69.00 70.00 74.000
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032769 GO:009166 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:0001863 GO:0000568 GO:0090568 GO:000568 GO:000568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 COTem SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodeling	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.84E-0: 7.9	5.17 5.08 4.69 4.45 4.48 4.41 4.41 4.41 4.45 4.29 4.05 % Associated Genes 7.79 5.88 11.11 11.11	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 69.00 74.000
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:009166 GO:0091663 GO:1901292 NEURON CLUSTER GOID GO:0070603 GO:1904949 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 COTerm SWUSNF superfamily-type complex ATPase complex ATPase complex nuclear transcriptional repressor complex hromatin remodeling chromatin remodeling	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.88E-0: 7.98E-0: 8.95E-0: 7.94E-0: 8.95E-0: 7.94E-0: 8.95E-0: 8.9	5.17 5.08 4.69 4.69 4.48 4.41 4.41 4.41 4.41 4.41 4.43 4.29 4.05 5.88 6 6 6 6 8 5.88 5.88 11.11 1.11 4.24 4.24	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 4.00 7.00 7.00	59.00 64.00 66.00 67.00 68.00 68.00 68.00 70.00 74.000
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEG0:04720 GO:0009166 GO:0091663 GO:0071300 GO:1901292 NEURON CLUSTE GOID GO:0070603 GO:1904949 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodeling chromatin remodeling NO80-type complex	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.89E-0: 8.95E-0: Term PValue Corrected with Benjamini-Hochberg 1.11E-0: 2.87E-0: 2.87E-0: 3.20E-0: 3.20E-0: 3.20E-0: 1.17E-0:	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29 4.05 6 4.29 4.05 5.88 11.11 11.11 11.11 4.24 4.24 4.24 13.64	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 7.00 7.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 70.00 74.000
GO:0086065 GO:0030374 GO:0030276 GO:003276 GO:0032729 GO:0032729 GO:0032729 GO:009166 GO:1901863 GO:1901863 GO:1901863 GO:1901863 GO:0091292 NEURON CLUSTE GOID GO:0007603 GO:1904949 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monooxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 COTerm SWUSNF superfamily-type complex ATPase complex ATPase complex nuclear transcriptional repressor complex hromatin remodeling chromatin remodeling	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.89E-0: 7.94E-0: 8.95E-0: 8.9	5.17 5.08 4.69 4.69 4.48 4.41 4.41 4.41 4.41 4.41 4.43 4.29 4.05 5.88 6 6 6 6 8 5.88 5.88 11.11 1.11 4.24 4.24	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 4.00 7.00 7.00	59.00 64.00 66.00 67.00 68.00 68.00 68.00 70.00 74.000
GO:0086065 GO:0030374 GO:0030276 GO:0032768 GO:0032769 GO:0093768 GO:0091663 GO:0091663 GO:0071300 GO:1901292 NEURON CLUSTEI GOID GO:0070603 GO:1904949 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodeling chromatin remodeling NO80-type complex	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.89E-0: 8.95E-0: Term PValue Corrected with Benjamini-Hochberg 1.11E-0: 2.87E-0: 2.87E-0: 3.20E-0: 3.20E-0: 3.20E-0: 1.17E-0:	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29 4.05 6 4.29 4.05 5.88 11.11 11.11 11.11 4.24 4.24 4.24 13.64	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 7.00 7.00 3.00	59.00 64.00 66.00 67.00 68.00 68.00 70.00 74.000
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GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:00032769 GO:0009166 GO:0091663 GO:1901863 GO:1901863 GO:1901292 NEURON CLUSTE GOID GO:0070603 GO:1904949 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 GO:0090568 Harris and the second	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodeling hNO80-type complex hINO80-type complex hINO80-type complex Distone deacetylase complex Cell cycle Regulation of TP53 Activity through Acetylation	6.17E-0: 7.43E-0: 7.43E-0: 7.82E-0: 7.85E-0: 7.85E-0: 7.89E-0: 7.94E-0: 7.9	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29 4.05 7.79 5.88 11.11 11.11 11.11 14.24 4.24 4.23 5.56 0.10.00	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 6.00 4.00 7.00 7.00 7.00 3.00	59.00 64.00 66.00 68.00 68.00 70.00 70.00 70.00 70.00 70.00 70.00 70.00 70.00 70.00 102.00 36.00 36.00 165.00 165.00 124.00 30.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:009166 GO:0091663 GO:0091663 GO:1901292 NEURON CLUSTE GOID GO:0090568 GO:0	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex Commatin remodeling NNO80-type complex NO80-type complex Cell cycle Regulation of TP53 Activity through Acetylation Progesterone-mediated cocyte maturation	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.88E-0: 7.88E-0: 7.88E-0: 8.95E-0: 8.95E-0: 8.95E-0: 8.95E-0: 8.95E-0: 2.87E-0: 2.87E-0: 2.87E-0: 3.20E-0: 3.2	5.17 5.08 4.69 4.65 4.48 4.41 4.41 4.41 4.41 4.35 4.29 4.05 5.88 11.11 1.11 4.24 4.24 5.56 4.03 10.00 4.04	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 7.00 7.00 3.00 4.00 7.00 3.00 4.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 70.00 74.00 74.00 74.00 74.00 102.00 36.00 165.00 165.00 165.00 165.00 165.00 122.00 124.00 30.00 99.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGC:04720 GO:0093768 GO:0093768 GO:0093769 GO:0091663 GO:0091663 GO:0091683 GO:0091292 NEURON CLUSTE GOID GO:0090568 GO:	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodelling histone deacety/ase complex No80-type complex Cell cycle Regulation of TP53 Activity through Acetylation Progesterone-mediated ocyte maturation aminoacy/1-RNA ligase activity	6.17E-0: 7.43E-0: 7.43E-0: 7.85E-0: 7.85E-0: 7.85E-0: 8.95E-0: 8.95E-0: 7.89E-0: 7.89E-0: 8.95E-0: 7.89E-0: 8.95E-0: 7.89E-0: 8.95E-0: 8.95E-0: 7.89E-0: 8.95E-0: 8.9	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.35 4.29 4.05 6.05 7.79 5.88 11.11 11.11 4.24 4.24 13.64 5.56 4.03 10.00 4.04 6.38	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 6.00 6.00 6.00 6.00 7.00 7.00 7.00 3.00 3.00 3.00 4.00 3.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 70.00 70.00 70.00 70.00 70.00 70.00 70.00 36.00 36.00 36.00 165.00 165.00 165.00 165.00 124.00 124.00 30.00 99.00 47.00
GO:0086065 GO:0030374 GO:0030276 GO:0032768 KEGG:04720 GO:0032729 GO:009166 GO:0091663 GO:0091663 GO:1901292 NEURON CLUSTE GOID GO:0090568 GO:0	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex ATPase complex nuclear transcriptional repressor complex chromatin remodeling NO80-type complex histone deacetylase complex Cell cycle Regulation of TP53 Activity through Acetylation Progesterone-mediated ocyte maturation aminoacyl-IRNA ligase activity	6.17E-0: 7.43E-0: 7.78E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.85E-0: 7.88E-0: 7.88E-0: 7.88E-0: 8.95E-0: 8.95E-0: 8.95E-0: 8.95E-0: 8.95E-0: 2.87E-0: 2.87E-0: 2.87E-0: 3.20E-0: 3.2	5.17 5.08 4.69 4.65 4.48 4.41 4.41 4.41 4.41 4.35 4.29 4.05 5.88 11.11 1.11 4.24 4.24 5.56 4.03 10.00 4.04	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 4.00 4.00 7.00 7.00 3.00 4.00 7.00 3.00 4.00	59.00 64.00 66.00 67.00 68.00 69.00 70.00 70.00 74.00 74.00 74.00 74.00 102.00 36.00 165.00 165.00 165.00 165.00 165.00 122.00 124.00 30.00 99.00
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GC:0086065 GO:0030374 GO:0030276 GO:0032768 KEGC:04720 GO:0093768 KEGC:04720 GO:0093768 GO:0093768 GO:009368 GO:0091683 GO:0091292 NEURON CLUSTE GOID GO:0070603 GO:0190568 GO:0090568 GO:0090568 GO:0090568 GO:0097346 GO:0000118 KEGG:04914 GO:00001875 GO:0016706 GO:0016706 GO:0016706 GO:0016706 GO:000681 GO:000681 GO:0000422	ligand-dependent nuclear receptor transcription coactivator activity clathrin binding regulation of monoxygenase activity Long-term potentiation positive regulation of interferon-gamma production nucleotide catabolic process positive regulation of muscle tissue development cellular response to retinoic acid nucleoside phosphate catabolic process R 2 GOTerm SWUSNF superfamily-type complex ATPase complex nuclear transcriptional repressor complex nuclear transcriptional repressor complex chromatin remodeling chromatin remodeling histone deacetylase complex NINO80-type complex Altabolic process Regulation of TP53 Activity through Acetylation Progesterone-mediated occyte maturation aminoacyl-TRNA ligase activity histone deacetylation poidoreductase activity, acting on paired donors, with incorporation or reduction of molecular oxygen, 2- oxoglutarate as one donor, and incorporation of one atom each of oxygen into both donors androgen receptor binding	6.17E-0: 7.43E-0: 7.43E-0: 7.85E-0: 7.85E-0: 7.85E-0: 8.95E-0: 8.95E-0: 7.89E-0: 7.89E-0: 8.95E-0: 7.89E-0: 8.95E-0: 8.95E-0: 7.89E-0: 8.95E-0: 8.9	5.17 5.08 4.69 4.55 4.48 4.41 4.41 4.41 4.42 4.05 6.638 4.29 4.05 5.86 11.11 11.11 4.24 4.24 4.35 5.86 4.03 10.00 4.04 6.38 4.35 5.82 6.52 6.52 6.52 6.52 6.52 6.52 6.52 6.5	3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 3.00 6.00 4.00 4.00 3.00 3.00 3.00 4.00 3.00 3.00 3.00 4.00 3.00 3.00 3.00 4.00 3.00 3.00 3.00 4.00 3	59.00 64.00 67.00 67.00 68.00 68.00 70.00 74.00 74.00 74.00 74.00 74.00 74.00 74.00 72.00 165.00 165.00 165.00 72.00 165.00 72.00 165.00 99.00 92.00 92.00 92.00 92.00 92.00 92.00 95.00
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GOID	GOTerm	Term PValue Corrected with Benjamini-Hochberg	% Associated Genes	Total # Nr. Genes Genes	268
GO:0002292	T cell differentiation involved in immune response	1.05E-04	6.45	4.00 62.0	
GO:0002286	T cell activation involved in immune response	2.95E-04	4.17	4.00 96.0	0
GO:0051310	metaphase plate congression	4.94E-04	5.26	3.00 57.0	0
GO:0002287	alpha-beta T cell activation involved in immune response	5.86E-04	5.36	3.00 56.0	0
GO:0002293	alpha-beta T cell differentiation involved in immune response	5.86E-04	5.36	3.00 56.0	0
GO:0043367	CD4-positive, alpha-beta T cell differentiation	6.34E-04	4.55	3.00 66.0	0
KEGG:04520	Adherens junction	7.01E-04	4.17	3.00 72.0	0
GO:0002294	CD4-positive, alpha-beta T cell differentiation involved in immune response	7.40E-04	5.45	3.00 55.0	0

Table 3.20 | GOs of gene clusters with high correlation scores from Table3.19.

Significant GO terms from gene lists for each coexpression cluster from Figure 3.10, D.

ACTR8	ACTR5	ADSL	CA6	ENO1	UTS2
ADSS	ACTR8	ADSS	SLC2A5	CA6	RNF220
AKT3	ADSS	ALMS1	EPB41	SLC2A5	ADSS
ALMS1	AKT3	ANKHD1	PPIH	H6PD	GRIN2A
NKRD45	ALMS1	AOX1	EBNA1BP2	MPL	MAPK3
NP32E	AMBRA1	CCT7	ST3GAL3	CDC20	NFATC3
	ANKRD45	CHRM3		MED8	TCF4
AOX1			B4GALT2		
ARNT	ANP32E	CNKSR2	DPYD	RNF220	JUND
ASB5	AP1S3	CUL3	ANP32E	ANP32E	DPP4
ASH2L	ATG13	DPP4	RPRD2	SEMA6C	HECW2
34GALT2	ATXN7	EP300	ARNT	IL19	SF3B1
BRD8	BAHD1	GPER	SEC16B	PFKFB2	MOB4
CA6	BAZ2A	GRIA1	SDCCAG8	PLXNA2	AOX1
CACNA1C	BOLL	GRIN2A	AKT3	SDCCAG8	CUL3
CACNA1D	CA6	HECW2	ADSS	AKT3	UGT1A8
CACNA2D3	CACNA1C	JUND	NT5C2	ADSS	UGT1A10
CCNH	CACNA1D	KLHL20	TAF5	SUFU	UGT1A9
DC25C	CCT7	MAPK3	PHF21A	NT5C2	UGT1A7
ENPL	CDC20	MASK-BP3	CACNA1C	TAF5	UGT1A6
CHST12	CDC25C	MEF2C	PPP1CC	PHF21A	UGT1A5
JIRH1A	CENPL	MOB4	CLIP1	CREB3L1	UGT1A4
LCN3	CHDH	NFATC3	KNTC1	DGKZ	ADSL
LIP1	CPEB1	NMUR2	GTF2H3	AMBRA1	EP300
LIPT LU			TCTN2	AMBRAT ATG13	
	CREB3L1	PDE4B			SH3RF1
REB3L2	CTNNA1	PLXNA4	GRIN2A	CTNND1	MEF2C
SGALNACT1	CTNND1	RNF220	UTP4	CACNA1C	ANKHD1
UL3	CUL3	SDCCAG8	SNTB2	BAZ2A	NMUR2
CYP26B1	DGKD	SF3B1	NCAN	MARS	GRIA1
DCTN1	DGKZ	SH3RF1	SUGP1	PLCB2	GPER1
NAJC18	DNAJC19	SLC4A5	MAU2	BAHD1	PLXNA4
NAJC19	DNAJC6	TCF4	GATAD2A	SH3GL3	CNKSR2
PYD	DPYD	UGT1A10	CYP26B1	GRIN2A	
BNA1BP2	DYSF	UGT1A5	EPC2	PSKH1	
P300	EGR4	UGT1A8	PSMD14	PSMB10	
PB41	ENO1	UTS2	HECW2	SLC12A4	
PC2	EPC2		SF3B1	WDR81	
PHB1	EPHA7		HSPD1	NT5M	
TF1	EPN2		SGO2	SREBF1	
GFR1	FBXO41		AOX1	GATAD2A	
GAB1	GABBR1		CUL3	DYSF	
GATAD2A	GATAD2A		UGT1A8	HECW2	
GBF1	GRIA1		UGT1A10	SF3B1	
GFRA3	GRIN2A			CUL3	
			UGT1A9		-
GPM6A	GRM3		UGT1A7	PDE6D	
GRIA1	H6PD		UGT1A6	DGKD	
GRIN2A	HARS		EP300	ACTR5	
GTF2H3	HARS2		RANGAP1	POLDIP3	
IDAC3	HDAC2		OXNAD1	SEMA3G	
HECW2	HECW2		PLCL2	ITIH3	
ISPA9	HSPA9		ITIH3	ITIH4	
ISPD1	IL19		ITIH4	THOC7	
NO80B	INO80B		CACNA1D	ATXN7	
NPP4B	INPP4B		EPHB1	PPP2R3A	
NPP5D	INPP5D		PPP2R3A	STAG1	
TGA9	ITIH3		PCCB	DNAJC19	
TIH3	ITIH4		STAG1	PPARGC1A	1
TIH4	JAK1		NCK1	PDE4D	-
LHL20	KAT5		DNAJC19	CDC25C	
(NTC1	KDM4A		PPARGC1A		
	KIAA1009				
AD1L1			SH3RF1	CTNNA1	
AU2	LEPR MAD1L1		CLCN3	HARS	
/EF2C			GPM6A	HARS2	
ALL5	MAP3K11		ASB5	ZMAT2	
ICAN	MARS		PDE4D	SPARC	_
ICK1	MBTPS2		BRD8	GRIA1	
IFATC3	MED8		CDC25C	GABBR1	
NISCH	MEF2C		HSPA9	SNAP91	
ILGN4X	MLL5		DNAJC18	CEP162	
IT5C2	MPL		HDAC3	PSMG3	
DXNAD1	NFATC3		GRIA1	MAD1L1	
PCCB	NGEF		MAD1L1	GRM3	
PDE4B	NT5C2		CHST12	CA8	
PDE4D	NT5M		KMT2E	MBTPS2	1
PHF21A	OXNAD1	1	CREB3L2		1
PLCL2	PCGF6		CLU	1	1
PLXNA2	PDE4B		NLGN4X		-
PARGC1A	PDE4B PDE4D		RPS6KA3		
			N730NA3		
PIH	PDE6D				
PP1CC	PEMT				
PP2R2A	PFKFB2				
PP2R3A	PHF21A				
SMD14	PLCB2				
RANGAP1	PLXNA2				
RASA1	POLDIP3				

RPRD2	PPP2R2A			
RPS6KA3	PPP2R3A			
SDCCAG8	PRKCB			
SEC16B	PRKD1			
SEMA3G	PSKH1			
SF3B1	PSMB10			
SGOL2	PSMG3			
SH3RF1	RNF220			
SLC2A5	SDCCAG8			
SLC4A5	SEMA3G			
SNTB2	SEMA6C			
ST3GAL3	SH3GL3			
STAG1	SLC12A4			
SUGP1	SLC2A5			
TAF5	SMG6			
TCF4	SNAP91			
TCTN2	SPARC			
TNN	SREBF1			
TNR	STAG1			
UGT1A10	STAT6			
UGT1A5	SUFU			
UGT1A8	TAF5	1	1	1
	THOC7			

Table 3.21 | Protein networks (string-db) in SZ risk connections.

Proteins recognized by string-db as part of an association network from the list of genes in Table 3.15 for NPCs, neurons, and glia. Highlighted groups of rows (in PGC2 set) for indicated genes that are located in the same or adjacent TADs (i.e., chromatin colocalization). Different colors are merely for ease of visualization.

