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**Fundamental boolean network modelling
for genetic regulatory pathways**

A thesis

submitted in partial fulfilment

of the requirements for the Degree of

Doctor of Philosophy

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at

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by

Leshi Chen

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Abstract

Fundamental boolean network modelling for genetic regulatory pathways

by

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A Boolean model is a switch-like behaviour model of which it is easy to ignore any effects at the intermediate levels. Boolean modelling has been applied in many areas, including mammalian cell cycle networks. However, little effort has been put into the consideration of activation, inhibition and protein decay networks to designate the direct roles of a gene or a synthesised protein, as an activator or inhibitor of a target gene.

Hence, we proposed to split the conventional Boolean functions at the subfunction level into activation and inhibition domains, taking into account the effectiveness of protein decay. As a consequence, two novel data-driven Boolean models for genetic regulatory pathways, namely the fundamental Boolean model (FBM) and the temporal fundamental Boolean model (TFBM), have been proposed to draw insights into gene activation, inhibition, and protein decay. The novel Boolean models could reveal significant trajectories in genes and provide a new direction on Boolean modelling research. The proposed novel Boolean models are fine-grained.

A novel network inference methodology named Orchard cube technology has been proposed to infer the related networks, namely fundamental Boolean networks (FBNs) and temporal fundamental Boolean networks (TFBNs) based on FBM and TFBM respectively. As a primary result of this study, an *R* package, called *FBNet*, has been developed based on the proposed methodology and has been used to demonstrate the FBNs and TFBNs for mammalian cell cycle pathways and acute childhood leukaemia pathways respectively.

Our experimental results show that the proposed FBM and TFBM could be used to explicitly reconstruct the internal networks of mammalian cell cycles and acute childhood leukaemia. Especially during the study, we produced the fundamental Boolean networks on the childhood acute lymphoblastic leukaemia gene expression data, which were produced in clinical settings. The pathways may be useful for pharmaceutical agents to identify any side effects when applying GC induced apoptosis on children.

Keywords: boolean modelling, boolean networks, data-driven boolean modelling, fundamental boolean modelling, fundamental boolean networks, temporal fundamental boolean modelling, temporal fundamental boolean networks

Publication and Presentations

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Currently, we are finalising another paper and prepare to publish it:

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Abbreviations

General Terminology Abbreviations

APC	Anaphase-promoting complex
BUC	Bottom-up computation
Cdks	Cyclin-dependent kinases (such as Cdk1, Cdk2)
CKI	Cyclin-dependent kinase inhibitor
CycD	Cdk4/6-Cyclin D complex
CycE	Cdk2/Cyclin E complex
CycA	Cdk2/Cyclin A complex
Cdc20	Cell-division cycle protein 20
Cdh1	Epithelial cadherin (E-cadherin), a classical member of the cadherin superfamily
CycB	Cdk1/Cyclin B complex
DNA	Deoxyribonucleic acid
E2F	A family of transcription factors (TF) that act as transcriptional regulators of G1-S transcription
GRNs	Genetic regulatory networks
GF	Growth factor
NP	Nondeterministic polynomial, a computational complexity class
NP-hard	A class of problems in computational complexity (Nondeterministic Polynomial acceptable problems)
p27	A member of Kip/Cip family, a group of CKIs
<i>R</i>	<i>R</i> - a statistical script programming language
RNA	Ribonucleic acid
Rb	Retinoblastoma protein
SI	Supplementary information
UbcH10	Cancer-related E2 ubiquitin-conjugating enzyme1

Model Terminology Abbreviations

AR	Accurate rate
ER	Error rate
FBM	Fundamental Boolean model
FBN	Fundamental Boolean network
MMR	Mismatched rate
PMR	Perfect matched rate

Boolean Function Notations

&	Logical <i>And</i> connector
	Logical <i>Or</i> connector
!	Logical negation symbol

Boolean Model Notations

\neg	A negation operator that changes a Boolean function from TRUE to FALSE or vice versa
\times, \cap	Logical <i>And</i> operator
$+$	logical <i>Or</i> operator
τ	An incremental variable presenting the number of time steps processed
ϑ	A decay period to reflect the fact that the attenuation or enhancement of the expression of mRNA requires time
σ_i^t	Boolean state of gene σ_i at time t
σ_i^{t+1}	Boolean state of gene σ_i at time t +1.
$l_a(i)$	Total number of fundamental Boolean functions activating the target gene, σ_i .
$l_d(i)$	Total number of fundamental Boolean functions deactivating the target gene, σ_i .
$f_{a_k}^i$	A fundamental Boolean function of the activation
$f_{d_k}^i$	A fundamental Boolean function of inhibition

$P[[x]]$	A Boolean function that takes a uniform distributed random number, μ , and an output of 1 if $\mu < x$ and 0 otherwise.
$V\{x\}$	Logical connective function of <i>Or</i>

Fundamental Boolean Network graphic types

FBNNet_ALL	A general FBN type that contains up-regulatory and down-regulatory pathways. It also refers to FBNNet type 0.
FBNNet_FAA	A forward FBN type that shows the up-regulatory pathways of giving activated genes. It also refers to FBNNet type 1.
FBNNet_FAI	A forward FBN type that shows the down-regulatory pathways of giving activated genes. It also refers to FBNNet type 2.
FBNNet_FIA	A forward FBN type that shows the up-regulatory pathways of giving inhibited genes. It also refers to FBNNet type 3.
FBNNet_FII	A forward FBN type that shows the down-regulatory pathways of giving inhibited genes. It also refers to FBNNet type 4.
FBNNet_BA	A backward FBN type that shows the regulatory pathways that drives a target gene to be activated. It also refers to FBNNet type 5.
FBNNet_BI	A backward FBN type that shows the regulatory pathways that drives a target gene to be inhibited. It also refers to FBNNet type 6.

Legend of All Fundamental Boolean Network Graphs

All fundamental Boolean network types of graphs in this thesis have the same legend as the picture shown below:

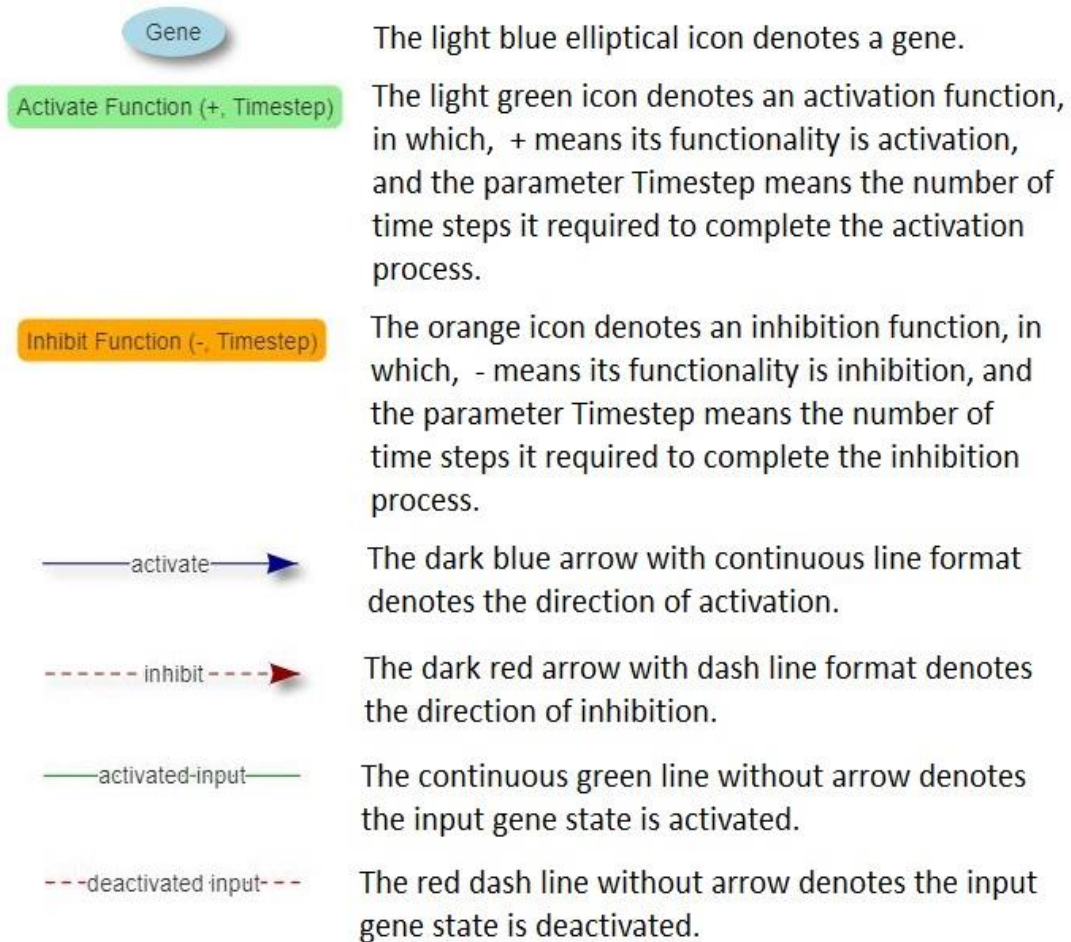


Figure 1 Legend of All Fundamental Boolean Network Graphs

Chapter 1: Introduction

Deoxyribonucleic acid (DNA) is a major component of chromosomes. It has the form of a double helix, consisting of hydrogen bonds, where adenine (A) binds to thymine (T), and cytosine (C) binds to guanine (G) (NIH, 2020a). DNA transports the genetic information that triggers the daily activities of living organisms, such as life, death and reproduction. DNA contains a multitude of functional segments, namely genes. A segment is a gene if it codes for one protein that performs a specific cellular function (NIH, 2020b). Proteins are the fundamental unit of all cellular functions. The process of gene expression is how a gene initially transcribes into messenger ribonucleic acid (mRNA) and then mRNA translates it into the protein (R. Albert, 2004).

The dominant belief of cellular functions is that a cellular function mainly depends on coordinated interactions between genes, RNAs and proteins, to form the foundation of genetic regulatory networks (GRNs) (Scitable, 2020). Within GRNs, activators and inhibitors play an important role in controlling the patterns of gene expression by activating or inhibiting cellular functions (I. Shmulevich, Dougherty, Kim, & Zhang, 2002a). Hence, interconnected knowledge about gene activation and inhibition is essential for uncovering the mechanisms of the apoptosis process with genetic regulatory networks (GRNs); these are crucial for cancer therapy today (I. Shmulevich et al., 2002a).

1.1. Genetic Activators and Inhibitors

As shown in Figure 1-1, an activator is a transcription factor (TF) type of protein that can increase the concentration of a protein through direct binding to the protein or the promoter sites of its genes, to increase its genetic activities. The process is termed gene activation (Saboury, 2009).

In contrast, an inhibitor is a repressor that decreases the concentration of the protein to reduce its genetic activities. The process is named gene inhibition (Saboury, 2009).

Genetic inhibitors can be used as pharmaceutical agents in human and veterinary medicine as well as being used in herbicides and pesticides (Fontes, Ribeiro, & Sillero, 2000; Saboury, 2009).