	Schizophrenia (n=23)	Healthy/Control (n=20)
Age		
Mean	82.2	83.3
SD	8.6	10
Sex, n (%)		
Male	8 (35)	7 (35)
Female	15 (65)	13(65)
PMI		
Mean	11.4	8
SD	3.6	4.2

Table 3.22 | Demographics of brain cohort used for proteomic analysis.Demographic information of postmortem adult brains that were assayed for
neuronal protein quantification with LC-SRM MS. PMI, post-mortem interval

Materials and methods

In situ Hi-C from hiPSC-derived cells

In situ Hi-C libraries were generated from 2 million to 5 million cultured hiPSC derived NPCs, glia, and neurons as described in (Rao et al., 2014) without modifications in the protocol. Briefly, in situ Hi-C consists of 7 steps: (i) crosslinking cells with formaldehyde, (ii) digesting the DNA using a 4-cutter restriction enzyme (e.g., Mbol) within intact permeabilized nuclei, (iii) filling in and biotinylating the resulting 5'-overhangs, (iv) ligating the blunt ends, (v) shearing the DNA, (vi) pulling down the biotinylated ligation junctions with streptavidin beads, and (vii) analyzing these fragments using paired end sequencing. As quality control (QC) steps, we checked for efficient restriction with an agarose DNA gel and for appropriate size selection using the Agilent Bioanalyzer after steps (v) and (vi). For the final QC, we performed superficial sequencing on the Illumina MiSeq (~2-3Mreads/sample) to assess guality of the libraries using metrics such as percent of reads passing filter, percent of chimeric reads, and percent of forward-reverse pairs (Table 3.1). For the forebrain directed differentiation neuronal library from subject S1, the Arima Hi-C kit (Arima Genomics, San Diego) was used according to the manufacturer's instructions. Hi-C read mapping and matrix generation

The Hi-C libraries were sequenced on the Illumina HiSeq1000 platform (125bp paired-end) (New York Genome Center). Technical replicates of subject

S2 NPCs, neurons, and glia were also sequenced to enhance resolution. Initial 273 processing of the raw 2 ×125 bp read pair FASTQ files was performed using the HiC-Pro analysis pipeline (Servant et al., 2015). In brief, HiC-Pro performs four major tasks: aligning short reads, filtering for valid pairs, binning, and normalizing contact matrices. HiCPro implements the truncation-based alignment strategy using Bowtie v2.2.3 (Langmead et al., 2009), mapping full reads end-to-end or the 5' portion of reads preceding a GATCGATC ligation site that results from restriction enzyme digestion with Mbol followed by end ligation. Invalid interactions such as same-strand, dangling-end, self-cycle, and single-end pairs are not retained. Binning was performed in 10kb, 40 kb and 100 kb nonoverlapping, adjacent windows across the genome and resulting contact matrices were normalized using iterative correction and eigenvector decomposition (ICE) as previously described (Imakaev et al., 2012), using HiC-Pro's default settings of 100 maximum iterations, filtering of the sparse bins (lowest 2%), and a relative result increment of 0.1 before declaring convergence (http://nservant.github.io/HiC-Pro/MANUAL.html). Data are reported in browserextensible-data-like (BED) format and visualized in the Washington University Epigenome Browser (http://epigenomegateway.wustl.edu). Hierarchical clustering was performed on the ICE-corrected intrachromosomal contact matrices after the bins with the 1% most extreme interaction values were excluded as largely artifactual. Clustering was performed using Ward's method on the 1, 5, 10, 25, 50, and 100% most variable remaining bins using (1correlation) as a distance metric. The results using the 10% most variable interaction bins, shown here in a cluster dendrogram and a Pearson correlation matrix, are representative of these results.

Hi-C loop calls using Juicer

Loop calling was performed using the software HiCCUPS (Rao *et al.*, 2014). To format data for HiCCUPS input, we remapped reads from Hi-C libraries using the Juicer pipeline (Durand, Shamim, *et al.*, 2016). Similar to HiC-Pro, the Juicer pipeline performs read alignment, filtering, binning, and matrix normalization. Samples were pooled for each cell type (S1 and 2 technical replicates from S2) to generate the maximum amount of coverage required for accurate loop calling. The resulting .hic matrix files (MAPQ > 0) were then used as input to HiCCUPS. The following parameters were set for HiCCUPS following the analysis in (Rao *et al.*, 2014): FDR threshold (f) = 0.10, 0.10; peak width (p) = 4, 2; window width (i) = 7, 5; merge distance (d) = 20 kb, 20 kb. Values for parameters correspond to calls made at 5kb and 10kb, respectively.

Representative neuronal and non-neuronal loops are presented in Figure 3.4. As the number of loops called is dependent upon the number of Hi-C contacts in the matrix (Forcato *et al.*, 2017), we also generated matrices with equivalent total Hi-C contacts via subsampling. hiPSC-derived Hi-C interaction matrices were randomly subsampled to 372,787,143 cis only contacts (the lowest number of cis contacts across all cell types) and HiCCUPS was rerun on the subsampled matrices. After loops were called for each cell type, we performed a reevaluation on this union set of loop loci. HiCCUPS was rerun using the union set of loop loci 275 as input to produce *q*-values for each loop in the union set for every cell type. By default, HiCCUPS does not output a *q*-value for every pixel. Hence, this reevaluation produced q-values for pixels in cells that did not pass the significance threshold. We then defined any pixel from the union set with a *q*-value < 0.10 with respect to the donut neighborhood surrounding the pixel to be a loop and defined the loop to be shared with any cell types having a *q*-value < 0.10 for the same pixel.

These loop calls were used for comparing loop calls between cell types. Loops were also called and subsampled as above for the GM12878 cell line using the processed data from (Rao *et al.*, 2014) found here:

www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE63525. Loop calls were overlapped with compartment calls (Materials and methods), such that AA, BB, and AB refer to loops with both anchors in A, both anchors in B, and one anchor in A and other anchor in B, respectively. Loops in chromosomes 4, 18, 19, and X were removed from this compartment analysis since the first principle component most likely corresponded to p versus q arm distinctions and not A versus B compartments.

Hi-C interactions at risk loci

To approach 3DG conformation in context of the disease-relevant sequences, we adapted the binomial statistics based mapping strategy previously described by Won et al (Won *et al.*, 2016). The set of schizophrenia

risk loci used in this study included the original (PGC2, Psychiatric Genomics Consortium) (Ripke *et al.*, 2014) risk sequences, or 108 physically distinct association loci defined by 128 index SNPs (corrected P 10⁻⁸) and an additional 37 loci from the CLOZUK (a series of UK cases registered for clozapine treatment with a clinical diagnosis of schizophrenia) study for a total of 145 loci defined by 179 independent genome-wide significant SNPs (corrected P < 5 × 10⁻⁸), determined by GWAS in 40,675 cases and 64,643 controls (Pardiñas *et al.*, 2018). A risk locus is defined as a collection of (SNPs) existing in linkage disequilibrium, ranging from 1bp to 8.9Mb (average 256.2 kb) in length and in total equivalent to approximately 0.012% of human genomic sequence.

To identify significantly enriched interactions involving a bin of interest with another bin, our principal approach was to first estimate the expected interaction counts for each interaction distance by calculating the mean of all intrachromosomal bin-bin interactions of the same separation distance throughout the raw intrachromosomal contact matrix. We used the R package, HiTC (Servant *et al.*, 2012), to facilitate manipulation of our HiC-Pro-produced raw contact matrices and estimation of the expected counts at various interaction distances. The probability of observing an interaction between a bin-of-interest and another bin was then defined as the expected interaction between those two bins divided by the sum of all expected interactions between the bin-of-interest and all other intrachromosomal bins. A *P* value was then calculated as binomial probability of observing the number of interaction counts or more between the bin-of interest and some other bin where the number of successes was defined 277 as the observed interaction count, the number of tries as the total number of observed interactions between the bin-of-interest and all other intrachromosomal bins, and the success probability as the probability of observing the bin-bin interaction estimated from the expected mean interaction counts. The Benjamini-Hochberg method was used to control false discovery rate (FDR) for *P* values determined for all interactions with a bin-of interest (includes all bins 1Mb up and downstream in our tests).

Generation of stable selected dCas9-VP64/VPR and Cas9 NPCs

All CRISPR-based epigenomic editing assays were performed on antibiotic selected dCas9-VP64 (VP64 as the tetrameric VP16 activator domain) and dCas9-VPR (VPR as the tripartite activator, VP64-p65-Rta) NPCs derived as described in (Ho *et al.*, 2017). For generation of Cas9 stable, selected NPCs, we used a plasmid of lentiCRISPR v2 gifted by Feng Zhang (Addgene plasmid # 52961). DNA sequencing with a U6 primer confirmed the identity. Lentiviral production and titration were performed as described previously (Ho *et al.*, 2016). Control S1 and S2 NPCs were spinfected with lentiCRISPR v2 virus as described (Ho *et al.*, 2017). 48 hours post-transduction, cells were selected by exposure to puromycin at 0.3 μ g/mL. Without transduction, all control cells died within around 5 days after the antibiotic addition. The puromycin-selected NPCs were subject to Western blot analysis of Cas9 expression. 30 μ g of proteins were electrophoresed in NuPAGE 4-12% Bis-Tris Protein Gels (NP0323PK2, Life Technologies) in 1× MES running buffer, 200 V constant, 35 min. Proteins were transferred onto nitrocellulose membrane (IB23002, Life Technologies) on the iBlot® 2 Dry Blotting System (program P3, 7:00 min). The membranes were incubated with primary antibodies against Cas9 (1:250, monoclonal, clone 7A9, Millipore) and b-Actin (1:10,000, mouse, 1406030, Ambion) overnight at 4°C. Then, membranes were incubated with the IRDye-labeled secondary antibodies for 45 min at RT in the dark on the rocker. Fluorescence was visualized using a Li-CorOdyssey Imaging System.

In vitro transcription and transfection of gRNAs

Guide RNAs (gRNAs) were designed on Benchling (www.benchling.com) using the CRISPR tool. gRNAs were generated via in vitro transcription (IVT) with the GeneArt Precision gRNA Synthesis Kit (Thermo Fisher Scientific, A29377) as per manufacturer instructions. Five gRNAs were designed per condition (i.e., "loop SNP", negative control, and positive control) and pooled for transfection. The genomic ranges within which loop-SNP gRNAs were designed (i.e., region spanning the SNP of interest and all gRNAs in the condition) were roughly 600 bp for *ASCL1*, 550 bp for *MATR3*, 460 bp for *EFNB1* (with 2/5 gRNAs directly overlapping the SNP), 300 bp for *SOX2*. Puromycin-selected (1µg/mL in NPC media; Sigma, #P7255) dCas9-VP64 and dCas9- VPR NPCs (Ho *et al.*, 2017) were seeded at a density of ~400,000 per well on Matrigelcoated (BD Biosciences) 24-well plates. Pooled IVT gRNAs (500 ng total RNA/well) and 2 µL EditPro Stem lipofectamine (MTI-GlobalStem, #GST-2174;

now, ThermoFisher, STEM00003) were diluted in 50 µL Opti-MEM (Thermo Fisher Scientific, #31985062) and added dropwise to each well. Cells were harvested with TRIzol for total RNA extraction 48 hours later. All experiments were conducted with 3 to 6 biological replicates from 1 donor (subject S1), generated in parallel, with the donor contributing isogenic dCas9-VP64 and dCas9-VPR effector cells. Each data point in Figure 3.8, D to F, represents one biological replicate within each condition. For each target gene promoter and candidate loop, control gRNAs were strategically placed into the middle third of the (linear) genome portion bypassed by the candidate loop. CRISPRa results were analyzed on PRISM with a one-way ANOVA across 3 conditions with a Dunnett's test for multiple comparisons. Cas9 mutagenesis was also performed as described above with the exception of the negative control, which in these experiments consisted of an empty transfection (i.e., lipofectamine + Opti-MEM without any gRNA). Cas9 results were analyzed with an unpaired *t* test comparing the loop-SNP and negative control conditions.

RNA transcriptomic correlation heatmaps

Pearson correlation coefficient matrices were calculated for gene expression in the childhood onset schizophrenia data set (Hoffman *et al.*, 2017) using R from lists of genes that are located in cell-type-specific loops anchored at schizophrenia risk loci and, as a subset of this list, sets of genes whose proteins participate in an association network for each of the three cell types (see below). Significance was computed calculating the absolute mean correlation coefficient

279

of each correlation matrix ("organization score") as a test statistic against a null 280 distribution generated by random gene sampling. Randomized gene lists were drawn only from the pool of genes with over 1 count per million (CPM) in at least 30% of the experiments described in (Hoffman et al., 2017). To generate a null distribution of organization scores for a given cell type that accounted for genomic distance and neighborhood effects, we began by randomly selecting a significant PGC interaction for that cell type. Using the bp genomic distance of this interaction we randomly selected two 10kb bins from the genome separated by the same distance. All genes overlapping these bins were then added to the list of genes with which to calculate the organization score. This process was iterated until enough genes were added to the list to match the number of genes used in the original cell-type-specific organization score. Finally, this protocol was repeated 1000 times to generate the null distribution of random organization scores. This distribution was then used to calculate significance of co-regulation (i.e., P = number of times $|r|_{avg}$ of the null exceeded that of the test heatmap / 1000). Note that STRING gene network transcriptomic analyses (Figure 3.13, B) were performed with 1000 random permutations of genes sampled from the full schizophrenia risk connectome (i.e., risk locus + risk locus connect genes) for each cell type.

Generation of hiPSC-derived cell types

NPCs were derived from human iPSCs, as described (Brennand *et al.*, 2011). Briefly, hiPSCs reprogrammed from two independent controls (Subjects

S1 and S2) fibroblasts were cultured as embryoid bodies and maintained in suspension in N2 medium (Invitrogen) for 7 days. They were then plated onto polyornithine/laminin-coated plates and visible rosettes were manually dissected after 1 week and cultured in NPC medium (Brennand *et al.*, 2011). NPCs were maintained at high density on Matrigel (BD Biosciences) and split approximately 1:4 once a week with Accutase (Millipore). In our hands, doubling time of hiPSC-derived forebrain NPCs is 3.69 ± 0.05 days, with the percentage of cells in G1 cell cycle phase is $62.3 \pm 0.2\%$, in S phase is $24.6\pm0.1\%$ and G2 phase is $6.5 \pm 0.03\%$ (Brennand *et al.*, 2015).

NPCs were further differentiated along the glial lineage as previously described (TCW *et al.*, 2017). Briefly, NPCs described above were seeded at a density of 15,000 cells/cm² on Matrigel-coated plates in astrocyte medium (ScienCell). Cells were split at 95% confluency, approximately every 6-7 days, to the initial seeding density with Accutase (Millipore). After 30 days of differentiation, glia were used in assays or were maintained in astrocyte medium (with 2% FBS), splitting 1:3 every week with Accutase.

To arrive at hiPSC-neurons via Neurogenin2 induction as previously described (Ho *et al.*, 2016), NPCs were seeded at low density on Matrigel-coated plates and transduced with lentiviral constructs to overexpress *NGN2*, driven by a TetO promoter, in NPC-medium. Doxycycline (Sigma, #D9891) was added in NPC medium to activate the system and puromycin (Sigma, #P7255) was introduced for selection. 2 days after doxycycline addition, puromycin is withdrawn and NPC medium was switched to neuron medium (Ho *et al.*, 2016). 282 Cells were maintained with regular half-medium changes until day 20 after first doxycycline addition. At this point, neurons were ready to be used for assays. In our hands, as described, combining puromycin induction and Ara-C treatment to block proliferation of dividing cells results in pure or near-pure (approaching 99-100% homogeneity by cell type) populations of postmitotic excitatory (glutamatergic) neurons, with other cell types undetectable in the culture (Ho *et al.*, 2016).

For forebrain neuronal differentiation, as previously described (Brennand *et al.*, 2011), for S1, NPCs were seeded at low density and cultured in neural differentiation medium (DMEM/F12, 1xN2, 1xB27-RA, 20 ng ml–1 BDNF (Peprotech), 20 ng ml–1 GDNF (Peprotech), 1mM dibutyryl-cyclic AMP (Sigma), 200nM ascorbic acid (Sigma) and 1 µgml–1 laminin (ThermoFisher Scientific) 1– 2 days later. Cells were maintained in differentiated cultures reflect the dual lineage potential of the NPC, and therefore contain a neural population of βIII-TUBULIN-positive neurons, which comprise 70-80% of all cells in the culture. Another 20-30% of cells in culture are glial fibrillary acidic protein (GFAP)-positive astrocytes. In addition, while the majority of forebrain hiPSC neurons express the glutamatergic marker VGLUT1, another (approximately 30%) subset of neurons expresses the GAD67 GABAergic marker gene (Topol, Tran and Brennand, 2015).

Immunofluorescent staining

NPCs: NPCs on coverslips (24-well plate) were fixed in ice cold 4% PFA (Electron Microscopy Sciences, #15714) for 10 minutes and rinsed with PBS (containing Ca²⁺ and Mg²⁺; Thermo Fisher Scientific 21300-058). Blocking was performed with a buffer consisting 0.5% BSA/0.1% Triton-X (Sigma, #T8787)/PBS for 30 minutes at 4°C. Cells were incubated overnight at 4 degrees with primary antibody that was diluted in the blocking buffer. Primary antibodies used were Nestin (Alexa 647-conjugated, BD. #560341; 1:100) and SOX2 (Goat, Santa Cruz, #sc-17320; 1:500). Cells were then washed twice with PBS for 5 minutes. Then, secondary Alexa 488 donkey anti-goat (Jackson Immuno, #705-545-147; 1:400) was added in 300 µL PBS for 1-2 hours at room temperature. Cells were then washed again twice with PBS. Nuclei were counterstained with DAPI (Sigma, #D9542) for 10-15 minutes in 300 µL PBS. Coverslips containing the fluorescent stained NPCs were mounted onto slides with AguaPolymount mounting solution (Polysciences Inc., #18606-20) that was equilibrated to room temperature. Mounted coverslips were then left to air-dry overnight in the dark.

Neurons: Followed the staining protocol as per (Ho *et al.*, 2016). Primary antibodies used were TUJ1 (Rabbit, Covance, #PRB-435P; 1:1000) and MAP2 (Chicken, ABcam, #ab5392; 1:500). Secondary antibodies used were Alexa 488 donkey anti-chicken (Jackson Immuno, #703-545-155) and Cy3 donkeyantirabbit (Jackson Immuno, #711-165-152), both at 1:400. Glia: Followed the staining protocol as per (TCW *et al.*, 2017). Primary antibodies used were S100β (Mouse, Sigma-Aldrich, #S2532; 1:1000) and Vimentin (Rabbit, Cell Signaling, #3932; 1:500). Secondary antibodies used were Alexa 488 donkey anti-mouse (Jackson Immuno, #715-545-151) and Alexa 647 donkey-a-rabbit (Jackson Immuno, 711-605-152), both at 1:400.

All imaging was performed with a Zeiss LSM 780 confocal microscope.

Hi-C A/B compartment calling

To identify boundaries of low-resolution A/B compartments associated with open and closed chromatin, respectively, the first eigenvector from PCA on the Pearson's correlation matrix of Knight-Ruiz balanced observed/expected interaction frequencies (Knight and Ruiz, 2013) was calculated using `pearson` and `eigenvector` functions within the pre module of the Juicer tools suite (https://github.com/theaidenlab/juicer/wiki/Pre) at 500kb and 1Mb resolution (Durand, Shamim, *et al.*, 2016). Positively and negatively signed PC1 regions were used to establish compartments. For each library, the PC1 region that had higher gene density was defined as open-chromatin-associated compartment A while the other region was defined as closed-chromatin-associated compartment B as per previously described methods (Lieberman-Aiden *et al.*, 2009).

Hi-C topologically associating domain calling

Topologically associated domains (TADs) were called using the `arrowhead` algorithm in the Juicer tools suite with a 10kb resolution and 2kb sliding windows, `-r 10000 –m 2000 –ignore -sparsity`, on the ICE-corrected contact matrices. The distributions of all called TADs were compared using contact matrices sampled to have similar cis interactions (see below). Statistically significant differences in distributions were determined using the Wilcoxon-Mann-Whitney test.

Cumulative distribution of loop size

Cumulative plots were generated using loop calls from subsampled Hi-C data. Two-sample two-tail Kolmogorov-Smirnov test was used to determine the *p*-value, testing the hypothesis that two samples come from the same distribution. Refer to the script "loop_distance.R" at the following link: https://github.com/tborrman/Schahram-project.