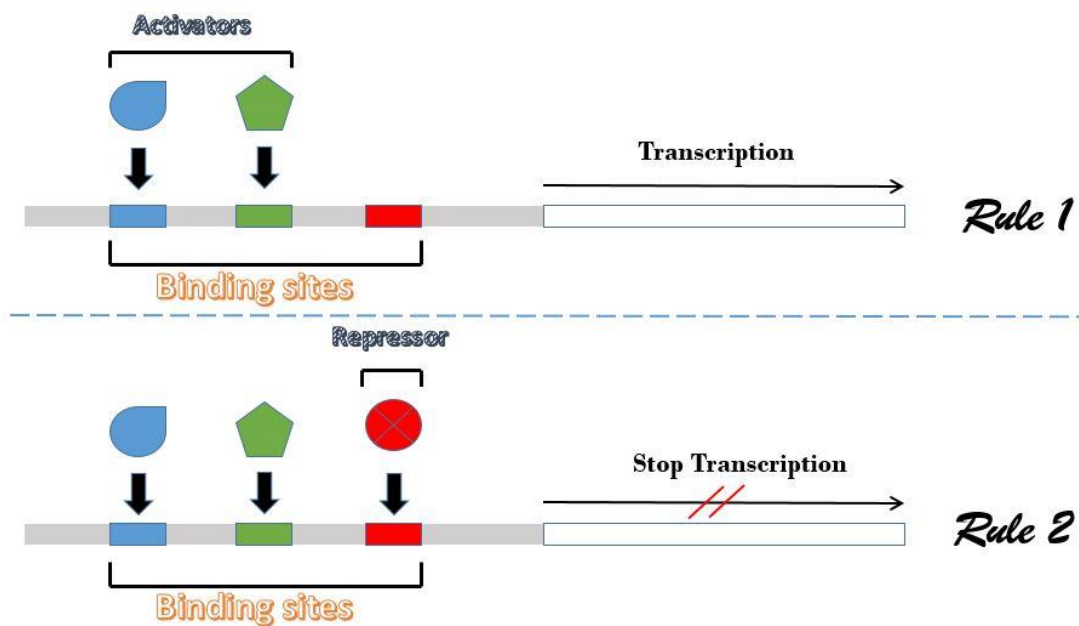


Figure 1-1 Illustration of gene activation and repression

1.2. Genetic Regulatory Networks

The model of GRNs contains nodes and edges where nodes characterise genes, proteins, enzymes or other chemical elements, and edges represent their interactions (Tušek & Kurtanjek, 2012). GRNs are used to describe complex systems consisting of many entities and the relationships among these entities. In systems biology, holistically studying GRNs between the functional status of proteins and gene expression patterns is critical to understanding the nature of cellular functions, such as the complicated gene expression patterns responding to different stressors or stimulators (I. Shmulevich et al., 2002a). Moreover, GRNs analysis provides valuable information for annotating the genome, uncovering biochemical systems in a cell and providing a framework that can derive new ideas to treat diseases, such as cancer (Tušek & Kurtanjek, 2012).

Facilitated by the emergence of biotechnologies, such as Affymetrix™ microarray technology, an enormous amount of high-throughput genetic data are being generated every day. The generated data enable reverse engineering of unknown regulatory networks, such as revealing the relationships among the functional genes in the mammalian cell cycle (Faure, Naldi, Chaouiya, & Thieffry, 2006; Ruz, Goles, Montalva, & Fogel, 2014) and leukaemia (Campbell & Albert, 2014; Hwang & Lee, 2010; Saadatpour, Albert, & Reluga, 2013; Saadatpour et al., 2011; Saez-Rodriguez et al., 2007; Wittmann et al., 2009; Zanudo & Albert, 2013). It is evident from these

examples that analysing massive data sets to understand the coordinated interactions among genes is still a significant challenge.

Different GRN models, such as ordinary differential equations (ODE) (Polyanin & Zaitsev, 2003), neural networks (Ling, Samarasinghe, & Kulasiri, 2013), information theory model (Z. Wang, Huang, Meng, & Tang, 2013), Bayesian networks (S. Y. Kim, Imoto, & Miyano, 2003) and Boolean networks (Akutsu, Miyano, & Kuhara, 1999), have been proposed to reconstruct genetic regulatory networks. However, the current experimental methods are usually insufficient to identify GRNs due to the lack of reproducibility for a large number of genes involved in complex GRNs (Liu, Zhang, Guo, Wei, & Chen, 2016). Even so, of the models, Boolean networks still attract much interest (H. C. Wu, Zhang, & Chan, 2014). Boolean network models do not need information about kinetic parameters (Abou-Jaoude et al., 2016; Barberis, Todd, & van der Zee, 2017; S. Liang, Fuhrman, & Somogyi, 1998; Traynard, Tobalina, Eduati, Calzone, & Saez-Rodriguez, 2017; Tušek & Kurtanjek, 2012; R. S. Wang, Saadatpour, & Albert, 2012) and have explicit regulatory rules when carrying vital information (Yufei, 2009) and are still complex enough to review non-trivial behaviour among the genes, in general (Samuelsson, 2006).

1.3. Boolean Modelling

Boolean modelling was initially presented by S. A. Kauffman (1969) after the discovery of the primary gene regulatory mechanisms in bacteria (Jacob & Monod, 1961). A Boolean model consists of Boolean variables in either of two binary states - *On* (1) or

Off (0) as in digital circuits, denoting gene activation or inhibition, respectively. Each Boolean variable in a GRN represents a gene with its next state determined by a Boolean function.

The fundamental premise of the Boolean network is that the genes exhibit switch-like behaviour during the regulation of their functional states, ensuring the movement of a GRN from one state to another (I. Shmulevich & Dougherty, 2005; I. Shmulevich et al., 2002a; Tušek & Kurtanjek, 2012). Hence, within signal processing theory, Boolean models can be transformed into electronic circuits to facilitate the study of the rich dynamics of Boolean networks (Yufei, 2009).