Gene expression v. loop analyses

To test whether looping architecture was associated with increased gene expression, we overlapped genes with our non-subsampled HiCCUPS loop calls. We defined Gene Body Loop genes to be genes that overlap with loop anchor loci and defined TSS Loop genes to be genes whose TSS lies within a loop anchor loci. We then calculated the mean log₁₀(FPKM + 1) for Gene Body loop genes and TSS Loop genes, and performed one-tail *Z*-tests to determine if expressions in gene sets were significantly high. Refer to the scripts "get_overlapping_genes.py" and "FPKM_cell_type_specific_boxplots.R" at the following link: <u>https://github.com/tborrman/Schahram-project</u>.

Gene ontology

To examine the functional role of significant interactions, genes present in bins participating in interactions of interest (e.g., brain-specific loops, celltype-specific schizophrenia risk interactions, etc) were tested for enrichment of gene ontology categories using ClueGO (adjusted *p*-value threshold of < 0.05 with Bonferroni step-down or Benjamini-Hochberg correction, at "Medium" or "Medium-high" network specificity, and with "GO Term Fusion" enabled to avoid redundant terms (Bindea *et al.*, 2009). For the brain-specific loops GO, we took the 818 loops shared by all NPCs, glia, and astrocytes from the non-subsampled datasets. For the GO of loops lost in the neurons or glia upon transitioning from NPCs, the union set of all loops called with HiCCUPS was filtered to extract only those that were *q*-value < 0.1 ("present") in NPCs and *q*-value > 0.1 ("lost") in neurons or glia. Genes were annotated from anchors of the top 1000 most significant loops lost in neurons or glia.

ATAC-seq and accessibility processing

To assess chromatin accessibility, ATAC-seq was performed as described in (Buenrostro *et al.*, 2015). Briefly, 50,000 cells from each sample were snap frozen, resuspended in cold lysis buffer (10 mM Tris-HCl pH 7, 10 mM NaCl, 3 mM MgCl₂, 0.01% Igepal CA-630) and spun down at 500 x g for 10 min at 4°C. Cells were incubated with 25 μ L TD buffer, 2.5 μ L Tn5 transposase (Illumina; Cat No: #FC-121-1030) and 22.5 μ L nuclease-free H₂0 at 37°C for 30 min. After purification (Qiagen; Cat No: #28004), transposed DNA fragments were amplified with custom Nextera PCR primers (Buenrostro *et al.*, 2013) for a total of 9 cycles. 287 To minimize GC and size bias, the final PCR cycle number needed to minimally amplify libraries was determined by a separate qPCR reaction as described in Buenrostro et al., 2015. Purified libraries were sequenced on an Illumina HiSeq 2500.

To evaluate accessibility, ATAC-seq raw FASTQs were quality (minimum Q20) and adapter trimmed using Trim Galore! v0.4.1

(https://www.bioinformatics.babraham.ac.uk/projects/trim_galore/). Trimmed reads were aligned to hg19 using `bwa mem` v0.7.17. Aligned reads were then Insertion-Deletion realigned and Base Quality Score Realigned using the Genome Analysis Tool Kit v3.1.1 (McKenna *et al.*, 2010). Duplicates were marked using Picard MarkDuplicates v1.137

(https://broadinstitute.github.io/picard/). Genome-wide accessibility signal was computed from R1 alignments with 5' ends shifted 4 bases and 3' ends shifted 5 bases towards the center of the purported transposition event. Shifted R1 alignments were then aggregated at each base pair. *NGN2*-neuron Rep1 and NPC libraries were subsampled to approximately 227 million reads, comparable to the lowest coverage library, *NGN2*-neuron Rep2. Aggregated coverage in the subsampled libraries was log2-converted. To evaluate genome-wide accessibility, the mean accessibility signal across 1kb-tiled windows was calculated. The mean ATAC-seq signal within HiCCUPS loop anchor bin coordinates was used to assess the accessibility of loop calls.

Estimating significance of rate of loop/eQTL overlap

In order to assess whether the observed rate of overlap of our brainspecific (i.e., loops shared by NPCs, neurons, and glia) Hi-C loops with the Common Minds Consortium brain cis-eQTLs (with FDR $< 10^{-5}$) was significant, we first constructed a set of background loops. To do so, we annotated the anchors of the observed loops with functional classes using ChIPseeker (Yu, Wang and He, 2015). We used separate annotations to represent the 0-1kb, 1-2kb, 2-3kb, 3-4kb, and 4-5kb upstream promoter. To retain any hidden nuance in exon/intron annotations, we also preserved intron and exon numbers 1 through 5 and a separate merged class of intron and exon numbers 6 or greater. Downstream intergenic and unassociated intergenic served as additional sets of annotations. We similarly annotated the set of all 10kb genomic bins used in creating the Hi-C contact matrix. To create the null sets, we randomly selected a genomic bin that matched the annotation of the first anchor of our observed loops, then scanned up and downstream for a second bin a similar distance away (within $\pm 25\%$ of the observed loop distance), with a matching functional annotation to the second loop anchor. For each loop, we tried up to 100 random bin selection iterations to find a suitable bin pair match (with a failure rate of approximately 0.1%). Thus, each null set was similar or identical in size to the observed loop set. All bins associated with our observed loops and the bins ± 20 kb flanking its anchors were excluded from the null sets. We overlapped the background loops with the Common Minds cis-eQTLs with FDR<10⁻⁵ to estimate

the null distribution of the rate of loop/eQTL overlap. To do so, we randomly sampled 1000 null sets as described and calculated the fraction of each that fully overlapped an eQTL—requiring both SNP and gene-body to overlap each loop anchor. The one-sided empirical *p*-value was defined as the fraction of null-loop/eQTL overlap rates that were greater than or equal to our observed brain-specific-loop/eQTL overlap rate.

Comparing interaction intensity within interactions with PCDH locus

Observed/Expected (O/E) interaction scores were extracted from the subsampled Glia, NPC, and Neuron *.hic files using juicer-tools (https://github.com/aidenlab/juicer/wiki/Data-Extraction) (Durand, Shamim, *et al.*, 2016). Interactions falling within the regions of interest were selected: 1. chr5:140120000:140222664 || chr5:140110382:140960850 2. chr5:139730111-139952633 || chr5:140023665-140119999 Violin plots for all bin-bin interactions falling within the regions of interest were plotted and significant differences in O/E scores were tested for using a Wilcoxon-Rank Sum test.

Single SNP-level eQTL analysis

For the 61 genes within the PCDH locus with above-threshold expression, gene-level gene expression quantitative trait loci (eQTL) were obtained for the rs111896713 genotype across the N = 579 genetically inferred samples within the published CommonMind Consortium dataset (Fromer *et al.*, 2016) A linear model on the imputed genotype dosages was used followed by an *f*-test with 290 Bonferroni correction for multiple testing.

Real-time qPCR

Quantitative expression analysis was performed with a QuantStudio 7 Flex Real-Time qPCR System using the Power SYBR Green RNA-to-Ct Real Time gPCR kit for primers (Thermo Fisher Scientific). 50 ng of RNA template was added to PCR master mix containing primers. qPCR conditions were 48°C for 15 minutes, 95°C for 10 minutes, and 40 cycles of 95°C for 15 seconds followed by 60°C for 60 seconds.

Determining cell-type-specific interactions

The distribution of all -log₁₀ *q*-values from Hi-C interaction mapping of PGC risk loci indicated the 95th percentile log q-value = 1.47. For an interaction to be cell type-specific, it has to exceed this threshold in one cell type while that same interaction must have log q-value below the 50th percentile (log₁₀ q = 0.33) in the other two cell types. Similar thresholds were applied for contacts anchored in the newer CLOZUK risk loci (95th percentile $-\log_{10} q = 1.42$, 50th percentile - $\log_{10} q = 0.30$).

RNA-seq and transcriptome processing

A reference transcriptome, built from recently published RNA-seq datasets filtered through a stringent set of quality controls, included 47 NPC and 47 neuronal cultures from 22 independent donors including 11 subjects diagnosed with childhood onset schizophrenia (5 females, 6 males) (using unmodified DSM

criteria, with age of onset ranging from 4-12 years of age) and 11 controls (5 females 6 males) (Hoffman *et al.*, 2017). The donors range in age at biopsy was 8-30 years, as described (see Table 1 and Supplemental Data 1, 2 from reference (Hoffman *et al.*, 2017)). Using a cut-off of > 1 counts per million (CPM) in at least 30% of the experiments, the reference set included altogether 56,632 expressed sequences, including annotated genes, non-coding RNA, etc. Random distance-matched samplings (1,000x) were run to assess the significance of pair-wise gene correlation strength for each of the risk-associated chromosomal connectomes of the 3 cell types separately.

In addition, we generated RNA-seq libraries of glia from donor #2 and NPCs and excitatory neurons from both donors #1 and #2 that were used for the Hi-C studies (who are independent from the donors for the reference transcriptome described above). Libraries were prepared with the Kapa Total RNA library prep kit with ribo-depletion and strand-specific cDNA library construction (Kapa Biosystems). The libraries were then sequenced (125bp paired-end) on the Illumina HiSeq2000 platform (New York Genome Center). RNA-seq reads were aligned with STAR v2.4.0g1 (Dobin *et al.*, 2013) to GRCh37, after which uniquely mapped reads overlapping genes were counted with featureCounts v1.4.4 (Liao, Smyth and Shi, 2014) (with ENSEMBL v70 annotations). FPKM values were generated using Cufflinks (Trapnell *et al.*, 2012) and then compared by hierarchical clustering, multi-dimensional scaling and principal component analyses to 2082 RNA-seq data sets from stem cells and their differentiated counterparts, and adult and fetal brain tissue, and blood as described in (Hoffman et al., 2017). Genes with > 1 count per million in 10% of the samples in each data set were retained. This left 12,670 genes in common across all data sets. All expression values were converted to log₂ RPKM, resulting in 12,670 genes common to all 2082 datasets. Of note, RNA-seg of subject S1 and S2 hiPSC-derived NPC clustered together with these previous NPC RNAseq and prenatal BRAINSPAN datasets from different individuals. However, S1 and S2 neuronal cultures (differentiated by NGN2 into glutamatergic neurons and also by neural differentiation medium and BDNF and GDNF growth factors into a more mixed population primarily comprised of forebrain-like neurons) clustered together with the previously generated RNA-seq from neuronal cultures (from a different set of individual) and in close proximity to pre- and postnatal BRAINSPAN brain tissue samples. Finally, our RNA-seq from the astrocyte-like glial cells (differentiated from NPC of Subject S1) did not cocluster with either NPC or neurons, as expected. Thus, transcriptome mapping further confirms the specificity of our various cell culture systems.

Protein-protein interaction network analysis

To assess whether the proteins of the genes associated with schizophrenia risk loops are linked by physical functions or interactions, we input lists of genes from cell type-specific schizophrenia risk chromatin contacts for each cell type into the STRING v10.5 database (<u>http://string-db.org</u>) (Szklarczyk *et al.*, 2015, 2017). Default settings were used except for 1) "confidence" as the meaning of network edges and 2) "high confidence (0.700)" for the minimum required interaction score. We noticed that in the NPC and glia high-confidence networks the significance could have been driven by numerous connections ("edges") among the UGT gene cluster, all located within one 10kb bin on the genome. While continuing our analyses with the remaining highest confidence protein-protein associations, we re-ran the same gene lists, with all but one UGT gene (*UGTA10*) removed, at medium confidence to confirm significant networks ($P_{NPC} = 0.0438$; $P_{Neuron} = 0.0876$; $P_{Glia} = 0.0134$).

Human postmortem prefrontal cortex tissue

Postmortem brain tissues were derived from the University of Pennsylvania Brain bank, for which subjects with schizophrenia were prospectively accrued. Subjects with psychiatric illnesses were evaluated for Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) diagnostic criteria for SCZ. Diagnosis was determined by consensus of at least two board-certified research psychiatrists after comprehensive review of medical records, direct clinical assessments and interviews with caregivers. Autopsy consent was obtained from the next of kin or a legal guardian in all cases, based on a protocol approved by the Institutional Review Board at the University of Pennsylvania. Control and psychiatric subjects were matched for sex, age, smoking history and postmortem interval Table 3.22. All subjects with schizophrenia were treated with antipsychotics except 6 (24%) at the time of death.

Biochemical fractionation

Whole tissue homogenates were prepared from gray matter of the dorsolateral prefrontal cortex (DLPFC), BA 9 or 46, of SCZ and matched control subjects (Table 3.22) using a variation of the protocol we have developed and validated in human postmortem brain tissue. 50 mg grey matter was homogenized in .5 ml solution A (0.32 M sucrose, 1mM MgCl² and 0.1mM CaCl²) with a Teflon pestle. ~45 µl of the homogenate (H) was saved, solubilized with 1% SDS and clarified by centrifugation.

Sample preparation and LC-SRM/MS

20 µg whole tissue homogenate preparations were mixed with the [$^{13}C_6$] brain ISTD at a ratio of 1:1 (µg/µg) and processed for LC-SRM/MS (liquid chromatography selected reaction monitoring mass spectrometry) analysis by on-gel trypsin digestion as described (MacDonald *et al.*, 2012). LC-SRM/MS analyses were conducted on a TSQ Vantage triple stage quadrupole mass spectrometer (ThermoFisher Scientific) with an Eksigent 2Dnano LC (Eksigent) and a CaptiveSpray source (Michrom). 5 µl (~2.5µg protein) sample was loaded on to a Magic C18 column (Michrom) at 1 µl/min for 12 min, and eluted at 750nl/min over a 25 min gradient from 3-35% mobile phase B (ACN containing .1% formic acid). SRM transitions were timed using 1 – 1.5 min retention windows, depending on the number of SRMs to be assayed. Transitions were monitored, allowing for a cycle time of 1 sec, resulting in a dynamic dwell time, never falling below 10 msec. The MS instrument parameters were as follows:

capillary temperature 275 oC, spray voltage 1100 V, and a collision gas of 1.4 mTorr (argon). The resolving power of the instrument was set to 0.7 Da (FWHM) for Q1 and Q3. Data were acquired using a chrom filter peak width of 4.0 sec.

Mass spectrometry data processing, informatics and statistics

Peak areas and area ratios were calculated within Pinpoint (Thermo Scientific). Raw files generated by LC-SRM/MS analysis were loaded into Pinpoint files containing target proteins/peptides/transitions. All individual SRM transitions and integration areas were manually inspected. Transitions for which the signal-to-noise ratio was below 3 were excluded from analysis. The ratios of the integrated areas for "light" endogenous peptides and "heavy" [¹³C₆]brain ISTD peptides were calculated to obtain peptide measures using multiple transitions per peptide. Peptide measures from the same protein were averaged to obtain the measure for that protein. Prior to comparison of SCZ and control groups, protein measures were normalized in each fraction. Outlier subjects were identified by manual inspection for the number of individual protein or enrichment value outliers and principle component analysis of the entire dataset, run as a plug-in in Excel. Permutation analysis to determine significant coexpression of synaptic proteins of interest was performed by randomly sampling 4 proteins from the pool of 182 synaptic proteins and calculating the organization score for each set of 4.

295

CHAPTER IV: ATLAS: A DATABASE LINKING BINDING AFFINITIES WITH STRUCTURES FOR WILD-TYPE AND MUTANT TCR-PMHC COMPLEXES

Preface

This chapter comprises work published in PROTEINS: Structure, Function, and Bioinformatics by myself, Jennifer Cimons, Michael Cosiano, Michael Purcaro, Brian G. Pierce, Brian M. Baker, and Zhiping Weng. The publication reference is "ATLAS: A database linking binding affinities with structures for wild-type and mutant TCR-pMHC complexes" *PROTEINS: Structure, Function, and Bioinformatics*. Vol. 85 Issue 5 May 2017 (Borrman *et al.*, 2017)

Data curation was performed by myself, Jennifer Cimons and Michael Cosiano. Frontend and backend of the ATLAS website was developed by myself and Michael Purcaro. All data analysis and regression modeling was performed by myself. Research was supervised by Zhiping Weng, Brian G. Pierce and Brian M. Baker. The paper was written and figures were produced by myself, Brian G. Pierce, Brian M. Baker, and Zhiping Weng with contributions from all coauthors. **Abstract**

The ATLAS (Altered TCR Ligand Affinities and Structures) database (https://zlab.umassmed.edu/atlas/web/) is a manually curated repository containing the binding affinities for wild-type and mutant T cell receptors (TCRs) and their antigens, peptides presented by the major histocompatibility complex (pMHC). The database links experimentally measured binding affinities with the corresponding three dimensional (3D) structures for TCR-pMHC complexes. The user can browse and search affinities, structures, and experimental details for TCRs, peptides, and MHCs of interest. We expect this database to facilitate the development of next-generation protein design algorithms targeting TCR-pMHC interactions. ATLAS can be easily parsed using modeling software that builds protein structures for training and testing. As an example, we provide structural models for all mutant TCRs in ATLAS, built using the Rosetta program. Utilizing these structures, we report a correlation of 0.63 between experimentally measured changes in binding energies and our predicted changes.

Introduction

The binding of a T cell receptor (TCR) to an antigenic peptide presented by a major histocompatibility complex (pMHC) is a fundamental step in cellmediated immunity. To eliminate pathogens and diseased cells, TCRs recognize foreign antigens displayed by MHC molecules on the surface of antigen presenting cells (Zinkernagel and Doherty, 1974; Babbitt *et al.*, 1985) (Figure 4.1, a). This recognition triggers T cell activation and an ensuing signaling cascade that leads to an antigen-directed cellular immune response.

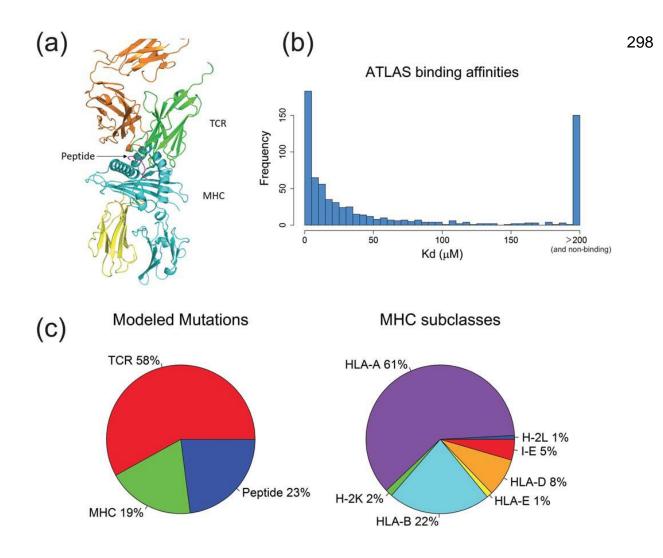


Figure 4.1 | ATLAS data statistics.

(a) A6/Tax/HLA-A*02:01 TCR-pMHC complex (PDB: 1AO7). TCR α and β chains (green, orange), MHC molecule (cyan), peptide (magenta), and β_2 microglobulin (yellow) are shown in cartoon style. (b) Histogram of the binding affinities of TCR-pMHC complexes in ATLAS. (c) Pie charts for percentage of entries with modeled mutations made to the TCR, the MHC or the peptide (left), percentage of MHC subclasses (right).

The ability to manipulate TCR-pMHC recognition has broad applications in

a variety of biomedical arenas. One example is adoptive T cell transfer, which

uses tumor recognizing T cells to eradicate cancer cells (Restifo, Dudley and Rosenberg, 2012). The TCRs of these T cells can be genetically engineered to enhance their affinities toward specific tumor antigens (Varela-Rohena *et al.*, 2008; Linette *et al.*, 2013; Morgan *et al.*, 2013). In a related approach, high affinity TCRs can be used as soluble biologic therapeutics to target tumorassociated antigens (Oates and Jakobsen, 2013). Another example is peptidebased vaccination, which uses peptides to selectively stimulate T cells capable of battling infections or cancers (Purcell, McCluskey and Rossjohn, 2007). Many tumor associated peptides are derived from self proteins and are only weakly immunogenic because TCRs that strongly recognize self-antigens have been eliminated during negative selection in the thymus. Thus attempts have been made to develop modified peptides that can selectively enhance T cell activation (Chen *et al.*, 2005; Cole *et al.*, 2010).

TCR-pMHC binding strength is an important parameter in determining the quality of the ensuing immune response. TCR affinity has been shown to correlate positively with T cell activation (Holler and Kranz, 2003; Aleksic *et al.*, 2010); however, robust immune responses appear to result from TCR affinities in an optimal range, which is not necessarily high (Stone and Kranz, 2013). Past an apparent affinity threshold, the strength of the T cell response may plateau or attenuate.

Another consideration while striving for a desirable immune response is to avoid the cross-recognition of TCRs between foreign and self peptides, which

can lead to autoimmune diseases, such as multiple sclerosis, type 1 diabetes, and paraneoplastic syndromes (Wooldridge *et al.*, 2012; Sethi *et al.*, 2013). Before any potential therapeutic use of T cells, it is vital to identify off-target binding (Obenaus *et al.*, 2015); this consideration is particularly important for engineered TCRs, as demonstrated by adverse events in clinical trials (Linette *et al.*, 2013; Morgan *et al.*, 2013).

Precise prediction and manipulation of both TCR affinity and specificity is therefore essential for designing effective T-cell-based therapeutics. A number of methods have been developed for altering TCR-pMHC interactions, including in vitro molecular evolution and structure-guided protein design (Holler *et al.*, 2000; Li *et al.*, 2005; Varela-Rohena *et al.*, 2008; Borbulevych *et al.*, 2009; Zoete *et al.*, 2013; Malecek *et al.*, 2014; Pierce *et al.*, 2014). Structure guided design algorithms can alter affinity and specificity directly and efficiently, but are limited by the accuracy of their scoring functions.

Prediction of protein-protein binding affinity from protein-complex structure is a challenging problem. When nine unique scoring functions developed for docking programs or web servers were tested on a benchmark of 81 protein complexes, correlations between scores and binding affinities were low or nonexistent (*r* ranging from -0.18 to 0.32) (Kastritis and Bonvin, 2010). More recent studies utilizing supervised learning methods have increased correlations between predicted and experimental affinities, and there is still room for improvement (Vreven *et al.*, 2012; Xue *et al.*, 2016). Prediction of changes in binding energy due to point mutations has seen greater success. Correlations between predicted and experimental $\Delta\Delta G$ in a study analyzing >1,500 point mutations ranged from 0.28 to 0.61 depending on the prediction method used (Geng, Vangone and Bonvin, 2016). Progress in $\Delta\Delta G$ prediction is critical to the field of TCR design where point mutants may be made to increase a TCR's affinity toward an antigen to trigger a robust immune response.

The improvement of TCR design algorithms requires access to both structural and binding data. We have built the ATLAS (Altered TCR Ligand Affinities and Structures) database (https://zlab.umassmed.edu/atlas/web/) to meet this demand. ATLAS links measurements of TCR affinity with structural information, and allows a user to query for a TCR, MHC, or peptide of interest. Results from such queries include details on affinity, mutation information, and structures of associated TCR-pMHC complexes that exist in the Protein Data Bank (Berman *et al.*, 2000). ATLAS includes structures for the relevant mutant TCRs that have been studied. If PDB structures for the relevant mutant complexes are not available, the database provides computationally modeled TCR-pMHC structures.

The immune epitope database (IEDB) (Vita *et al.*, 2015) and the Anti-Jen database (Toseland *et al.*, 2005) both provide binding affinities for TCR-pMHC complexes; however, these databases are peptide-epitope-centric and do not allow the user to query specific TCRs. Furthermore, there is no direct link between affinity and structural data in these databases. The IEDB does allow the

user to filter queries based on the availability of X-ray crystallography and surface plasmon resonance (SPR) experiments; however, in many cases a query using one peptide epitope will return multiple TCR-pMHC complexes that contain the peptide. Hence, to correctly match a TCR-pMHC complex with its reported binding affinity, the user needs to manually inspect the literature.

In comparison with IEDB and AntiJen, ATLAS allows the user to search specific TCRs, MHCs, and peptides. Full datasets in ATLAS can also be downloaded as flat files. With the goal of providing a repository to train and test next generation TCR design strategies and scoring functions, ATLAS also provides experimental details such as the resolutions of the structures and references for each of its entries. As low resolution structural data may skew scoring results, this information will be critical for the selection of a subset of the data to optimize prediction algorithms. As of this writing, the database includes data only for $\alpha\beta$ TCRs, but can be readily extended as more experimental data for the $\gamma\delta$ TCR family becomes available.

Results

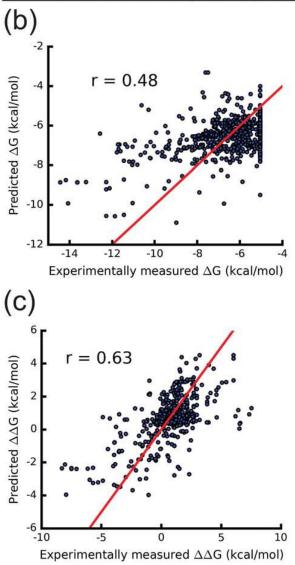
The ATLAS database currently contains affinity and structural data on human and mouse TCR-pMHC complexes, with a total of 694 measured affinities ranging from high nanomolar affinities to low affinities with $K_D > 200 \mu$ M (Figure 4.1, b, c). As more affinity data exist than structural data, ATLAS also contains a set of models for all TCR-pMHC affinity entries lacking crystal structures (see Materials and methods). Below, we first illustrate a usage case for the data in ATLAS, and then we describe how to query the ATLAS database via the web interface.

Using the data in ATLAS to develop energy functions

As a proof of concept, we performed multiple linear regressions using ATLAS data to develop scoring functions capable of affinity prediction. We examined two cases of energy prediction: prediction of TCR-pMHC binding energy, ΔG , and prediction of change in binding energy upon mutation, $\Delta \Delta G$ (see Materials and methods). Prior to regression analysis, no correlation was found between experimental and predicted ΔGs (or $\Delta \Delta Gs$) using the Rosetta modeling software's standard scoring function (Leaver-Fay et al., 2011). Eight energy terms from Rosetta—solvation, hydrogen bonding expressed in four terms, attractive and repulsive van der Waals, and a statistical pair potentialwere used in the regression model (Figure 4.2, a).

(a)

Energy term	2.16	P-value 10 ⁻⁴¹
Lennard Jones van der Waals energy; attractive		
Lennard Jones van der Waals energy; repulsive	0.03	0.67
Lazaridis-Karplus solvation energy	1.67	10-23
statistics-based pairwise energy	0.16	0.04
backbone-backbone hydrogen bonds; distant in primary sequence	-0.60	10-14
backbone-backbone hydrogen bonds; close in primary sequence	-0.24	10-5
sidechain-backbone hydrogen bonds	-0.31	10 ⁻⁵
sidechain-sidechain hydrogen bonds	0.49	10-12



304

Figure 4.2 | Results of predicting binding free energies in ATLAS. (a) Table of coefficients and p-values for all energy features of the regression analysis. One insignificant feature (repulsive van der Waals) is highlighted in red. (b) Scatterplot of predicted ΔG versus ΔG determined by SPR for all ATLAS entries. Linear regression analysis was performed to predict ΔG using the following best set of features: attractive van der Waals energy, Lazaridis–Karplus solvation energy and all four hydrogen bond energy terms. r = 0.48 and RMSE = 1.48 kcal mol⁻¹. (c) Scatterplot of predicted $\Delta \Delta G$ versus $\Delta \Delta G$ determined by SPR for 575 mutant ATLAS entries determined by regression analysis using the following best set of features: attractive van der Waals energy, Lazaridis–Karplus solvation energy and side-chain-side-chain hydrogen bond energy. r = 0.63 and RMSE = 1.58 kcal mol⁻¹. Red line represents perfect prediction.

Performing leave-one-out cross-validation (LOO-CV) on all 694 ATLAS

entries, we report a correlation of 0.65 between predicted ΔG and experimentally measured ΔG with a root mean square error (RMSE) of ~1.09 kcal mol⁻¹. However, many of the ATLAS entries differed by only a few residues and used the same PDB structure as the template for structure modeling. To accurately assess scoring function performance, we ran a cross-validation scheme where the training set for each prediction excluded any entries that used the same PDB structure as the template to model the structures. For example, many ATLAS entries were mutants of the A6-Tax/HLA-A2 TCR-pMHC complex and used the 1AO7 PDB structure as the template for modeling the mutant structures. For prediction of any of these mutants, we exclude from the training set all other mutants that were also modeled using 1AO7 as the template. Following this leave-one-complex-out cross-validation (LOCO-CV) scheme, we report a correlation *r* of 0.45 between experimental and predicted ΔG s and an RMSE of 1.52 kcal mol⁻¹. 305

The attractive van der Waals energy along with the solvation energy were 306 the most important features for prediction, judged by the *P* values for these features (10^{-41} and 10^{-23}). We asked whether steric clashes in the modeled structures might have caused the poor performance of the repulsive van der Waals term. However, even after removing outlier entries with unfavorable repulsive van der Waals terms from the regression model, the coefficient for the repulsive van der Waals term remained insignificant. To extract the best predicting combination of features, we implemented LOCO-CV on all 255 combinations of the eight energy features. A slight increase in performance was seen when the van der Waals repulsive and pair potential terms were removed from the regression model, r = 0.48 and RMSE = 1.48 kcal mol⁻¹ (Figure 4.2, b).

We then proceeded to build multiple linear regression models for prediction of $\Delta\Delta G$ s. We included all multiple residue mutation cases in this study; however, 73% of the $\Delta\Delta G$ mutations were single residue mutations. Following the LOCO-CV scheme and analyzing all feature combinations, we report the maximal correlation of 0.63 between experimental and predicted $\Delta\Delta G$ with an RMSE of 1.58 kcal mol⁻¹ (Figure 4.2, c). This best performing model used only the attractive van der Waals energy, solvation energy, and side-chain–side-chain hydrogen bond energy to predict $\Delta\Delta G$.

Given that the majority of structural data used for training in the regression model was designed via Rosetta, we assessed the accuracy of these modeled structures. Twenty-one out of the 694 ATLAS entries had both wildtype and mutant crystal structures available (Table 4.1). Seven of these 21 entries were point mutations to residues with at least one side-chain dihedral angle (γ). The other 14 entries were either point mutations to Ala or were complexes designed with multiple mutations. We used these seven entries to evaluate the accuracy of the modeled mutant structures in ATLAS. The Dunbrack rotamer library was used to assess whether Rosetta designed mutant side chains had the same rotamers as those of the mutant crystal structures (Shapovalov and Dunbrack, 2011). Here, a side-chain conformation is defined as one of the combinations of multiple χ angle conformations for a side-chain. We found that four of the seven mutant side chains were of identical conformation to the side chain in the crystal structure. For two of the three side chains with incorrect conformations, only the terminal χ angle was inaccurate (χ^2 in Asp and χ^3 in Glu). Hence, only one sidechain, a mutation from Val to Arg, was completely mismodeled (Table 4.2). Although this is only a small sample of the entire dataset, it provides some evidence that the majority of the designed ATLAS mutations have correctly modeled side-chain conformations.

The modeled TCR-pMHC complexes from the regression analysis were generated using a fixed backbone approach. However, the complementarity determining region (CDR) loops of TCRs can change conformations upon binding with pMHC (Gagnon et al., 2006; J. B. Reiser et al., 2003; J. B. Reiser et al., 2002). To assess whether modeling flexibility of CDR loops could improve affinity prediction performance, we also generated another set of modeled complexes via the Rosetta backrub application which accounted for flexibility of 308 the CDR loops. These structures were then employed in our LOCO-CV scheme. Analyzing all features combinations, we saw a modest reduction in Δ G prediction performance, r = 0.45 and RMSE = 1.50 kcal mol⁻¹ for the set of best performing features. A larger reduction in performance was found in prediction of $\Delta\Delta$ G, r =0.47 and RMSE = 1.80 kcal mol⁻¹ (compared with r = 0.63 and RMSE = 1.58 kcal mol⁻¹ for the fixed backbone approach) (Figure 4.3). This result was not entirely surprising as previous studies have reported poorer correlations when modeling backbone flexibility compared with using fixed backbone calculations (Mandell and Kortemme, 2009).

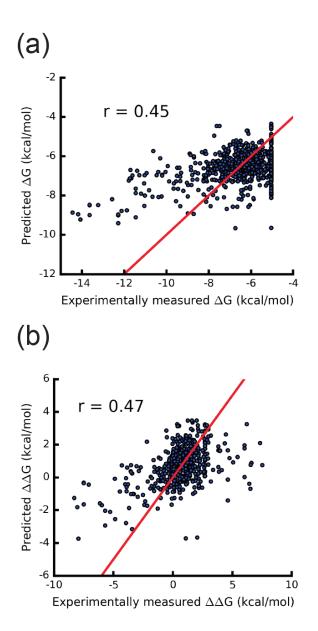


Figure 4.3 | Results of predicting binding free energies in ATLAS after modeling flexibility of CDR loops.

(a) Scatterplot of predicted ΔG versus ΔG determined by SPR for all ATLAS entries. Linear regression analysis was performed to predict ΔG using the following best set of features: attractive van der Waals energy, Lazaridis–Karplus solvation energy and backbone-backbone hydrogen bond energy close in primary sequence. r = 0.45 and RMSE = 1.50 kcal mol⁻¹. (b) Scatterplot of predicted $\Delta\Delta G$ versus $\Delta\Delta G$ determined by SPR for 575 mutant ATLAS entries determined by regression analysis using the following best set of features: attractive van der Waals energy, Lazaridis–Karplus solvation energy, statisticsbased pairwise energy, and repulsive van der Waals energy. r = 0.47 and RMSE 310 = 1.80 kcal mol⁻¹. Red line represents perfect prediction.

Although there is room for improvement, TCR-pMHC binding affinity prediction is feasible through the use of the structural information in ATLAS. As a starting point for engineering TCRs, peptides, or MHCs to enhance binding affinity, the ATLAS database is a useful resource to guide the design process.

The web-based user interface of ATLAS

As many users may be only interested in a specific TCR or peptide, ATLAS provides a searchable interface so that the user can extract the relevant data of interest. To browse the entire ATLAS dataset, the user may simply submit a search leaving all fields with their default parameters.

As a demo, we queried for all entries that contain the human A6 TCR, the MHC allele HLA-A*02:01, and a peptide whose amino sequence contained LFGYPVY, with binding free energies lower than -6 kcal mol⁻¹ (Figure 4.4, a). Note that the user may also search ATLAS by specifying *TRAV* or *TRBV* genes, as well as MHC allele or class. Submission of the search form brings the user to the results page (Figure 4.4, b). Each ATLAS entry (row) of the search results corresponds to a unique TCR-pMHC complex with an experimentally determined binding affinity and a 3D structure which can be used as a template for design. The binding affinity is reported in both K_D (μ M) and ΔG (kcal mol⁻¹). The *PDB* column provides the PDB ID for a structure matching the TCR-pMHC complex with the reported experimental binding affinity. For many entries an exact

structure corresponding to the recorded binding affinity does not exist. To make 311 use of such binding data, the *Template PDB* column refers to the PDB ID for a template structure for which a TCR-pMHC complex matching the reported binding affinity may be generated by modeling the mutations described in the *TCR mutation*, *MHC mutation*, and *Peptide mutation* columns of the entry. For further information on each entry, the results page provides a link to the abstract of the publication in which the binding affinity was determined in the *PMID* column. Lastly, the query results can be downloaded as individual files directly from the results page.

(a)

Search ATLAS

Search Results

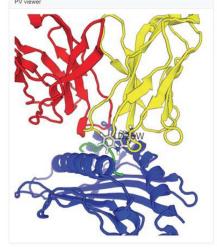
Search through the ATLAS database by selecting a specific TCR, MHC allele, energy upper bound, or peptide sequence motif. The ATLAS search returns entries satisfying all selected search fields. To browse the entire dataset simply click 'Search' leaving all fields with their default parameters. For a more extensive description of search categories and criteria please see the help page.

TCR	(1)	MHC	(2)	Energy	3) Peptide (4
Name:	\bigcirc	Class:	\bigcirc	ΔG (kcal mol ⁻¹) <	Peptide Sequence:
A6	*	all	٠	-6.00	LFGYPVY
TRAV:		Allele:			
all	*	HLA-A*02:01	*		
TRBV:					
all	*				



TCR name ↓	TCR mutation	TCR mutation chain	MHC allele	MHC mutation	MHC mutation chain	Peptide	Peptide mutation	К ₀ (µМ)]↑	∆G (kcal mol ⁻¹) ⊥⊺	PDB	Template PDB	PMID
A6	WT	nan	HLA-A*02:01	WT	nan	LLFGYPVYV	WT	2.113502935	-7.74	1A07		1876716
A6	D26M	A	HLA-A*02:01	WT	nan	LLFGYPVYV	WT	3.014285714	-7.53		1A07	18767161
46	D26V	A	HLA-A*02:01	WT	nan	LLFGYPVYV	WT	21.1	-6.38		1A07	18767161
46	D26W	A	HLA-A*02:01	WT	nan	LLFGYPVYV	WT	0.327868853	-8.84		1A07	18767161
46	R27F	A	HLA-A*02:01	WT	nan	LLFGYPVYV	WT	1.469489415	-7.95		1A07	1876716

(c)



Template PDB 1A07: Template PDB 1A07 with the following mutation(s): TCR mutations: D28W TCR mutations: D28W MHC mutations: None Peptide mutations: None	Download	
Designed model of 1AO7 with the following mutation(s): TCR mutations: D26W TCR mutation chains: A MHC mutations: None MHC mutation chains: None	Template PDB 1A07:	
TCR mutations: D26W TCR mutation chains: A MHC mutations: None MHC mutation chains: None	Template	
TCR mutation chains: A MHC mutations: None MHC mutation chains: None	Designed model of 1AO7 with the following m	utation(s):
MHC mutations: None MHC mutation chains: None	TCR mutations: D26W	
MHC mutation chains: None	TCR mutation chains: A	
	MHC mutations: None	
Peptide mutations: None	MHC mutation chains: None	
	Peptide mutations: None	
	C Designed	
Designed		

Figure 4.4 | ATLAS web interface and data accession.

(a) The search page for querying the ATLAS database. ATLAS is searchable by TCR (1) and MHC (2) features, binding energies ΔG below a user specified kcal/mol (3) and by case-insensitive peptide sequence motifs (4). (b) The search results page linking binding energies to complex structures. The PDB structure specified in (1) refers to a structure identical to the TCR-pMHC used in the binding assay. The template PDB structure in (2) can be designed to replicate the TCR-pMHC used in the binding assay by modeling the mutations listed in the TCR mutation, MHC mutation, and Peptide mutation columns. (c) Protein Viewer and individual PDB downloads. An example shows the selection of PDB ID 1AO7 from the Template PDB column for the D26W TCR mutant. Template structures and Rosetta designed structures are both available for download.