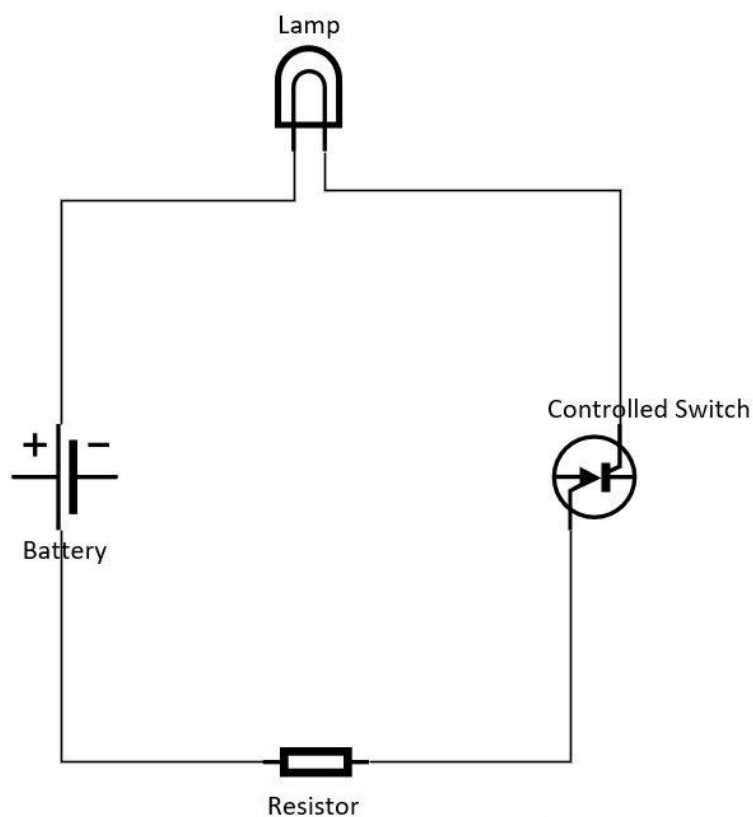


Figure 1-2 A simple series circuit representing a Boolean model

By definition, the conventional Boolean network is wired in the format of a series circuit, as shown in Figure 1-2. When there is a perturbation, the control switch could be *On/Off*, which then turns the lamp *On/Off*. The resistor represents a functional rule that controls the light intensity of the lamp, i.e., it controls the expression level of the lamp. Because a conventional Boolean model only has two states, it can serve only as a series circuit, and the output of the circuit is either expressed or not expressed.

1.4. Fundamental Boolean Model and Networks

At present, the leading emerging biological network inference methods to recognise functional modules are motivated either by the definition of gene regulatory networks or functional networks in which an edge indicates a functional relationship (Lazzarini et al., 2016). Functional relationships are also a subset of entities that describe, explain or predict a biological process or phenotype (Lazzarini et al., 2016). Minimal effort has been made into the construction of activation, inhibition, and protein decay networks that could specify the direct roles of a gene or its synthesised protein as an activator or an inhibitor, as well as be split into the domains of up-regulatory pathways and down-regulatory pathways. This is because the current Boolean networks do not provide an intuitive way to detect single activation or inhibition pathways for a target gene. For example, a Boolean function combines the effect of the current state of its multiple regulators and determines the following gene expression status of gene *CycA* (Hopfensitz, Mussel, Maucher, & Kestler, 2013; Ruz et al., 2014), as shown below:

$$E2F \& !Rb \& !Cdc20 \& !(Cdh1 \& UbcH10) | CycA \& !Rb \& !Cdc20 \& !(Cdh1 \& UbcH10) \rightarrow CycA$$

where E2F, Rb, Cdc20, Cdh1 or UbcH10 are possible genes combined to regulate gene CycA. The Boolean function of CycA combines both activation and inhibition pathways that require further investigation to determine their activation and inhibition components. As shown in this example, the compressed Boolean function cannot intuitively differentiate the characteristics of the gene activators or inhibitors even though the compressed rule can divide it into multiple subfunctions. A compressed Boolean function is a Boolean formula that contains disjunctions with various subfunctions. Hence, a compressed Boolean function can be split into a set of *And* Boolean functions by the disjunction *Or*. For example, a Boolean function $P \& Q | A \& B \& C \& (D | E)$ can be divided as follows:

$$\Upsilon = \{P \& Q, A \& B \& C \& D, A \& B \& C \& E\}$$

where Υ is a set of subfunctions. This weakness can be a significant problem in deciphering larger GRNs with many genes and relationships. Furthermore, by the definition of Boolean models, a single Boolean function always determines the next state of a gene as the behaviour of a series circuit, as illustrated in Figure 1-2. However, this may not be biologically true because a gene may remain activated within a period of decay time, even though activator/activators do not exist (R. Albert, 2004). The conventional Boolean functions that were originally defined by S. A. Kauffman (1969) are hard-wired with the hypothesis of biological determinism. However, genetic regulations are fundamentally stochastic (Raj & van Oudenaarden, 2008; Yufei, 2009).

One reason for this is that the expression of a gene usually incorporates natural random biochemical reactions that are involved in the processes of transcription and translation of mRNAs and proteins (Raj & van Oudenaarden, 2008). Probability Boolean networks (I. Shmulevich & Dougherty, 2005; I. Shmulevich et al., 2002a; I. Shmulevich, Dougherty, & Mang, 2002b) are motivated to address this hard-wired issue. They introduce stochastic mechanisms in which a gene can link with multiple Boolean functions and where each function has a probability indicating the chance that the function can impact the target gene (Raj & van Oudenaarden, 2008). The total probability for all the functions of a gene sum to 1, which means only one function will be selected to determine the next Boolean status of the target gene at a particular time. After that, the probability Boolean network model still contains the major downside of the conventional Boolean model in which a randomly selected function discounts the fact that an unregulated gene may still be in the state of being activated. A target gene can also be regulated competitively and concurrently by another rule, such as the competitive inhibition as illustrated in Fontes et al. (2000). Finally, the current Boolean models that are wired, as shown in Figure 1-2, may not, in reality, be able to elucidate biological phenotypes accurately.

Consequently, to make the conventional Boolean functions clearer, we proposed to split the conventional Boolean functions at the subfunction level, into activation and inhibition domains, taking into account the effectiveness of protein decay. Since gene activation and inhibition are the two most fundamental components of sophisticated cellular machinery, we apply the term “fundamental Boolean functions” to represent

these subfunctions. A fundamental Boolean function could be regarded as a genetic regulatory function or regulatory complex function that determines activation or inhibition activity. For example, the Boolean function of *CycA* can be decomposed into six fundamental Boolean functions: with fundamental Boolean functions *CycA* and *E2F* being TRUE and representing the activation functions, and *Rb*, *Cdc20*, (\neg *E2F* & \neg *CycA*) and (*Cdh1* and *UbcH10*) being TRUE and triggering the inactivation functions. The fundamental Boolean model can serve as both a series and a parallel circuit, as shown in Figure 1-3:

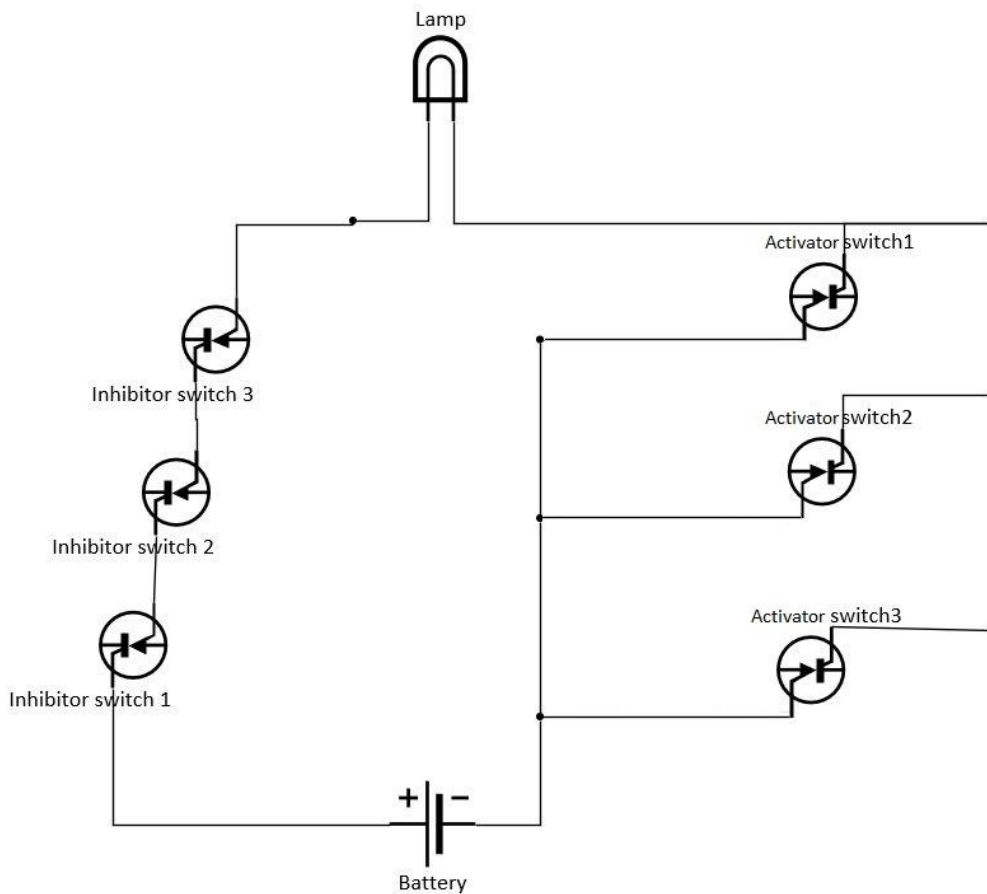


Figure 1-3 Series and parallel circuits representing a fundamental Boolean model

The activator switch represents a gene activation function that can turn on the target gene (the lamp). If any inhibitor exists (one with the inhibitor switched on), the target gene will be turned off immediately regardless of the presence of activators. The model shown in Figure 1-3 is still a Boolean model as the series circuit shown in Figure 1-2 because it has the same Boolean output, i.e., expressed or not expressed. However, it wires the subfunctions of activation as a parallel circuit and the inhibition subfunction as a series circuit. The conceptual theory of fundamental Boolean modelling and networks was published in 2018 (Chen, Kulasiri, & Samarasinghe, 2018), as a part of the research documented in this study.

1.5. Motivation and Objectives

Gene activation and inhibition are the fundamental concepts of the genetic regulatory pathways. However, the roles of the gene activation and inhibition in the traditional Boolean modelling can not be intuitively understood. Besides, a gene may remain activated within a period of decay time when there are no activators present. To address the gaps, we proposed to split the conventional Boolean functions at the subfunction level, into activation, inhibition domains, and take account of the effectiveness of protein decay. Hence, the objectives of this research mainly focus on developing a better understanding of the fundamental mechanisms in genetic regulatory networks about cancer-related treatments, such as leukaemia networks, based on the data extracted from the study by Schmidt et al. (2006).

Genetic regulatory networks are essential for scientists in understanding how the target genes have been regulated. We chose Boolean modelling as the principal method to analyse cancer-related genetic networks. Hence, we ask these questions:

- a) What is the current situation of Boolean modelling for genetic networks?
- b) What is the current treatment of childhood leukaemia? Are there any downsides from the treatment?
- c) Can we apply the Boolean model to gain meaningful insights into microarray time series data, even though the data are short time series data?
- d) Can the novel Boolean model improve understanding of genetic regulatory networks on the induction of leukaemia related apoptosis?
- e) Can we apply the Boolean model to identify down-regulatory pathways as well as up-regulatory pathways?

The literature review in chapter 2 addresses the first two questions. For the remaining questions, we address them by demonstrating the proposed novel fundamental Boolean modelling on cell cycle and acute childhood leukaemia pathways.

1.6. Thesis Structure

This thesis addresses questions within the theory of a novel data-driven network model to explore complexity in the fundamental Boolean model and fundamental Boolean networks (Chen et al., 2018). The novel data-driven Boolean model, namely, the fundamental Boolean model (FBM), aims to encapsulate insights into gene

activation, inhibition and protein decay (Chen et al., 2018). This novel model provides mechanisms to analyse the activation and inhibition pathways intuitively. The new structure of the Boolean network facilitates a data mining method to extract the fundamental Boolean functions from genetic time series data either as a long time series or a short time series. In this research, we aim to illustrate fundamental Boolean modelling that can provide direct insights into the genetic activation and inhibition networks by demonstrating the application of the proposed novel model using cell cycle and leukaemia data. The data for the application of the cell cycle is a synthetic long time series while, for childhood leukaemia, we adopt the real data extracted from Schmidt et al. (2006) research, which is a short time series.

Figure 1-4 presents the thesis structure, which starts from the introduction in chapter 1 and then provides a brief literature review in chapter 2 on current Boolean modelling, microarray data analysis and cancer-related research such as cell cycle and leukaemia. We then illustrate the theory of the novel Boolean models, i.e., the fundamental Boolean model and temporal fundamental Boolean model, in detail in chapter 3. Because the concept of the fundamental Boolean modelling proposed in chapter 3 is novel, there is no existing methodology to infer the related networks. Hence, we proposed a methodology to infer the related networks, discussed in chapter 4. In chapters 5 and 6, we present the applications of the novel Boolean modelling on cell cycle data and leukaemia data, respectively. Finally, chapter 7 concludes the thesis and discuss any interesting points.