Selecting a PDB ID from the results page brings the user to the PV

Javascript Protein Viewer (Marco Biasini, 2015) and downloadable PDB content.

Continuing the demo, we selected the template PDB 1AO7 for the D26W TCR

mutant from our previous results page (Figure 4.4, c). The Rosetta modeled

mutant structure is displayed in the PV viewer. The modeled mutant tryptophan

side-chain of the TCR is highlighted in ball-and-stick style in the protein complex.

From this page the user can download the individual template PDB complex

along with the Rosetta-designed mutant PDB complex, both structures adjusted

for consistency as described in the Materials and methods section.

Downloading data tables of ATLAS

The Downloads page provides the four tables used to build ATLAS in Microsoft Excel format. The *TCR gene table* contains the *TRAV* and *TRBV* genes for all TCRs in ATLAS. The *MHC class table* contains the classes for all MHC alleles. The *ATLAS table* provides all of the structural and affinity data for each ATLAS entry and is the extended version of tables found by browsing or searching the web interface. Lastly, we also provide the set of consistency adjusted TCR-pMHC structures described earlier, *TCR-pMHC structures*, which contain the template PDB structures and the mutant structures. We provide two sets of mutant structures: (1) structures predicted using the fixed backbone approach and (2) structures predicted allowing flexibility in CDR loops via the Rosetta backrub application. All mutant structures are generated using the mutation information recorded in ATLAS.

Discussion

The multiple search parameters of ATLAS make it particularly useful for studying specific subsets of TCR or MHC. One recent application involved identifying TCRs that recognized the human Class I MHC allele HLA-A* 02:01 and TCRs that recognized human Class I MHC alleles that were not HLA-A*02:01. The links to PDB structures were used to make structural comparisons between the two groups of TCRs (Blevins *et al.*, 2016). Additionally, the option to search entries by *TRAV/TRBV* genes, MHC allele, and peptide sequence allow for comparisons to be made involving these parameters. For example, searching by a variable chain segment can allow the user to compare the effects that mutations within the shared chain have on binding affinity. The accumulation of affinity values for all published binding studies also allows for the identification of potentially important residues for pMHC recognition. Similarly, searching by peptide sequence can identify all TCRs known to recognize a particular peptide

(or a substring of residues in a peptide), as well as how mutations on the peptide 315 impact TCR binding.

As a further example to demonstrate the utility of a large TCR database, ATLAS was recently used in a separate study to identify single point mutations when training a generalized approach for engineering TCRs (Riley *et al.*, 2016). In this study, nearly 200 point mutations in multiple HLA-A2 restricted TCRs (A6, B7, DMF5, and DMF4) and one HLA-B8 restricted TCR (LC13) were modeled using Rosetta and utilized in a multiple linear regression model. Using Rosetta energy terms and molecular dynamics derived flexibility terms as predictor variables and the experimental binding energies as the response variable, a score function was parameterized which emphasized van der Waals forces, solvation effects, and flexibility. This score function was rigorously crossvalidated and found to estimate the effects of any given mutation relative to wild type with an average error of <1.5 kcal mol⁻¹ and was used to identify additional affinity enhancing mutations in the B7, DMF5, and DMF4 TCRs.

We have developed the ATLAS database as a centralized resource to link structural and binding data for TCR-pMHC complexes, with an emphasis on the impacts of mutations within TCR-pMHC interfaces. The database can be queried multiple ways, and when structures do not exist, ATLAS provides modeled structures, as well as the means to visualize experimental or modeled structures. We anticipate that ATLAS will be useful in the design and optimization of TCRs, including the development of next-generation design algorithms for TCR-pMHC interactions. It can also be used in combination with other large datasets of structural and affinity data, such as the AB-Bind database of antibody affinities (Sirin *et al.*, 2015), which would be useful in structure-based immune receptor design. Beyond this, the database may serve as a resource for studies aiming to correlate structural and biophysical binding data with immunological outcomes.

Tables

Wildtype	Mutant	TCR	TCR mutation	MHC mutation	MHC mutation	Peptide mutation
PDB	PDB	mutation	chain		chain	
2VLJ	2VLR	S99A	В	WT	nan	WT
3MV7	3MV9	Q55A	В	WT	nan	WT
3MV7	3MV8	Q55H	В	WT	nan	WT
1AO7	1QSE	WT	nan	WT	nan	V7R
1AO7	1QRN	WT	nan	WT	nan	P6A
1AO7	1QSF	WT	nan	WT	nan	Y8A
2AK4	3KXF	WT	nan	Q65A T69A Q155A	A A A	WT
1AO7	3QFJ	WT	nan	WT	nan	Y5F
3HG1	4JFF	D27F R28L I50T S52R N53E Y71H V93D A94G K96R S97L L44P S50W V51G G52P I53F T100M E102G L103W F104Q	A A A A A A A A A A	WT	nan	WT
4JFF	4JFD	WT	nan	WT	nan	G4A
4JFF	4JFE	WT	nan	WT	nan	17A
1AO7	4FTV	A99M G100S G101A R102Q	B B B B	WT	nan	WT
3VXR	3VXS	WT	nan	WT	nan	F6L
4PRI	4PRH	WT	nan	WT	nan	E5D
4G8G	4G9F	WT	nan	WT	nan	L6M
2F54	2F53	Q50P S51F S52W G49S A50V I52M	A A A B B B	WT	nan	WT
2BNR	2P5E	Q51T S52P S53W T95L S96L G97D S99T G50A A51I G52Q I53T V95L	A A A A A A A B B B B B	WT	nan	WT
2BNR	2P5W	Q51T S52P S53W G50S A51V I53M T95L S96L G97D S99T V95L	A A A B B B A A A A B	WT	nan	WT
3QDG	4L3E	D26Y L98W	A B	WT	nan	WT
3QIU	3QIW	WT	nan	WT	nan	K9E
4P23	4P46	Y31A	A	WT	nan	WT

 Table 4.1 | ATLAS entries with both wildtype and mutant crystal structures available.

Designed PDB	Experimental PDB	Designed Rotamer	Experimental Rotamer	Designed χ Angles	Experimental χ Angles
3MV7_WT_nan_Q55H_B_WT.pdb	3MV8.pdb	2,6	2,6	175.53, -150.03	167.17, -143.99
1AO7_WT_nan_WT_nan_V7R.pdb	1QSE.pdb	2,2,2,3	3,1,2,2	-163.59, -173.07, -170.48, -86.61	-110.06, 46.25, -175.26, 162.23
1AO7_WT_nan_WT_nan_Y5F.pdb	3QFJ.pdb	3,1	3,1	-61.62, 97.64	-47.74, -77.15
3VXR_WT_nan_WT_nan_F6L.pdb	3VXS.pdb	3,2	3,2	-65.24, 166.16	-69.84, 160.56
4PRI_WT_nan_WT_nan_E5D.pdb	4PRH.pdb	3,1	3,5	-72.25, -15.16	-95.35, 115.36
4G8G_WT_nan_WT_nan_L6M.pdb	4G9F.pdb	3,2,1	3,2,1	-59.85, 169.33, 72.42	-63.51, 177.08, 68.47
3QIU_WT_nan_WT_nan_K9E.pdb	3QIW.pdb	3,2,5	3,2,3	-64.02, 165.74, -55.63	-42.88, -165.85, 60.11

Table 4.2 | Rotamer analysis for designed mutations.

Materials and methods

Data collection

To collect data suitable for training and testing TCR-pMHC scoring functions, we required all ATLAS entries to meet the following two criteria: (1) The affinity of the TCR-pMHC must be measured experimentally with purified proteins (most frequently) using SPR or isothermal titration calorimetry (ITC); and (2) The 3D structure of the complex has been determined experimentally, or for mutants, a template wild-type structure exists in the PDB. To provide the most comprehensive list of TCR-pMHC complexes, we did not make any quality restrictions pertaining to the affinity or structure data; instead, we recorded the resolution of crystallographic structures in the full dataset flat files available in the Downloads page.

To identify TCR-pMHC complexes for inclusion in ATLAS, we first found all crystallographic structures of TCR-pMHC complexes in the IMGT database (Ehrenmann, Kaas and Lefranc, 2010) verified by a careful inspection of the corresponding PDB entries. We next manually searched the literature for experiments measuring the affinity of each TCR-pMHC complex, including measurements with TCR or MHC mutants and/or peptide variants. If we could identify quantitative data on binding affinity, we then proceeded to include the TCR-pMHC complex in ATLAS, along with experimental details as metadata for the entry. We describe these metadata fields in the following sections.

Metadata fields

As many of the binding experiments recorded in ATLAS tested the effects of mutations on TCR-pMHC affinity, it follows that the majority of ATLAS entries pair mutant affinity data with wild-type template structures as opposed to the actual mutant TCR-pMHC complexes to which the binding affinities refer. Thus we recorded detailed information on the mutations such that one could build a 3D structure model of the mutant complex that corresponded to the affinity data, given the template structure. With this application in mind, the data tables of ATLAS are designed not only for information, but also for easy parsing by protein design software. The following five fields represent the information required for modeling a mutant TCR-pMHC complex structure from a template structure.

- TCR mutation: <wild-type residue><residue number><mutant residue>
- *TCR mutation chain*: <chain>; A for α or B for β chain
- MHC mutation: <wild-type residue><residue number><mutant residue>
- *MHC mutation chain*: <chain>; A for α chain B for β chain
- Peptide mutation: <wild-type residue><residue number><mutant residue>

Some fields may be left empty if one or more molecules in the template structure are the same as the molecules used to measure affinity. In the case of a complete match between the complex structure and the complex tested for binding, all mutation fields may be left empty. As mentioned previously, all entries are required to have at least a template structure and in some case both experimental and template structures for mutants exist. These cases could be particularly helpful in assessing the accuracy of the structural modeling.

Inconsistent chain naming, structure boundaries, and prevalence of water molecules complicate the design and scoring process. To overcome these challenges, we also supply a set of files for all experimentally determined TCR pMHC structures in ATLAS with the following consistency adjustments: renaming of chains, truncation of chains to the binding domains, and removal of water molecules. When there were multiple complexes in the asymmetric unit of a crystal structure, the first complex was selected.

Protein modeling

As a proof of principle, we wrote a script to parse ATLAS and build models using Rosetta for all listed mutations upon the adjusted template structures. These models were built using the fixed backbone design option of Rosetta, fixbb, and are available for download. The example parser script, "build_models.py," is also available at the Github site of ATLAS (https://github.com/weng-lab/ATLAS). Although this example is specific for design via the Rosetta protein modeling software, the ATLAS database can be easily integrated with any other design software.

Regression analysis

Many ATLAS entries have low affinities with unreliable ΔG s or undetectable binding. Hence, we assigned all entries with a ΔG > -5.05 kcal mol⁻¹ (K_D > 200 μ M) and all non-binding entries to have ΔG = -5.05 for the regression analysis.

The following equation was used to calculate ΔG for each independent

variable in the regression model:

$$\Delta G = \Delta G_{COMPLEX} - (\Delta G_{TCR} + \Delta G_{pMHC}), \tag{1}$$

where $\Delta G_{COMPLEX}$ is the energy of a feature in the regression model for the TCRpMHC complex and ΔG_{TCR} and ΔG_{pMHC} are the energies of a feature for the isolated TCR structure and isolated pMHC structure, respectively.

Similarly, we use the following equation to define $\Delta\Delta G$ for changes in energy upon mutation:

$$\Delta \Delta G = \Delta G_{MUT} - \Delta G_{WT}, \qquad (2)$$

where ΔG_{WT} is the ΔG for a wild type TCR-pMHC entry and ΔG_{MUT} is the ΔG for a mutated version of the wild type (mutations may involve multiple residues).

Coefficients for the regression models were estimated by the ordinary least squares method. P-values were calculated from the *t* statistics of the coefficients using a two tailed *t* test. All regression calculations were made using the python statistics module statsmodels.

Architecture

The backend of ATLAS was built using the archetypal web service solution stack, LAMP, consisting of Linux (Ubuntu version 14.04), Apache (version 2.4.7), MySQL (version 5.5.41) and PHP (version 5.5.9). The front end was designed using the Bootstrap framework (version 3.3.5). All programs related to ATLAS are available at github (https://github.com/weng-lab/ATLAS).

CHAPTER V: HIGH-THROUGHPUT MODELING AND SCORING **OF TCR-PMHC COMPLEXES TO PREDICT CROSS-REACTIVE** PEPTIDES

Preface

This chapter is adapted from a manuscript currently under review at Bioinformatics authored by myself, Brian G. Pierce, Thom Vreven, Brian Baker, and Zhiping Weng titled: High-throughput modeling and scoring of TCR-pMHC complexes to predict cross-reactive peptides.

Peptide sequence extraction and filtering, modeling of TCR-pMHC complexes, computational scoring, and prediction performance analysis was performed by myself. Original project hypothesis was conceived by Brian G. Pierce. Research was supervised by Thom Vreven, Zhiping Weng and Brian M. Baker. The paper was written and figures were produced by myself, Brian G. Pierce, Thom Vreven, Brian M. Baker, and Zhiping Weng.

Abstract

The binding of T cell receptors (TCRs) to their target peptide MHC (pMHC) ligands initializes the cell-mediated immune response. In autoimmune diseases such as multiple sclerosis, the TCR erroneously recognizes selfpeptides as foreign and activates an immune response against healthy cells. Such responses can be triggered by cross-recognition of the autoreactive TCR with foreign peptides. Hence, it would be desirable to identify such foreignantigen triggers to provide a mechanistic understanding of autoimmune diseases. 324 However, the large sequence space of foreign antigens presents an obstacle in the identification of cross-reactive peptides.

Here, we present an *in silico* modeling and scoring method which exploits the structural properties of TCR-pMHC complexes to predict the binding of crossreactive peptides. We analyzed three mouse TCRs and one human TCR isolated from a patient with multiple sclerosis. Cross-reactive peptides for these TCRs were previously identified via yeast display coupled with deep sequencing, providing a robust dataset for evaluating our method. Our method accurately selected the top binding peptides from sets containing more than a hundred thousand unique peptides.

Introduction

As a surveillance mechanism against pathogens and cancer, T cells of the host immune system use their $\alpha\beta$ T cell receptors (TCRs) to inspect other cells. Targets recognized by TCRs are peptides bound and presented by the host major histocompatibility complex (MHC) proteins on the outer surface of the cellular membrane, and the peptide epitope may derive, for instance, from a viral protein. TCR recognition triggers complex signaling pathways that lead to a variety of outcomes, such as the destruction of infected or diseased cells, T cell proliferation, and release of pro-immune cytokines.

Determining peptide epitopes that can be recognized by TCRs is of considerable interest, impacting fields ranging from virology to cancer immunotherapy. Peptide immunogenicity involves three steps, each of which have been addressed via various predictive algorithms: peptide processing (Bhasin and Raghava, 2004; Nielsen *et al.*, 2005), peptide binding to an MHC (Andreatta and Nielsen, 2015; Jurtz *et al.*, 2017; O'Donnell *et al.*, 2018), and TCR recognition of the peptide-MHC (pMHC) complex (Tung *et al.*, 2011; Pierce and Weng, 2013; Schneidman-Duhovny *et al.*, 2018; Lanzarotti, Marcatili and Nielsen, 2019; Ogishi and Yotsuyanagi, 2019; Riley *et al.*, 2019). While progress has been made in predicting the outcome of each step, the fixed size of the TCR repertoire relative to the much larger number of possible peptide epitopes that T cells may encounter presents a particularly significant challenge.

Even with a TCR repertoire estimated to lie in the tens of millions, estimates are that any particular TCR would need to recognize at least one million different pMHC complexes in order to provide sufficient immune coverage (Mason, 1998; Sewell, 2012). This high level of cross-reactivity has been verified using combinatorial peptide libraries (Maynard *et al.*, 2005; Wooldridge *et al.*, 2012). Thus, although specificity is considered a hallmark of immunity, TCRs display significant cross-reactivity. Even if such cross-reactivity can be rationalized at a high level from structural and biophysical principles (Singh *et al.*, 2017), determining the range of peptides recognized by a specific TCR remains a major goal in immunology. Demonstrating the biological significance of the

problem, TCR cross-recognition of self-peptides is believed to underlie various autoimmune disorders (Gravano and Hoyer, 2013), and patient deaths have occurred due to unanticipated "off-target" recognition of TCRs used in clinical trials for cancer immunotherapy (Linette et al., 2013; Morgan et al., 2013).

Given the availability of TCR-pMHC structural information, together with advances in protein design and prediction methodologies, in principle, the peptide specificity profile of a TCR should be predictable using *in silico* methods. One challenge, however, is the availability of detailed experimental datasets against which such prediction methods could be benchmarked. In addition to combinatorial peptide libraries, Garcia and colleagues have used yeast display of pMHC libraries coupled with TCR staining and deep sequencing to assess the specificity profiles of TCRs (Birnbaum et al., 2014; Adams et al., 2016; Gee et al., 2018). With each yeast cell expressing a unique random peptide, these libraries allow for affinity-based interrogation of over one hundred million peptides against a query TCR. Affinity based selection proceeds through multiple rounds where yeast libraries are enriched for yeast that bound bead-multimerized TCR. Subsequent deep sequencing of yeast DNA from final selection rounds produces enrichment counts for peptides selected by the query TCR. Such experiments provide rich datasets for developing and benchmarking in silico approaches to evaluating TCR specificity.

Here, we used structure-based *in silico* methods to predict the specificity profiles for four TCRs assessed using yeast display and deep sequencing: 2B4, 326

226, 5cc7, and Ob.1A12 (Birnbaum *et al.*, 2014). Three of these TCRs recognize 327 a peptide derived from moth cytochrome C presented by the murine class II MHC protein I-E^k (Newell *et al.*, 2011). The fourth (Ob.1A12) was isolated from a patient with relapsing-remitting multiple sclerosis and recognizes a peptide derived from the myelin basic protein presented by the human class II MHC protein HLA-DR2) (Wucherpfennig *et al.*, 1994). The deep sequencing data provided more than 100,000 peptides for each TCR, including both binders and non-binders, ideal for benchmarking structure-based *in silico* methods.

Using the crystal structures for the four TCR-pMHC complexes (Hahn *et al.*, 2005; Newell *et al.*, 2011; Birnbaum *et al.*, 2014), we modeled all of the query peptides within the TCR-pMHC complexes and scored the structural models to predict cross-reactive peptides for each of the four TCRs. Our modeling and scoring approach was capable of recovering cross-reactive peptides from large pools of primarily non-binding peptides for each TCR tested. We further show that our method outperforms the strategy of selecting peptides closest in sequence to each TCR's cognate peptide epitope (i.e., the target peptide found in crystallographic structure), underscoring the value of including structural information in epitope prediction.

Results

High-throughput modeling reproduces experimentally observed enrichment of binder peptides

Previously described experimental yeast display and deep sequencing generated libraries that were enriched for peptides specifically recognized by four TCRs (Birnbaum *et al.*, 2014). Beginning with the crystallographic structures of the 2B4, 226, 5cc7, and Ob.1A12 TCRs in complex with their cognate pMHC complexes, we computationally modeled and scored the peptides with sequences in the preselection libraries and four sequential selection rounds— 347,210 peptides for 2B4, 811,481 peptides for 226, 809,156 peptides for 5cc7, and 514,906 peptides for Ob.1A12, and 2,482,753 peptides in total (Birnbaum *et al.*, 2014).