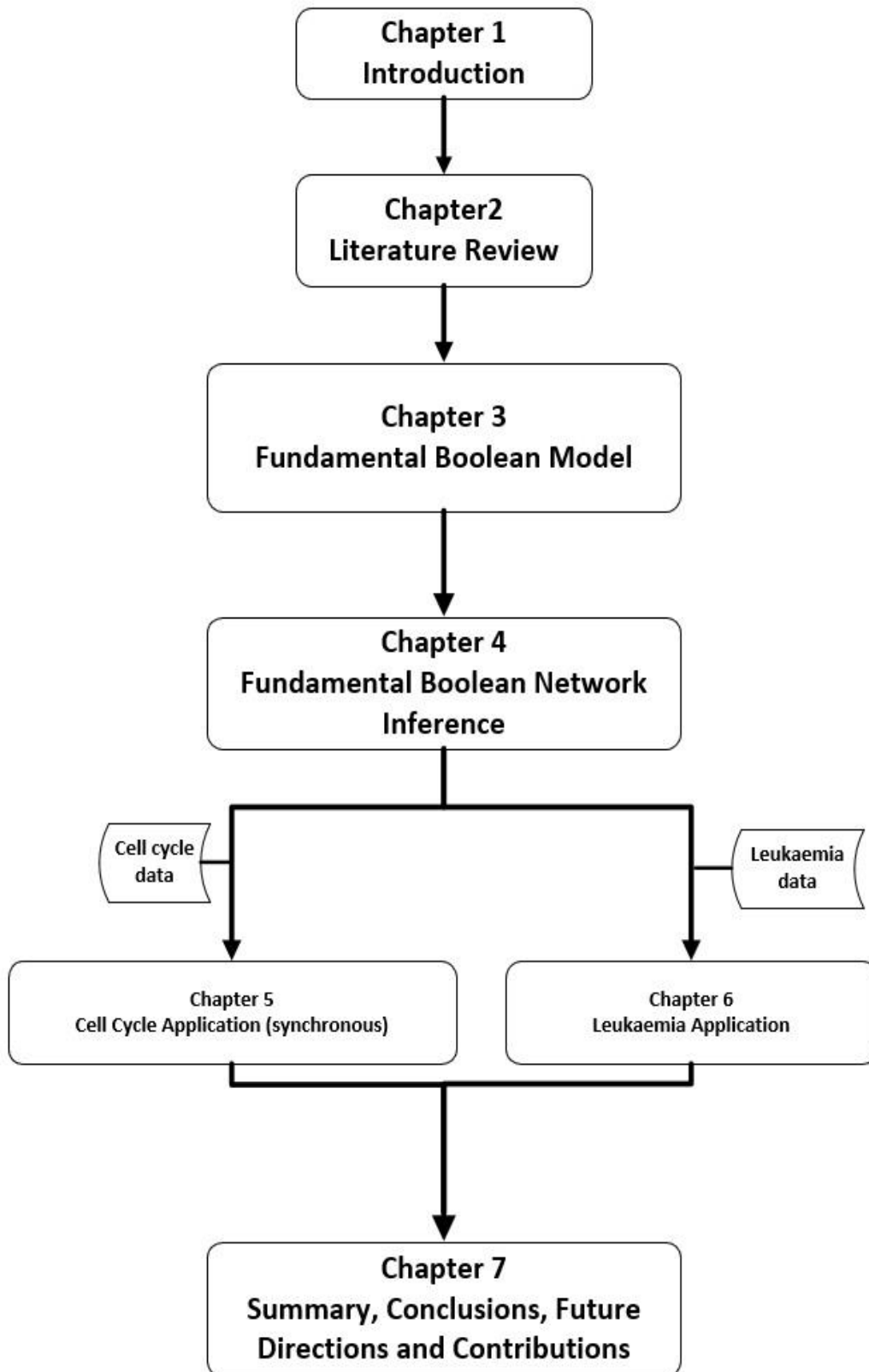


Figure 1-4 Chapter Organisation of the Thesis

Chapter 2: Literature Review

This research is in the domain of systems biology, which is an approach in biomedical research that seeks to understand a broader picture of genetic regulatory systems at the cell level, facilitated by Boolean modelling. Therefore, this chapter provides an overview of the relevant background knowledge about Boolean modelling, microarray expression data and cancer. This research proposed a novel Boolean model, targeting time series data, using clinical data for leukaemia. Hence, in section 2.1, we provide an extensive review of Boolean network modelling, based on the previous research. In section 2.2, we discuss the background knowledge of microarray DNA data and the standard approach to normalising the data. Section 2.3 provides background knowledge on cell cycles and acute childhood leukaemia.

2.1. Boolean Network Modelling

The Boolean model (also called the switching model) is a simple, discrete dynamic model that disregards the effects from any intermediate level (Tušek & Kurtanjek, 2012) and is one of the most interesting in the field of GRNs (P. Li, Zhang, Perkins, Gong, & Deng, 2007; Ouyang, Fang, Shen, Dougherty, & Liu, 2014; I. Shmulevich & Dougherty, 2005; R. S. Wang et al., 2012; Zhiyuan, Simone, Zhaoyang, & Chao, 2014). Boolean network models are predominantly used for qualitatively describing large-scale system dynamics (R. S. Wang et al., 2012). There is evidence that Boolean models can be fruitful in analysing regulatory and signalling networks (Abou-Jaoude et al., 2016). Dating back to 1961, a group of researchers proposed to apply Boolean algebra

to model cellular circuits following the discovery of specific gene regulation mechanisms: the first regulatory circuit in bacteria (Jacob & Monod, 1961). (Sugita, 1963) was then the first to document explicit modelling of bacterial genetic circuits with symbolic logic, firming the term molecular automaton. A few years later, Kauffman (1969) proposed to use a synchronous Boolean update scheme to analyse the dynamic properties of generic Boolean network models, focusing on asymptotic properties. Soon afterwards, Thomas (1973) proposed the use of an asynchronous Boolean update scheme to address the network controlling lysis-lysogeny decisions in the lambda bacteriophage and then, progressively refined the logical formalism with the introduction of multi-value variables, threshold values and Boolean parameters (Thomas, 1978). In the 1990s, the studies of Kauffman and Thomas led to a conclusion that alternative stable states (also named attractors) can be associated with different cell types and the logical state transitions can be associated with gene evolution over time (S. A. Kauffman, 1993; Thomas & D'Ari, 1990). This conclusion laid the foundation for today's Boolean modelling studies in molecular (Abou-Jaoude et al., 2016) such as mitochondrial outer membrane permeabilization (MOMP) regulation (Tokar, Turcan, & Ulicny, 2013). The concept of Boolean modelling has been intensively applied in modelling gene regulation (Bornholdt, 2005, 2008; M. Davidich & S. Bornholdt, 2008; S. A. Kauffman, 1969; Thomas, 1973).

Boolean networks only have two distinct values: *On* and *Off* (1 and 0). According to the original definition of by Kauffman (1969), a Boolean network is defined as graph

$G(V, F)$, where V annotated with a collection of states $X = \{x_i | i = 1, \dots, n\}$, together with a set of Boolean functions F :

$$F = \{f_i | i = 1, \dots, k\}, f_i: \{0, 1\} \rightarrow \{0, 1\} \quad (2.1.1)$$

where each node v_i (the i th item) has been associated with a Boolean function f_i , with inputs into the states of the nodes connected to v_i . The state of the node v_i at time t is expressed as $v_i(t)$. k delegates the last item. Hence, the state of that node at time $t+1$ is given by:

$$v_i(t + 1) = f_i(x_{i1}, x_{i2}, \dots, x_{ik}) \quad (2.1.2)$$

where x_{ij} is the state of the nodes connected to v_i and $k < n$ denotes the total number of genes involved in the Boolean function, f_i .

Boolean models have been categorised into two main types of schemes based on the similarity of timescales for all biological events (Gershenson, 2004; R. S. Wang et al., 2012): synchronous and asynchronous. In synchronous systems (also called deterministic systems) all variables are expected to have similar updating timescales, i.e., one unit at a time, and all components will update simultaneously:

$$\sigma_i^{t+1} = B_i(\sigma_{i_1}^t, \sigma_{i_2}^t, \dots, \sigma_{i_{k_i}}^t) \quad (2.1.3)$$

where $B = \{B_1, B_2, \dots, B_n\}$ is a set of Boolean functions (R. S. Wang et al., 2012) and $\sigma_{i_1}, \sigma_{i_2}, \dots, \sigma_{i_{k_i}}$ is a set of Boolean variables of size k . In contrast, all variables will update non-simultaneously in asynchronous schemes if most of the timescales of the biological actions are different, i.e., each component will update at their specific time unit.

$$\sigma_i^* = B_i(\sigma_{i_1}, \sigma_{i_2}, \dots, \sigma_{i_{k_i}}) \quad (2.1.4)$$

where the asterisk denotes the variable (σ_i^*) ($i = 1, 2, \dots, n$) is derived from the set of inputs $\sigma_{i_1}, \sigma_{i_2}, \dots, \sigma_{i_{k_i}}$. The inputs can be the gene states from the current or previous time point (R. S. Wang et al., 2012).

Both schemes can map to a directed graph $G(V, E)$, where the node-set $V = \{v_1, v_2, \dots, v_n\}$ corresponds to Boolean variables with size n , and the edge set, E , matches the Boolean functions in the model. Each edge has a direction with a sign indicating how the input node affects the target node (positively or negatively) (R. S. Wang et al., 2012). A vector $(\sigma_{1(t)}, \sigma_{2(t)}, \dots, \sigma_{i(t)}, \dots, \sigma_{n(t)})$ is referred to as the state of the system at time t (R. S. Wang et al., 2012). The i th vector variable ($\sigma_{i(t)}$) denotes the state of the node v_i at time t . Each node can be linked to a gene, a protein or a metabolite in order to elucidate the dynamics of biological systems using Boolean networks.

Synchronous Boolean networks embed the hypothesis that a gene state at a given time point is determined by the state of a subset of genes at an earlier time point. A downside of the synchronous dynamics is that it does not allow the temporal separation of multiple regulatory activity changes (Faure et al., 2006). Another drawback is that the synchronous Boolean network model cannot measure differences in the speed of signal propagation in the context of biological systems and that results in differences in the rates of signal propagation between cells (Hwang & Lee, 2010).

The most popular synchronous Boolean models are random Boolean networks (Drossel, 2009; Gershenson, 2002, 2004; S. Kauffman, Peterson, Samuelsson, & Troein, 2003; Lynch, 2007; Mc, 2002; Samuelsson, 2006; I. Shmulevich, Lahdesmaki, Dougherty, Astola, & Zhang, 2003), temporal Boolean networks (Silvescu & Honavar, 2001), probabilistic Boolean networks (Dougherty & Shmulevich, 2003; Harri, Harri, Ilya, & Ilya, 2006; I. Shmulevich & Dougherty, 2005; I. Shmulevich et al., 2002a; I. Shmulevich et al., 2002b; I. Shmulevich, Dougherty, & Zhang, 2002a, 2002b; I. Shmulevich, Gluhovsky, Hashimoto, Dougherty, & Zhang, 2003), threshold Boolean networks (Higa, Andrade, & Hashimoto, 2013), stochastic Boolean networks (J. Liang & Han, 2012; Z. Wang et al., 2008), Petri Boolean models (Berestovsky, Zhou, Nagrath, & Nakhleh, 2013; Chaouiya, 2007) and switching Boolean networks (Hwang & Lee, 2010).

In the model of the random Boolean network (RBN), the random Boolean functions control the state of each node by randomly selecting Boolean functions from the 2^{2^k} possible K input. The randomly selected Boolean functions are then kept fixed afterwards. After studying the dynamics of these RBNs, Kauffman (1969) claimed that the existence of a phase transition in an RBN of size N depends on the value of parameter K (R. Albert, 2004; S. Kauffman et al., 2003; S. A. Kauffman, 1969). If K is more than 2, there are approximately N/e (e is the base of the natural logarithm) possible cycles of scales that exponentially lengthen to N . If K is equal to 2, both the number and duration of the limiting cycles are approximately the square root of the network size N , i.e., \sqrt{N} (R. Albert, 2004).