To increase the computational throughput in modeling the structures of these approximately 2.5 million peptides, we performed a restricted structural modeling procedure using Rosetta's fixed backbone design application, fixbb, which optimizes side-chain conformations on a fixed backbone using the Rosetta energy function (Leaver-Fay, Snoeyink and Kuhlman, 2008). We retained TCR and MHC side chains in the conformations adopted in the crystallographic structures with their cognate pMHCs (Figure 5.1, A and B).

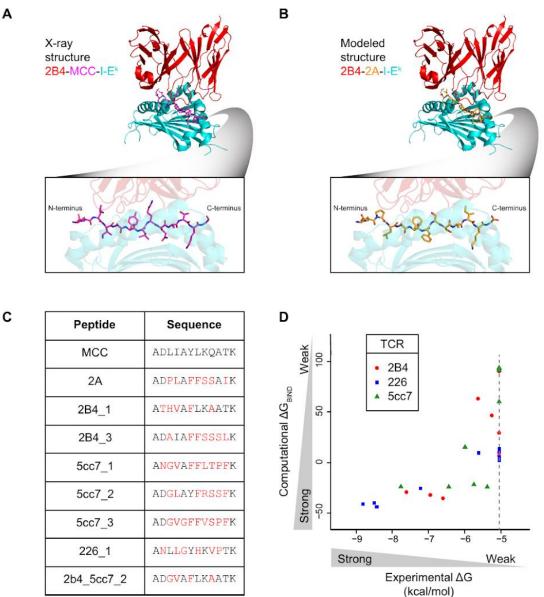


Figure 5.1 | Prediction of TCR-pMHC binding free energies.

(A) Crystal structure of TCR-pMHC interface for the 2B4 TCR (red) interacting with the MCC peptide (magenta) displayed by the I-E^k MHC (cyan). Bottom box: profile of the MCC peptide. (B) Fixed backbone model structure of TCR-pMHC interface for the 2B4 TCR (red) interacting with the 2A peptide (yellow) displayed by the I-E^k MHC (cyan). 2A peptide was modeled onto the backbone of the MCC peptide in (A), TCR and MHC protein structures remain identical to (A). Bottom box: profile of the 2A peptide. (C) Table of peptide names and amino acid sequences. Amino acids in red differ from the MCC peptide at the corresponding

А

329

position. (**D**) Scatter plot of ΔG_{BIND} from computational modeling and scoring versus ΔG from experimental binding energies determined by SPR from Birnbaum et al. for peptides in Table (C). Pearson correlations are 0.73, 0.98 and 0.70 for TCRs 2B4, 226, and 5cc7, respectively. Correlation for the entire set of peptides = 0.70. Peptides with unreliable K_Ds due to weak or non-binding interactions were assigned a K_D of 200 µM (ΔG = -5.05 kcal/mol, gray dotted line). Individual TCR correlations were similar if we assigned a ΔG of 0 kcal/mol for these weak/non-binding peptides and the correlation for the entire set remained the same (r = 0.70).

Once each TCR-pMHC model was generated, we scored the full complex ($G_{COMPLEX}$) and isolated components ($G_{TCR/MHC}$, $G_{PEPTIDE}$) using Rosetta's score application (see Materials and methods). These scores were combined to produce a binding score, ΔG_{BIND} , which accounted for the peptide's interaction energy with both the MHC and the TCR.

To quantitatively assess our peptide modeling and scoring approach, we examined sets of peptides for which experimental binding free energies were available for the 2B4, 226, and 5cc7 TCRs (Figure 5.1, C) (Birnbaum *et al.*, 2014). Correlations between ΔG_{BIND} and experimentally measured binding free energies were greater than 0.69 for all TCRs, and 0.70 for the entire set together (Figure 5.1, D).

We next examined the distributions of ΔG_{BIND} across the four experimental selection rounds of the yeast display library where each successive round was further enriched in cross-reactive peptides via TCR selection. Indeed, ΔG_{BIND} scoring of modeled complexes revealed an increasing enrichment of favorable energy scores for peptides in each subsequent selection round, in congruence with the subsequent enrichment of cross-reactive peptides for each round (Figure

5.2). Thus, relying on a relatively simple structural modeling method to enable computational throughput permits the recovery of experimentally determined peptides bound by TCR.

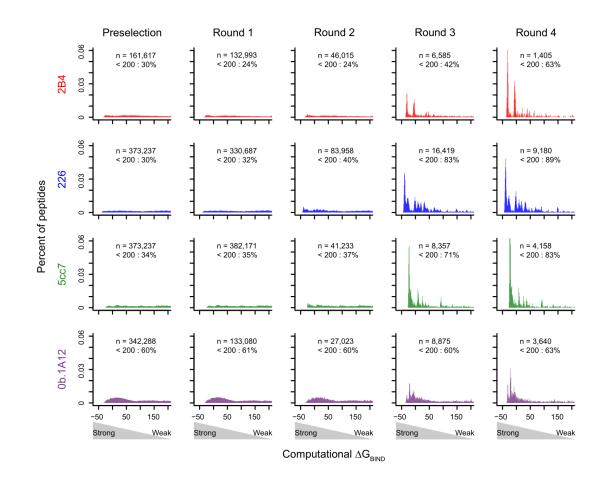


Figure 5.2 | Distributions of ΔG_{BIND} for peptides recovered from different selection rounds.

We generated structural models of TCR-pMHC complexes using peptide sequences from all experimental selection libraries. For each unique peptide recovered in each selection round, we modeled its structure bound to MHC and TCR and computed ΔG_{BIND} for the TCR-pMHC complex. The probability densities for ΔG_{BIND} are plotted for each round of selection for the four TCRs analyzed in this study, 2B4, 226, 5cc7 and Ob.1A12. The probability density is defined such that the histogram has a total area of one. (n = total number of unique peptides in

331

the given round; < 200: percent of peptides in the round with ΔG_{BIND} less than 200).

Footnote: The deep sequencing results present only a sample of the total unique peptides in each selection round. It is thus important to note, the Round 4 peptides while a subset of the full experimental pre-selection library with >10⁸ peptides, is not a strict subset of the >10⁵ unique peptides recovered from sequencing the pre-selection library.

Correlation between top computationally selected peptides and top

experimentally selected peptides

To examine the extent to which our modeling and scoring approach selected the peptides recognized by the TCRs with the strongest affinities, we compared the 50 peptides with the most favorable ΔG_{BIND} to the 50 peptides with the most reads recovered by deep sequencing after the fourth round of selection for the 2B4, 226, 5cc7, and Ob.1A12 TCRs (Birnbaum et al., 2014). Rather than simply selecting from the round-four peptides, which are highly enriched in binders, we asked the computational method to identify top-scoring peptides from the pool of unique peptides in the union of the preselection and the round four libraries, providing for a more unbiased test. We note, while the full preselection pool contains all the round four peptides, the deep sequencing data present only a sample of the preselection pool peptides. Hence, we use the union of both the preselection and round four sequencing to ensure the true binders are accounted for. For a successful computational method, we would expect peptides with the most favorable ΔG_{BIND} to be members of the smaller, round-four peptide sets and to share amino acid preferences with the peptides that have the most abundant

reads in the fourth round. The amino acid preference generated using the 50 top 333 experimentally selected peptides (50 peptides with the most abundant reads counts in round four) illustrated binding motifs distinct for each TCR (Figure 5.3, A). Among the 50 peptides that had the most favorable ΔG_{BIND} according to our modeling and scoring method, 41, 45, 24, and 38 were among the peptides in the round-four library for the 2B4, 226, 5cc7, and Ob.1A12 TCRs, respectively. Therefore, our scoring method was capable of identifying true binders within a large pool consisting primarily of non-binder peptides.

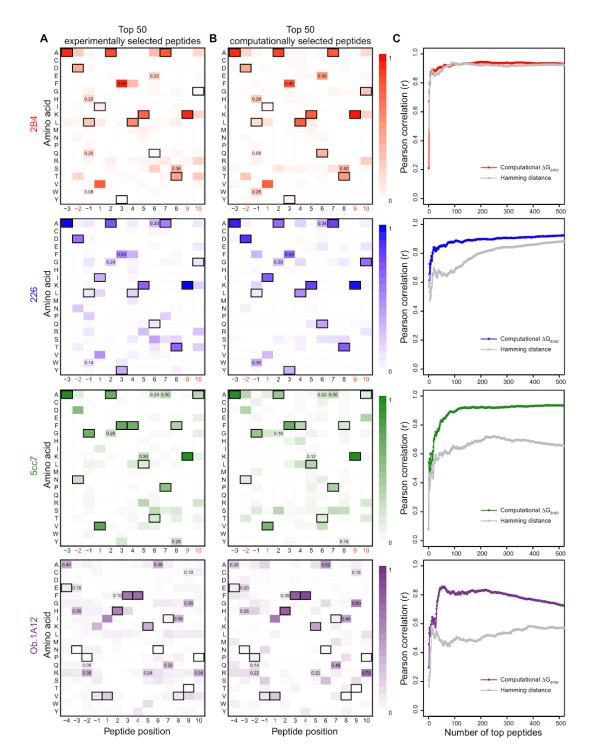


Figure 5.3 | Amino acid frequencies for top peptides selected by yeast display or by computation for mouse and human TCRs.

(A) Heatmaps represent the amino acid frequencies at peptide positions for the 50 peptides with the most abundant reads in the fourth round of selection for four TCRs (2B4, 226, 5cc7, and Ob.1A12). (B) Amino acid frequencies at peptide positions for the 50 peptides with the most favorable ΔG_{BIND} . Peptide pool for ΔG_{BIND} computation was the union of the preselection library and round 4 library (>10⁵ peptides). The peptide residues from the template TCR-pMHC structures used for modeling, (2B4, 226) MCC, (5cc7) 5c1, and (Ob.1A12) MBP are outlined in black. Peptide positions restricted in the yeast display libraries to maintain MHC binding are marked in red beneath heatmap. Correlations of frequencies between the experimental and computational heatmap for 2B4, 226, 5cc7, and Ob.1A12 TCRs are 0.91, 0.85, 0.83, and 0.85 respectively (excluding restricted positions). (C) Pearson correlations between the experimental and computational ΔG_{BIND} heatmaps as a function of the number of top selected peptides used to generate the heatmaps for each TCR (red, blue, green, purple). Pearson correlations between the experimental heatmap and heatmap generated from peptides with lowest hamming distance to wildtype peptide for each TCR (2B4-MCC, 226-MCC, 5cc7-MCC, Ob.1A12-MBP) as a function of the number of top selected peptides used to generate the heatmaps (gray).

To further examine how the best scoring peptides compared to those identified experimentally after TCR selection, we compared heatmaps of amino acid preferences for the 50 top experimentally selected peptides and the top 50 computationally selected peptides (50 peptides with the most favorable ΔG_{BIND} from the union of preselection and round 4) for each TCR (Figure 5.3, A and B). Many sequence features were shared between the top-scoring peptides and the peptides with the most abundant read counts. To quantify similarity between heatmaps, we flattened the heatmap matrices into vectors and calculated the Pearson correlation between them. Excluding those anchor positions restricted in the libraries for MHC binding, correlations between the heatmaps representing

strongly selected and top-scoring peptides were 0.91, 0.85, 0.83, and 0.85 for TCRs 2B4, 226, 5cc7, and Ob.1A12, respectively.

We also assessed a different binding score that only incorporated interaction energies between TCR and pMHC. A comparison of the different binding scores revealed the inclusion of interaction energies between peptide and MHC as in ΔG_{BIND} was critical to the success of cross-reactivity predictions (Figure 5.4). A possible reason is that some of the peptides in the preselection library may not bind stably to the MHC. Because a stable peptide-MHC interaction is a prerequisite for TCR binding, the incorporation of interaction energies between peptide and MHC improved the correlation between prediction and experiment. We also tested the $G_{COMPLEX}$ score by itself. While ΔG_{BIND} represents interactions between the peptide and TCR and also the peptide and MHC, GCOMPLEX additionally accounts for deformations of the peptide itself. However, predictive performance of $G_{COMPLEX}$ was worse than that of ΔG_{BIND} with correlations between the heatmaps representing strongly selected and topscoring peptides being 0.81, 0.85, 0.80, and 0.26 for TCRs 2B4, 226, 5cc7, and Ob.1A12, respectively.

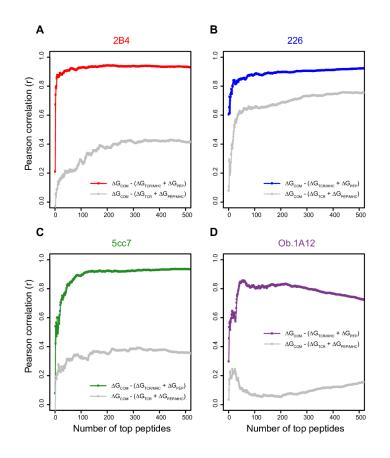


Figure 5.4 | Prediction comparison of scoring approaches.

Pearson correlations between the experimental and computational ΔG_{BIND} heatmaps of cross-reactivity profiles (See Figure 5.3) as a function of the number of top selected peptides used to generate the heatmaps for each TCR (A, B, C, D). Correlations for scoring approach such that

 $\Delta G_{BIND} = G_{COMPLEX} - (G_{TCR/MHC} + G_{PEPTIDE})$

is shown in red, blue, green and purple lines. Correlations for scoring approach such that

 $\Delta G_{BIND} = G_{COMPLEX} - (G_{TCR} + G_{PMHC})$

is shown in gray.

We note that in the case of Ob.1A12, our scoring method was successful 338 in assigning favorable scores to peptides carrying the 'HF' motif as was found experimentally (Birnbaum *et al.*, 2014). It is evident from the experimental heatmap that Ob.1A12 is tolerant of amino acid substitutions outside the anchor residues and the central HF motif. This feature of Ob.1A12 is also captured by our modeling and scoring method, with the exception of a strong preference for Lys at position -1 in the experimental heatmap and a few other less frequent substitutions our method could not reproduce.

The 2B4, 226 and 5cc7 TCRs all recognize the MCC peptide (ADLIAYLKQATKG), which is presented in the TCR-pMHC crystal structures of 2B4 and 226, but the crystal structure for 5cc7 had a different peptide (5c1, ANGVAFFLTPFKA). Both the experimentally selected peptides and the topscoring peptides by our modeling method revealed peptide motifs similar to these cognate peptides (their residues are in black boxes in Figure 5.3).

Because our modeling method started with the ternary complex structure containing cognate peptides, our method may simply favor peptides with similar sequences. Nevertheless, our scoring method does reproduce many amino acid substitutions seen in the top experimentally selected peptides (marked with amino acid frequency in Figure 5.3). We defined a substitution to be shared between the experimental and computed peptide sets if the frequency of the mutant amino acid at its peptide position was 2 fold higher than what would be expected by chance in both heatmaps (based on the NNK codon library used to design the yeast-display libraries). The following substitutions are shared between the two heatmaps for 2B4: L-1H, L-1Q, L-1W, Y3F, Q6E, and T8S. For 226, the shared substitutions are L-1W, A2G, Y3F, and Q6A. For 5cc7, the shared substitutions are A2G, 5LK, T6A, P7A, and F8Y. For Ob.1A12, the shared substitutions are E-4A, N-3H, N-3E, P-2Q, P-2R, H2F, K5R, N6A, I7Q, V8I, T9G, T9C, P10R. Hence, although our modeling method may be biased toward the cognate peptide, our modeling and scoring method is still capable of identifying target peptides with beneficial or permissible mutations.

To provide a quantitative assessment of whether our method outperformed a baseline method simply based on sequence similarity to the cognate peptide, we computed the Hamming distance between the cognate peptide sequence and the sequence of each peptide in the union of the preselection and the round-four library for each TCR. The top peptides with the highest sequence similarity to the cognate peptide (lowest Hamming distance) were used to generate heatmaps of amino acid preference. The correlation between these heatmaps and heatmaps of the top experimentally selected peptides was compared to the equivalent correlation derived from the top ΔG_{BIND} peptides as described above (Figure 5.3, C). For one TCR (2B4) similar correlations were found when peptides were selected based on the ΔG_{BIND} score or Hamming distance. However, a notable improvement in correlation was detected for the other three TCRs (226, 5cc7, and Ob.1A12) when peptides were selected based on ΔG_{BIND} as opposed to mere sequence similarity with cognate peptide. The strong improvements in correlation for three of the four TCRs 3 highlight the value of incorporating structural information in next-generation peptide prediction algorithms.

Discussion

Numerous methods exist for the prediction of peptide binding to either class I or class II MHC molecules and have achieved high accuracy dependent upon the training and testing data utilized (Zhao and Sher, 2018). However, far fewer tools are available for prediction of TCR binding to pMHC and the accuracy of existing tools show room for improvement (Tung *et al.*, 2011; Pierce and Weng, 2013; Lanzarotti, Marcatili and Nielsen, 2018; Schneidman-Duhovny *et al.*, 2018; Ogishi and Yotsuyanagi, 2019). Utilizing structural information from four TCR-pMHC complexes, we present a high throughput modeling and scoring approach capable of successfully selecting cross-reactive peptides from large pools of primarily non-binding peptides. Our method outperforms the approach based on sequence similarity to the cognate peptides.

Several other groups incorporated structural information of the TCRpMHC interface to aid in binding prediction. In a recent study, optimized FoldX and Rosetta energy terms were used to predict peptide binding given the sequences of MHC, TCR and a query peptide (Lanzarotti, Marcatili and Nielsen, 2018). The authors noted that the availability of a high sequence identity structural TCR template and successful prediction of peptide binding to MHC were vital to the success of their TCR-pMHC binding prediction. Similarly, the method ITCell utilizes atomic statistical potentials to predict a TCR's peptide epitope from all possible peptides in the full-length parent protein when given sequences of class II MHC, the TCR variable region, and the parent protein antigen as input (Schneidman-Duhovny *et al.*, 2018). In the majority of test cases, ITCell ranked the correct peptide epitope among the top 20 peptides among all peptides that could result from the parent antigen.

Benchmarking sets for the aforementioned methods were generated by using overlapping peptides from the parent protein sequence of the cognate peptide as negatives (excluding the cognate), based on the assumption that parent protein sequence would harbor only peptide epitopes for a single TCR, which resulted in ~10²-10³ query peptides per TCR-pMHC test case (Lanzarotti, Marcatili and Nielsen, 2018; Schneidman-Duhovny *et al.*, 2018). A more exact set of non-binding peptides would require experimental evidence for failed binding. Here, we present deep-sequencing results from yeast display as a robust and larger benchmarking tool for TCR epitope prediction. In particular, each preselection library provided >10⁵ peptides, which were not selected by the TCR of interest and are likely negative non-binding peptides. Although 10⁵ peptides is still a small subset of the theoretical diversity for the 13-mer (~8.1 x 10^{16}) and 14-mer (~1.6 x 10^{18}) peptides, they represent a larger challenge than previous benchmarks for predicting TCR epitopes.

Like our study, the success of both of the aforementioned methods relied on accurate template-based modeling of the TCR-pMHC complex (Lanzarotti, 341

Marcatili and Nielsen, 2018; Schneidman-Duhovny et al., 2018). In our work, modeling of the TCR-pMHC was simplified because crystal structures of TCRpMHC complexes existed for all four TCRs investigated and only structural changes resulting from the different peptide sequences needed to be accounted for. Previous studies showed the TCR's complementarity determining region (CDR) loops can be flexible and change their conformations upon ligand binding (Reiser et al., 2002, 2003; Gagnon et al., 2006; Scott et al., 2011; Pierce and Weng, 2013). Furthermore, it has been shown CDR flexibility can contribute to cross-reactivity (Reiser et al., 2003; Hawse et al., 2014). It may be surprising how well our modeling and scoring method performed without making any structural adjustments to the TCR molecules. It is unlikely our modeling method could predict antigens that require large backbone movements, or altered binding orientation, of the TCR for recognition. However, while our modeling method is conservative in terms of modeling any structural changes of the TCRs CDR3 loops, it appears to perform well in providing poor scores for unfavorable peptides.

Large conformational changes of the peptide can also occur upon TCR binding. For example, the DMF5 TCR that recognizes the MART-1 melanoma antigen presented by the class I MHC protein HLA-A2 was shown to cross-react with the DRG class of peptides that are chemically distinct from MART-1 (Gee *et al.*, 2018). DMF5 TCR binding to an HLA-A2-presented DRG-class peptide led to a 'register shift' in the peptide, causing a C-terminal peptide extension from the

342

MHC binding groove (Riley *et al.*, 2018). Identification of cross-reactive peptides 343 with such large structural adjustments relative to cognate peptide would be missed by our fixed-backbone peptide-modeling approach, as would instances in which MHC deformations are required (Borbulevych *et al.*, 2009; Borbulevych, Piepenbrink and Baker, 2011). However, peptides of class II pMHC complexes (i.e., those studied here) typically do not bulge from the groove and class II pMHC complexes are thus less prone to backbone rearrangements (Tynan *et al.*, 2005; Ayres, Corcelli and Baker, 2017). Hence, the success seen here with class II complexes may not fully translate when predicting cross-reactivity in class I systems, although we should anticipate success with conformationally simpler modes of cross-reactivity that involve more commonly observed molecular mimicry mechanisms (Macdonald *et al.*, 2009; Borbulevych, Piepenbrink and Baker, 2011).

Even when accounting for simple molecular mimicry mechanisms in crossreactivity, peptide side-chain must also be precise because a single erroneous side-chain conformation could lead to false positive or false negative predictions. While we do not have an estimate for the accuracy of side-chain modeling for our modeled peptides here, our previous work showed Rosetta's side-chain optimization methods performed well, albeit on a limited set of TCR-pMHC point mutations (Borrman *et al.*, 2017). As advancements in technology allow for faster and more accurate modeling of larger conformational changes, future studies may focus on allowing for flexibility in CDR loops, MHC, and peptide backbone to potentially identify cross-reactive peptides with distinct structural and chemical 344 signatures.

To score the modeled TCR-pMHC structures, we accounted for the interactions made by the peptide with the TCR and the MHC using Rosetta's scoring application with default weights for energy terms. Future work could potentially improve upon our results by optimizing energy term weights using machine learning approaches and taking advantage of structural and chemical trends in known TCR-pMHC complexes. For example, there is evidence that immunogenic peptides are enriched in hydrophobic amino acids at peptide centers (Calis et al., 2013), and structural modeling combined with neuralnetwork optimized scoring has been used to predict neoantigen immunogenicity (Riley et al., 2019). Here, we employed timesaving modeling and scoring methods to efficiently interrogate large pools of peptides for binding. Future studies optimizing score functions and weights for predicting TCR cross-reactivity might take into account the consequences of the weak affinities TCRs have for their ligands, which can stem from 'imperfect' interfaces that are traditionally difficult to discriminate between with default functions.

One potential application of our method is in cancer immunotherapy. Accurate identification and targeting of neoantigens (peptides derived from mutated tumor proteins) could lead to successful development of immunotherapeutics. Recent work highlighted the importance of incorporating MHC binding strength, self-similarity to reference antigen, and peptide-centric features to accurately predict neoantigen immunogenicity (Bjerregaard *et al.*, 2017; Smith 345 *et al.*, 2019). Building on this and other work, structural modeling and scoring of peptide neoantigens in the context of the full TCR-pMHC complex rather than the MHC alone may provide additional insights beneficial to immunogenic prediction (Riley *et al.*, 2019).

Many efforts have been made to enhance TCR affinity for tumor and viral antigens (Holler et al., 2000; Li et al., 2005; Chervin et al., 2008). However, enhanced affinity may lead to increased cross-reactivity (Linette et al., 2013; Riley and Baker, 2018; Hellman et al., 2019). To check for unwanted crossreactivity of engineered TCRs, one may perform alanine scanning of the antigen to identify motifs essential for binding and then searching for possible selfantigens in a protein sequence database (Obenaus et al., 2015). The alanine scanning can be expedited using DNA barcode-labeled MHC multimers (Bentzen et al., 2016, 2018). A more direct approach is to interrogate all human peptides for cross-reactivity, like the recent T-Scan method which utilized the lentiviral delivery of an antigen library spanning the entire human proteome into antigenpresenting cells. Selected peptides confirmed the cognate MAGE-A3 epitope along with several novel cross-reactive endogenous self-peptides (Kula et al., 2019). Our modeling and scoring method could represent an *in silico* approach with a similarly broad coverage. We could scan all peptides of the entire human proteome computationally for possible binding to an engineered TCR granted a template TCR-pMHC crystal structure is available. The thus identified crossreactive antigens could be further tested experimentally using binding assays or 346 assays measuring immunogenic response.

Materials and methods

Sequence extraction

Sequencing data were downloaded from the Sequence Read Archive (SRA) under project code SRP040021 and converted to FASTQ files using the SRA Toolkit. Sequencing reads were split by barcode into their individual selection rounds. Nucleotide sequences were translated into amino acid sequences and peptide sequences containing stop codons or unknown amino acids were discarded. The resulting counts for each unique peptide were recorded for each round of selection for each TCR.

Peptide structure modeling

Template TCR-pMHC complex structures were downloaded from the protein data bank (PDB) with the following PDB IDs: 3QIB (2B4-MCC-I-E^k), 3QIU (226-MCC-I-E^k), 4P2R (5cc7-5c1-I-E^k), and 1YMM (Ob.1A12-MBP-HLA-DR2). To reduce computation time the structures were truncated to contain only the binding interface (up to residue 83 for the class II MHC α chain and residue 93 for the β chain). Each TCR was truncated to just contain the variable domains, excluding the constant domains that are distal from the binding interface. Water molecules were also removed to simplify scoring and for consistency across TCR-pMHC structures with different resolutions. To model peptides onto the template TCR-pMHC structures, we utilized the fixed backbone application, fixbb,

of the Rosetta suite of programs (Version 3.5) (Leaver-Fay, Snoeyink and Kuhlman, 2008; Leaver-Fay *et al.*, 2011), with parameters "extrachi_cutoff 1 –ex1 –ex2 –ex3" to increase χ angle rotamer sampling for side-chain placement of peptide residues. All side chains of the TCR-pMHC aside from those modeled on the peptide were left in their original poses. The following is an example Rosetta command used for peptide structural modeling:

rosetta_source/bin/fixbb.linuxgccrelease -database rosetta_database/ -s
pdb -resfile my_resfile -suffix my_label -extrachi_cutoff 1 -ex1 -ex2 ex3

Prediction of peptide–MHC/TCR binding free energy

Each modeled TCR-pMHC complex was scored using the following formula:

$$\Delta G_{BIND} = G_{COMPLEX} - (G_{TCR/MHC} + G_{PEPTIDE})$$
(1)

such that G_{COMPLEX} is the Rosetta score for the entire TCR-pMHC complex, G_{TCR/MHC} is the Rosetta score for the TCR and MHC chains bound without the peptide, and G_{PEPTIDE} is the score for the isolated peptide in its bound conformation. To score each component we used Rosetta's scoring application, score (Leaver-Fay *et al.*, 2011). This scoring function is a linear combination of 19 energy terms, including van der Waals, solvation, electrostatics, and hydrogen bonding interactions along with other statistical potentials. The weights for the energy terms were left in their default settings (those specified in the standard.wts file along with the score12.wts_patch file found in Rosetta's weights directory). The following is an example Rosetta command used for scoring modeled TCR-pMHC structures (i.e. scoring commands for GCOMPLEX, GTCR/MHC, and GPEPTIDE):

rosetta_source/bin/score.linuxgccrelease -database rosetta_database/ -s
my_pdb.pdb -out:file:scorefile outputfile.sc

CHAPTER VI: DISCUSSION

Defining compartment principles by liquid chromatin Hi-C

Knowledge pertaining to mechanisms driving compartmentalization of chromatin states in the nucleus is lacking. In Chapter II, I sought to answer fundamental questions regarding compartment formation and maintenance. This goal was accomplished by applying a new experimental technique, liquid chromatin Hi-C, to quantitatively assess chromatin interaction stability and dissolution kinetics of 3D chromatin conformations.

Through liquid chromatin Hi-C, we fragmented the human genome to varying sizes and assessed whether compartment formation was perturbed. We estimate that compartmentalization requires chromatin fragments to be at least 11±4 kb in length. Digesting the genome into fragments less than 6 kb disrupts the canonical compartment checkerboard pattern and increases mixing of A and B loci.

Furthermore, digesting chromatin into less than 6 kb fragments leads to a loss of short range intra-chromosomal interactions and an increase in long range intra-chromosomal interactions as well as inter-chromosomal interactions. The loss of short range interactions is not uniform across the genome, but instead dependent on chromatin state. Specifically, we find loci in A compartments lose more short range interactions in comparison with loci in B compartments.

Tracking these changes in interactions throughout time we assign a halflife of interaction stability, t_{1/2}, to every 40 kb locus, representing the time it takes to reach half of the maximal level of interaction loss. Correlating $t_{1/2}$ with various 350 sub-nuclear structures, we find interactions with lamin associated domains to be the most stable, and interactions near nuclear speckles to be the least stable.

Our results are in accordance with polymer models describing strong heterochromatic B-B interactions driving compartmentalization in nuclei (Falk *et al.*, 2019). We find homotypic A-A interactions to be only slightly stronger than A-B interactions after a 4-hour digest of chromatin into fragments smaller than 6 kb. Furthermore, after 16 hours of digestion, where the majority of fragments are smaller than 3.5 kb, the A-A interaction preference is completely abolished. In contrast, the B-B interaction preference remains distinct in some loci even after 16 hours of digestion.

The unstable A-A interactions reported here, contradict models where stable interactions between active regions drive compartmental segregation. Nevertheless, transcription can successfully predict compartment patterning in various genomes and blocking of transcription leads to a reduction in compartment strength (Rowley *et al.*, 2017). When analyzing the effect of gene expression on chromatin stability we discovered expressed regions in B compartments had shorter half-lives than unexpressed B regions. Such a result is in line with active regions being more or less dispensable in maintaining chromosome conformations. However, when analyzing expressed versus non-expressed regions in the highly active A1 subcompartment, we find a small but significant increase in stability (longer t_{1/2}) for expressed regions. This longer t_{1/2}

would imply that regions of expression in open chromatin domains, harbor some 351 mechanism of chromatin interaction stabilization. However, it is unclear whether such small increases in $t_{1/2}$ are a consequence of transcription, Pol II occupancy, epigenetic modifications, or other factors. In reference to this conundrum, super enhancer regions, rich in the active histone mark H3K27ac, are known to cluster together upon cohesin depletion, providing evidence that some active regions can form stable contacts, but are ordinarily obstructed by other 3D chromatin structures (Rao *et al.*, 2017).

In the context of transcription's role in compartmentalization, our results indicate transcription and active regions, in general, have a minor effect on compartment maintenance and the formation of stable homotypic chromatin interactions. This position is supported by both polymer simulations (Falk *et al.*, 2019) and by the presence of compartments seen in mouse sperm which is transcriptionally inert (Jung *et al.*, 2017). However, we cannot definitively rule out a role for transcription in compartmentalization. Liquid chromatin Hi-C investigates the architecture of purified nuclei, with a potentially altered transcriptional state compared to intact cells. Modifications to liquid chromatin Hi-C, for instance, performing the cross-linking step on cells as opposed to extracted nuclei (extracted nuclei were used for easier digestion by restriction enzymes), could help to establish the role of transcription in compartmentalization.

Liquid chromatin Hi-C also provides insight pertaining to the mobility of chromatin positioned within active or inactive compartments. Chromatin diffusion and mobility results largely from incessant Brownian motion which can be modeled as a random walk due to random bombardment of molecules (Marshall et al., 1997; Carmo-Fonseca, Platani and Swedlow, 2002). We note the most extensive mixing between A and B loci only occurs after chromatin is segmented into fragments smaller than 6 kb. This upper limit in length may be informative in terms of chromatin diffusion. Assuming the diffusion of chromatin fragments through the nucleoplasm conforms to a random walk, the conformation of these fragments may affect their own mobility. The persistence length of chromatin (length below which chromatin behaves as a rigid rod) is estimated to be 2.4 kb (Dekker et al., 2002; Dekker, 2008). Hence, the persistence length is on the same order of magnitude as the upper limit fragmentation length (6 kb) required for extensive mixing and compartment disruption. It is possible longer fragments, greater than 6 kb, can form higher order 3D conformations which obstruct and limit fragment mobility.

Our estimations of t_{1/2} suggest heterochromatic loci exhibit slower dissociation kinetics relative to euchromatic loci, with the slowest dissociation kinetics found near lamin associated domains at the nuclear periphery. Modeling chromatin mobility as a random walk, its diffusional motion may be modulated or restricted by sub-nuclear structures such as the nucleolus or nuclear envelope (Carmo-Fonseca, Platani and Swedlow, 2002). We find proximity and interactions with such sub-nuclear structures also modulate t_{1/2}. Such differences 353 in mobility have been documented in yeast with telomeres displaying low mobility, due in part to their tethering with the nuclear periphery (Heun *et al.*, 2001). Furthermore, such telomere tethering restricts mobility of genes linearly proximal to telomeres, which is in agreement with predictions from random walk polymer models (Avşaroğlu *et al.*, 2014). Most relevant to our study, an excised repressed locus in yeast also shows constrained mobility localizing to the nuclear periphery. When the locus is derepressed, the excised locus shows enhanced mobility capable of sampling space throughout the entire nucleoplasm (Gartenberg *et al.*, 2004). Of important note in Gartenberg et al.'s results, excision of loci from the polymeric constraint of full chromosomes led to enhanced mobility in both repressed and derepressed loci. Our work builds on this study, by interrogating the spatial positioning of excised fragments with one another across the entire genome and throughout time.

One of the most difficult challenges faced in analysis of liquid chromatin Hi-C results was decoupling effects from both fragmentation and homotypic compartment attraction on chromatin interaction stability. In an ideal experiment to accurately compare interaction stabilities, one would cut the genome into equal segments at exactly the same time and then measure the dissolution kinetics. However, restriction enzymes have biases for where and when they begin cutting their restriction sites which themselves are not equally distributed across the genome. Such biases convolute comparisons of interaction stability between loci which may be fragmented to different extents at different times. We 354 accounted for such biases via measuring a timecourse of digestion efficiency. However, while we measured residuals of $t_{1/2}$ derived from deviations of expected $t_{1/2}$ based on digestion efficiency, small perturbations in $t_{1/2}$ may be undetected due to over or under correction.

Correlating t_{1/2} with chromatin marks, we show polycomb bound chromatin have short half-lives compared to other condensed heterochromatic regions. In many cases, polycomb bound regions are located in larger A compartment regions. The neighboring chromatin environment of a loci potentially has an effect on its dissolution kinetics. For instance, the shorter t_{1/2} of polycomb bound regions may be attributed, in part, to excision of a condensed piece of chromatin within an open chromatin environment. In such situations, more complex models accounting for neighboring sites may need to be investigated to accurately assign interaction stabilities to distinct loci.

The development of liquid chromatin Hi-C sets a foundation for investigating chromatin stability under a variety of experimental conditions. To test effects of nuclear positioning, liquid chromatin Hi-C could be applied to the inverted nuclei (euchromatin localized to nuclear periphery and heterochromatin localized to nuclear interior) of rod photoreceptors in nocturnal mammals. The static Hi-C maps between inverted and conventional nuclei look similar in terms of compartment formation (Falk *et al.*, 2019). However, dynamic differences related to compartment stability may exist. For example, it would be interesting to test whether A compartments in inverted nuclei, which are relocated to the nuclear periphery, display the same levels of instability seen in conventional nuclei.

In terms of revealing principles of compartment formation, adaptations of liquid chromatin Hi-C could be applied to nuclei or cells inhibited for transcription, or depleted for factors contributing to compartment formation such as HP1 α , lamins, or polycomb-group proteins.

Furthermore, while we assume our results presented here in human lymphoblastoid cells are representative of general chromatin characteristics, it would also be insightful to test whether our results are conserved across varying cell types, tissues, and species. Such experiments could provide insights to developmental processes. For instance, a switch from strong A-A interactions to strong B-B interactions occurs upon differentiation of embryonic stem cells to cortical neurons (Bonev *et al.*, 2017). Comparing t_{1/2} values for A and B loci between embryonic stem cells and cortical neurons could help unravel the mechanisms driving these conformation changes.

Liquid chromatin Hi-C would also be applicable in cell cycle studies. For example, chromatin mobility is shown to be high in G1 cells but markedly slower in S phase (Heun *et al.*, 2001). Also, compartments are shown to disappear upon transitioning from G2 phase to prometaphase (Gibcus *et al.*, 2018). Examining changes in t_{1/2} between these cell cycle stages could expand these results and provide valuable insights into maintenance of chromosome structures throughout 356 the cell cycle.

Chromatin loops and liquid chromatin Hi-C

We show fragmentation of the genome to segments smaller than 6 kb results in a loss of loop structures. Following the loop extrusion model, we propose this may be due to cohesin rings sliding off fragmented chromatin ends near loop anchors destabilizing loop contacts (Sanborn *et al.*, 2015; Fudenberg *et al.*, 2016). We support this hypothesis by showing that after one hour of digestion, loop contacts disappear in Hi-C maps and cohesin is released from DNA in chromatin fractionation assays.

At 10 kb resolution, the mean size of identified K562 loops is 670 kb. Hence, it may be surprising that when the genome is cut by HindIII into 10-25 kb sized fragments, loop contacts are still present. With average loop size being greater than half a megabase, HindIII fragmentation would lead to a fragmentation of internal loop structure in the majority of loops. This implies the anchors of loop loci do not require a continuous chromatin loop body to sustain contacts. Such a conjecture is still in accordance with loop extrusion models, whereby cohesin could be in contact with CTCF loop anchors prior to fragmentation and thus aid in the preservation of anchor contacts.

When fragments are smaller than 6 kb, we propose cohesin may slip off nearby fragmented ends destabilizing loop anchor contacts leading to a loss in cohesin chromatin binding and a disappearance of 3D looping interactions. 6 kb of linear DNA is estimated to be ~ 2,040 nm in length (van Holde, 1989). Considering the relative size of the cohesin complex, with an estimated ring diameter of ~ 40 nm, traversing the necessary fragment length for release is not definitively reconciled (Haering *et al.*, 2002). However, as cohesin is shown to form loops at a rate up to 2.1 kb per second *in vitro*, and the conformation and DNA packing density of fragments at loop anchors is unknown, dissociation of cohesin from fragment ends may be a reasonable hypothesis (Davidson *et al.*, 2019). It should be noted, cohesin mediated loop formation is dependent on cohesin's ATPase activity (Davidson *et al.*, 2019; Kim *et al.*, 2019). As there may be insufficient ATP levels to support active loop extrusion in isolated nuclei, future modifications to liquid chromatin Hi-C, examining the effects of varying ATP concentrations, may support or contradict current models of looping mechanisms.

Visually and computationally, we identify loops in Hi-C maps as dots of significantly high interactions standing out above background (Rao *et al.*, 2014). Considering the increase in A-B interactions in our DpnII digested Hi-C maps, random mixing of fragments less than 6 kb in size may inadvertently wash out looping signal. Of particular relevance, in our aggregation plots of loop loci, looping signal was highly reduced, but not completely abolished after 4 hours of DpnII digestion. As Hi-C maps represent a population average, it is unclear whether remaining loop contacts are driven by a subset of nuclei in a particular

state. Future advancements in single cell studies, could help to resolve the effect 358 digestion has on loop structure per cell.

Further obfuscating conclusions, the manner in which cohesin pseudotopologically or non-topologically interacts with chromatin to form loops is still under investigation, along with the number of cohesin complexes required for loop formation (Davidson *et al.*, 2019; Kim *et al.*, 2019). Future studies defining how cohesin, CTCF, and other factors interact with chromatin to form loops will be critical to understanding how loops are maintained and how these conformations regulate nuclear processes. We propose liquid chromatin Hi-C experiments will be impactful in such studies by providing information pertaining to the dynamics and stability of loop contacts.

3D genome architecture and schizophrenia

In Chapter III, we sought to expand potential schizophrenia risk genes by investigating the 3D interactions occurring at coding and nocoding risk loci. As schizophrenia risk variants are shown to be elevated in neuronally expressed genes (Genovese *et al.*, 2016; Skene *et al.*, 2018), we applied hiPSC technology to generate near pure populations of NPCs, glial cells, and excitatory neurons (Takahashi and Yamanaka, 2006; Brennand *et al.*, 2011; Ho *et al.*, 2016; TCW *et al.*, 2017). To map the 3D architecture of the different brain cell types, we performed Hi-C experiments and investigated resultant 3D contacts in the context of schizophrenia risk (Lieberman-Aiden *et al.*, 2009; Rao *et al.*, 2014).

To identify candidate schizophrenia risk genes, we first set out to map all 359 chromatin loop structures in our brain cell types, presuming some of these loops could link risk variants to candidate risk genes. Loop calling was accomplished through two separate approaches based on either global or local enrichments of interactions (Ay, Bailey and Noble, 2014; Rao *et al.*, 2014; Mifsud *et al.*, 2017). A large percentage of loops were conserved across our cell types and genes overlapping brain-specific loops were enriched for brain specific gene ontology terms. Notably, neurons displayed decreased loop numbers and harbored larger sized loops relative to NPCs and glial cells, suggesting differentiation rewires the 3D genome in a cell type specific manner.

Upon analyzing loops in schizophrenia risk loci, we found an increase in neuron specific and NPC specific loops overlapping risk loci in comparison to glial specific loops. We then tested whether NPC loops, linking risk loci to candidate genes, had regulatory effects via CRISR/Cas9 based editing assays. Both CRISPR activation and deletion assays in NPCs confirmed regulatory roles for some risk loci. In several cases, editing of a risk locus changed the expression of a loop linked gene located hundreds of kilobases away.

We annotated genes overlapping our cell type specific loops as either within a risk locus or connected to a risk locus via a cell type specific looping interaction. This resulted in a set of risk locus and risk locus connect-genes (potentially containing novel risk genes) for each cell type. Integrating these gene sets with independent gene expression and protein expression datasets revealed patterns of significant coregulation across expression samples. We propose such 360 coregulation patterns exemplify regulatory pathways involving risk loci and 3D architectures underlying the genetic etiology of schizophrenia.

Our work in Chapter III was largely inspired by a similar study linking 3D genome architecture to schizophrenia in the human brain (Won *et al.*, 2016). Won and colleagues performed Hi-C on human brain tissue from the germinal zone (composed primarily of NPCs) and the cortical plate (composed primarily of post-mitotic neurons). Significant Hi-C interactions between schizophrenia risk loci and candidate risk genes were identified and validated for regulatory potential via CRISPR/Cas9 editing assays. We employed a similar methodology, but took advantage of hiPSC technology to uncover cell-type specific 3D genome architecture changes as opposed to changes seen in bulk tissue.

Recent work of the PsychENCODE Consortium has revealed the importance of cell type in characterization of variance among transcriptional datasets (Wang *et al.*, 2018). As an example, the gene for dopamine receptor, *DRD3*, displayed expression levels that varied greater in cell type than in bulk tissue measurements across individuals in a population. Such examples highlight that cell-type proportions in individuals largely account for cross-population variation in bulk tissue expression. Further illustrating the importance of cell type, schizophrenia risk genes identified by Hi-C and eQTL data were shown to be highly expressed in single cell profiles of excitatory neurons (Wang *et al.*, 2018). Beyond cell type specific changes in transcription, we show cell type specific

changes in 3D genome architecture also take place in the brain and we provide 361 evidence supporting functional roles for these changes.

Before our work, cell type specific 3D genome architecture changes were also presented in mouse NPCs and cortical neurons either derived from *in vitro* differentiation or from primary tissue (Bonev et al., 2017). In mouse, cell type specific loop formations were associated with increased gene expression. We present similar findings here, whereby genes overlapping loop anchors have significantly higher expression compared to random gene sets. We also show changes in loop formations may be accompanied with changes in gene expression. For example, expression of the loop anchored CUX2 gene is high in neurons possessing the loop, but lower in NPCs where the loop interaction is reduced, and not expressed in glia where the loop interaction is abolished. Our results indicate a large portion of genome architecture is conserved between brain cell types. However, cell type specific changes exist with regulatory and potentially clinical relevance.

Provided sufficient sequencing depth, loops can be visualized on Hi-C maps at 10 kb resolution. To identify loops in our cell types we thus pooled replicate samples to obtain sufficient coverage. Hence, we do not have statistically significant evidence that neurons contain less loop formations than NPCs or glia. However, processing of independent datasets in mouse and human reveal a similar loss of loops upon differentiation from NPCs to neurons (Won et al., 2016; Bonev et al., 2017). In our processing of mouse ES cells,

NPCs, and neurons, we note an increase in loops upon transitioning from ES cells to NPCs (Bonev *et al.*, 2017). This increase in loops is also supported by an independent study where loss of pluripotency is accompanied by a widespread gain of chromatin loops (Pękowska *et al.*, 2018). Such a gain occurs upon transitioning from mouse ES cells to neural stem cells and is proposed to effect the regulation of developmental genes (Pękowska *et al.*, 2018). However, this model of developmental reorganization could be challenged by considering the technical variance in Hi-C quality metrics for these different cell types. Our work supports a hypothesis that loops are gained upon differentiation from ES to NPCs and then lost upon further differentiation from NPCs to neurons.

Along with a loss in neuronal loops we report an increase in neuron loop size relative to NPC and glia. Once again, we find this increase in neuron loop size is also consistent with an increase in loop size upon the NPC to neuron transition when processing independent Hi-C datasets of mouse and human tissue (Won *et al.*, 2016; Bonev *et al.*, 2017). Previous studies have reported knockout of the cohesin release factor WAPL leads to an increase in larger loop structures as WAPL acts to restrict loop extension (Haarhuis *et al.*, 2017). Intriguingly, gene expression of *Wapl* in mouse is decreased in neurons relative to NPCs, potentially contributing to the larger loop structures in neurons (Bonev *et al.*, 2017). While the larger neuronal loops agree with the larger loop structures seen in WAPL knockout cells, the decreased number of loops in neurons

(Haarhuis *et al.*, 2017). As population Hi-C studies are incapable of distinguishing 363 whether multi-dot clusters in Hi-C maps are representative of multiple loops in a single cell or variable sized loops across different cells, further studies refining annotation of loop size and number will be critical toward understanding developmental changes in 3D chromatin organization.

We note in CRISPR/Cas9 editing assays, deletion of loop-connected risk loci leads to an increase in expression in both ASCL1 and EFNB1 genes. Such an increase is suggestive that risk loci carry a repressive as opposed to enhancer function (Traxler et al., 2016). Furthermore, CRISPR/Cas9 activation assays show a reduction in gene expression as opposed to enrichment when activators are targeted to risk loci. It has been reported that there is an inverse correlation between basal level expression and CRISPR-based activated expression across genes (Konermann *et al.*, 2015), and in some cases weak activators can cause repression rather than activation in highly expressed genes (Li et al., 2017). Our RNA-seq analysis estimated EFNB1 expression to be above the 95th percentile in NPC samples. Additionally, targeting of an activator to the promoter of *EFNB1* as opposed to risk loci led to a minimal increase in expression level or no increase dependent on activation system. Conversely, targeting of the ASCL1 promoter caused up to 4-fold changes in expression of ASCL1. Hence, while we can speculate on the mechanisms driving such regulation patterns, further tests would be required to unravel precisely how these risk loci regulate their target genes. It would be particularly insightful to

investigate whether 3D architecture changes in NPCs occur upon CRISPR/Cas9 364 editing, as such changes may also have regulatory effects.

We show significant coregulation occurs between genes annotated as risk locus and risk locus-connect when compared to gene sets with similar genomic distance distributions. However, it remains unclear whether coregulation of gene sets is driven by participation in significant 3D interactions, or whether coregulation is a result of genes residing in and near schizophrenia risk loci. Deeper analyses could examine coregulation patterns of genes in schizophrenia risk loci which are not involved in looping contacts, and conversely examine coregulation of genes that make looping contacts in other regions of the genome. Such analyses could help further delineate the role of 3D genome architecture in the context of schizophrenia.

Our results support a hypothesis where schizophrenia pathology can be explained, in part, by variant loci at enhancer elements misregulating their spatially proximal target genes. We show CRISPR/Cas9 based editing of risk loci can lead to target gene expression changes. However, these genome edits did not identically mimic the schizophrenia mutations. Furthermore, these tests were conducted on genomes derived originally from non-schizophrenic human samples. Future studies performing Hi-C experiments on samples derived from patients with schizophrenia could examine directly whether disease specific 3D architectures exist. For instance, it would be interesting to discover the percentage of our risk locus and risk-locus connect loops that are actually perturbed in schizophrenia genomes harboring the risk variants. Applying hiPSC 365 technology to such studies could also further parse out the role of cell type in schizophrenia etiology.

There is currently a limited number of chromosome conformation datasets for cell types of the human nervous system. Our work represents a primary resource to the scientific community highlighting functional differences in 3D architectures between brain cell types. Similarly, a recent study described celltype-specific 3D contacts associated with cell-type-specific expression within three different iPSC derived human neuronal cell types: excitatory neurons, hippocampal dentate gyrus-like neurons and lower motor neurons (Song *et al.*, 2019). As expression and 3D architecture changes occur even within neuronal subtypes, mapping out cell type specific architectures and regulatory pathways may prove essential for characterizing the pathology of schizophrenia and other psychiatric diseases.

Our study expanded the set of candidate genes conferring schizophrenia risk by integrating 3D interactions with previously annotated risk loci. Beyond GWAS and Hi-C assays, different groups have worked to further expand and refine the map of genetic schizophrenia risk by incorporating data from ChIP-seq, RNA-seq, NOMe-seq, and QTLs (Rhie *et al.*, 2018; Wang *et al.*, 2018). Such studies aid in the construction of enhancer profiles, and highlight their variability across cell types, individuals, and in disease. Future studies incorporating single cell protocols may help to further elucidate the mechanistics behind cell type specific roles in disease. For instance, single cell Hi-C assays could be used to 366 interpret the role of the cell cycle in brain specific 3D architecture relevant to schizophrenia risk (Nagano *et al.*, 2017).

Prediction of TCR-pMHC binding energies and cross-reactive peptides

Chapters IV and V focus on prediction of TCR-pMHC binding energies and prediction of cross-reactive peptides, respectively. In both cases, prediction is based on structural modeling and scoring of TCR-pMHC complex interactions.

In Chapter IV, we developed a publicly accessible and queryable database, ATLAS, to link 3D structures of TCR-pMHC complexes to associated binding energies. Utilizing ATLAS data, we performed multilinear regression analyses to assess performance in prediction of TCR-pMHC binding energy by combinations of energy features calculated from crystal structures. We report a correlation of 0.48 between experimental and predicted binding energies, along with a correlation of 0.63 between experimental changes in binding energies upon mutation and our predicted changes.

In Chapter V, we repurposed deep sequencing data from yeast display of pMHC libraries (Birnbaum *et al.*, 2014) to create a functional benchmark for *in silico* prediction of cross-reactive peptides for specific TCRs. Structural modeling and scoring of peptides in TCR-pMHC complexes resulted in a high concordance between experimental and predicted TCR cross-reactivity profiles. In addition, we show incorporation of structural information to predict cross-reactive peptides improves upon prediction of cross-reactivity based solely on sequence

similarities. Hence, our work highlights the value of including structural information in cross-reactive antigen prediction.

ATLAS expands on other immunological databases, such as IEDB and Antijen, by directly linking TCR-pMHC affinity measurements to complex structure (Toseland *et al.*, 2005; Vita *et al.*, 2015). Incorporating all affinity and structural information currently available, our work sets a baseline for TCR-pMHC binding energy prediction within a machine learning framework. We hypothesize predictions will improve as more affinity and structural data becomes available, along with advances and optimizations to machine learning methods.

Our study in Chapter V expands upon structure based peptide prediction methods such as ITCell, and Lanzarotti and colleague's TCR:p:MHC pipeline (Lanzarotti, Marcatili and Nielsen, 2018; Schneidman-Duhovny *et al.*, 2018). In particular, we integrated deep sequencing data with structural modeling of TCRpMHC complexes. Compared to previous methods, our integrative approach increased the peptide test space by several orders of magnitude. As T cells are required to interrogate a vast space of peptide antigens for efficient immunological protection, our work pushes toward the development of larger scale clinically applicable prediction methods.

A caveat to the work presented in both Chapters IV and V is the unknown level of accuracy in our modeled mutations and peptides, respectively. We were able to determine that the majority of modeled side chain conformations were correct in a small subset of ATLAS entries containing structures of both wildtype and mutant complexes. However, it is not clear what percentage of mutations or 368 peptides are mismodeled among our entire datasets. It also remains to be understood the effect mismodeled side chain conformations have on binding energy or cross-reactive antigen prediction. Mismodeled residues involved in contacts between TCR and pMHC may disrupt essential interactions; however, the degree to which a single mismodeled χ angle affects prediction is unclear. Deeper analyses investigating patterns in side chain conformations at specific regions in the TCR-pMHC interface may aid in the validation of modeled structures and predictive performance.

In regard to cross-reactive peptide prediction, we show structural modeling and scoring is successful in prediction of binding peptides from large sets of nonbinding peptides. This method was successful for the four TCR structures investigated in Chapter V; yet, it remains to be seen how such methods would perform on any given TCR. The large majority of TCRs engage pMHC in a canonical diagonal docking orientation (Rossjohn *et al.*, 2015). However, conformational changes in CDR loops and the antigen recognition surface have been reported across a wide selection of TCR-pMHC complexes (Baker *et al.*, 2012). The studies of Birnbaum et al. suggest some TCRs may also be more cross-reactive than others, capable of tolerating a higher number of amino acid substitutions at specific peptide positions (Birnbaum *et al.*, 2014). Applying our scoring and modeling methods to a variety of TCRs would help to answer how different structure conformations affect cross-reactivity profiles and prediction performance.

We focused our studies on prediction of TCR-pMHC binding. While binding is a prerequisite to TCR signaling, the relationship between affinity and signaling is not clearly defined. TCR-pMHC affinity and subsequent signaling are correlated, but high affinity does not necessarily ensure a strong signaling response (Holler and Kranz, 2003; Stone and Kranz, 2013). Our work, concentrating on the first step in a T cell mediated immune response, inspires further development of models relating TCR-pMHC affinity with downstream signaling. For instance, the kinetic proofreading model hypothesizes TCRs must engage with pMHC with a sufficient dwell time to produce an activation signal, while also disengaging quickly enough to allow the pMHC molecule to interact with other TCRs in the contact zone (Mckeithan, 1995; Bridgeman et al., 2012). Another important note, we only consider the 1:1 interaction between a TCR molecule and pMHC molecule; however, T cell activation results from the clustering of multiple TCRs at the cell surface into supramolecular activation clusters (Monks et al., 1998). It follows that more complex models taking into account energies and kinetics on the macromolecular and supramolecular scale will be essential to accurate prediction of T cell mediated immune responses.

As more TCR-pMHC complexes are solved with matched affinity data, it will be interesting to see how incorporation of new data optimizes weight estimates in scoring functions and affects overall prediction performance. When we applied scoring weights optimized by ATLAS to cross-reactive antigen prediction, results were poorer than using default scoring weights from Rosetta. We assume this is largely due to the fact that ATLAS is trained on structures with peptide bound to MHC. Hence, weights are not trained to account for peptides which are incapable of MHC binding. It follows, future optimization of scoring functions may need to incorporate MHC binding for successful cross-reactive prediction from peptide sequence alone.

In terms of clinical use we propose our methods could be used to advance the fields of TCR design in T cell based therapeutics and TCR testing for crossreactivity. While our prediction performance lacks the accuracy required for cross-reactivity testing in a clinical setting, we provide evidence that computational approaches can be used to discriminate binding peptides from large pools of non-binders. We hypothesize further integration of deep sequencing and structural data will lead to improvements in antigen prediction and pave the way for interrogation of large peptide sets derived from the human proteome and foreign pathogens.

Conclusion

Much is yet to be discovered in terms of how the 3D architecture of the genome regulates cell function. This thesis presented principles governing genome compartmentalization and the formation of chromatin loops. These two structural phenomenons represent essential genomic features across a wide variety of organisms. As such, a deep understanding of the mechanisms driving formation of compartments and loops is critical to our understanding of cellular 371 processes. The results of this thesis contribute to characterizing these structural phenomenons and may provide further insights into disease pathology where such phenomenons are perturbed.

By identifying chromatin loop formations in hiPSC derived NPCs, neurons, and glial cells, this thesis expanded the set of schizophrenia risk genes by linking risk loci with loop linked genes. Our results define cell type specific schizophrenia related "chromosomal connectomes", consisting of schizophrenia risk loci and their 3D proximal genes. Such connectomes harbor genes associated with 3D architecture changes during development and genes critical to neuronal and chromatin remodeling processes. We propose our reported chromosomal connectomes provide a foundation for further studies aimed at characterizing the etiology of schizophrenia.

Lastly, this thesis presents methods for prediction of TCR-pMHC binding energies and cross reactive peptides. We developed a public database, ATLAS, to facilitate modeling and scoring of TCR-pMHC interactions by the scientific community. We used ATLAS to train scoring functions for successful prediction of TCR-pMHC binding energies and changes in binding energy upon mutation. By integrating deep sequencing data with structural methods, we also present successful predictions of TCR cross-reactivity upon interrogation of large peptide sets. We hope our database, methods, and results support future *in silico* approaches to TCR design and cross-reactivity prediction. You want to know how I did it? This is how I did it, Anton. I never saved anything for the swim back.

-Andrew Niccol (1997), GATTACA

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