To reveal how the expression level of other genes influences the expression of a gene at some state in a process during the stage of the process preceding it in multiple time steps. Silvescu and Honavart et al. (2001) introduced temporal Boolean networks (TBNs), which extended the works of (Akutsu et al., 1999; S. Liang et al., 1998) on the inference of Boolean networks ($BN(n, k)$, where n is the total number of nodes under consideration and k is the number of network connections based on n) to handle multiple time steps. The model, denoted as $TBN(n, k, T)$ where $k \ll n$, enables the current state of each gene to be affected by a Boolean function of the states of most k genes at times in $\{t \dots t-(T-1)\}$ (Silvescu & Honavar, 2001). The temporal Boolean networks fundamentally renovated the Boolean networks from a Markov(1) to a Markov(T) model, and T referred to the length of the time window during which a gene can influence another gene (Silvescu & Honavar, 2001). Markov model, particularly the Markov chain model, is a stochastic model used to model the state of a system with a random variable that updates through time, and the random variable depends only on the distribution of a previous state (Gagniuc, 2017). Silvescu and Honavar (2001) demonstrated that the temporal Boolean networks could be inferred from time series data. However, the primary obstruction to applying temporal Boolean networks to a real system is the lack of sufficiently long time series data.

Threshold Boolean networks have a Boolean function for each gene so that the output value depends only on the sum of its input signals (Hwang & Lee, 2010). The limitation of a threshold Boolean network is that it relies entirely on the completion of network

information. If the network is incomplete, this results in a modelling anomaly (Hwang & Lee, 2010).

The asynchronous update only allows one gene or component to be updated at a random time, resulting in a nondeterministic illustration of the dynamics (Siebert, 2011), which are very complex and encompass many incompatible or unrealistic pathways (Faure et al., 2006). Variations in asynchronous Boolean models, such as non-deterministic asynchronous Boolean networks and deterministic asynchronous Boolean networks (Gershenson, 2004) have been proposed to address these different issues.

Hybrid models that combine synchronous and asynchronous transitions can demonstrate the flexibility of the combination of different updating assumptions (Faure et al., 2006). Berestovsky et al. (2013) combined Petri nets, which have been developed as a promising tool to analyse from purely qualitative to more complex quantitative models (Chaouiya, 2007), and Boolean networks to model integrated cellular networks, i.e., the integrated hybrid model (IHM). The hybrid model has already been demonstrated on three main cellular biochemical processes: signal transduction, transcription regulation and metabolism (Berestovsky et al., 2013; Chaouiya, 2007).

Some conventional approaches have been proposed to infer Boolean functions: *Symbolic* approaches (Batt, de Jong, Page, & Geiselman, 2008; Langmead & Jha, 2008; Yoon, 2005), the *Best-Fit Extension* algorithm (Berestovsky & Nakhleh, 2013),

geneFAtt (Zheng et al., 2013), *Chi-square test* (H. Kim, Lee, & Park, 2007) such as *Pearson's Chi-square test*, and the reverse engineering algorithm (REVEAL) (S. Liang et al., 1998) which was later extended to allow for multiple discrete states as well, to let the current state depend as a window of the previous states (Hecker, Lambeck, Toepfer, van Someren, & Guthke, 2009). Mussel, Hopfensitz et al. (2010) proposed a tool named the *BoolNet* R package for generating, reconstructing and analysing Boolean networks from time series using the Best-Fit Extension (Ilya Shmulevich, Yli-Harja, & Astola, 2002) and REVEAL algorithms (S. Liang et al., 1998). These tools provide methods to identify the attractors of synchronous, asynchronous and probabilistic Boolean networks. This package has been demonstrated as a tutorial by Hopfensitz et al. (2013). The advantage of this package over other existing tools, such as GINsim (Gonzalez, Naldi, Sanchez, Thieffry, & Chaouiya, 2006), BooleanNet (I. Albert, Thakar, Li, Zhang, & Albert, 2008) and BN/PBN toolbox in Matlab, was that it supported all three network types, i.e., the synchronous, asynchronous and probabilistic Boolean networks.

With the facilities of the Boolean modelling tools that have emerged, Boolean networks have been successfully applied to yeast (M. I. Davidich & S. Bornholdt, 2008; S. Kauffman et al., 2003; Kazemzadeh, Cvijovic, & Petranovic, 2012; F. Li, Long, Lu, Ouyang, & Tang, 2004), flower morphogenesis of wall cress, *Arabidopsis thaliana* (Espinosa-Soto, Padilla-Longoria, & Alvarez-Buylla, 2004), *Drosophila melanogaster* (R. Albert & Othmer, 2003; Ghysen & Thomas, 2003; Sanchez & Thieffry, 2001), mitochondrial outer membrane permeabilization (MOMP) regulation (Tokar et al.,

2013), the mammalian cell cycle (Faure et al., 2006; Ruz et al., 2014), light- and carbon-signalling pathways (Thum, Shasha, Lejay, & Coruzzi, 2003), hepatocyte signal networks (Schlatter et al., 2012), apoptosis networks (Kazemzadeh et al., 2012; Mai & Liu, 2009; Schlatter et al., 2009; Schleich & Lavrik, 2013), NF-kappaB and IL-6 mediated by miRNA (Xue, Xia, & Wenzhong, 2013) and leukaemia (Campbell & Albert, 2014; Hwang & Lee, 2010; Saadatpour et al., 2013; Saadatpour et al., 2011; Saez-Rodriguez et al., 2007; Wittmann et al., 2009; Zanudo & Albert, 2013).

In these successful applications, M. I. Davidich and S. Bornholdt (2008) predicted the biological cell cycle sequence of fission yeast using a Boolean model in a parameter-insensitive way, with 47 kinetic constants that were necessary for the ordinary differential equations (ODE) approach were eliminated. Faure et al. (2006) studied the dynamics of the mammalian cell cycle Boolean model, with synchronous, asynchronous or hybrid treatment of the concurrent transitions. Moreover, the signal transduction network of abscisic acid has been proven to induce stomatal closure (S. Li, Assmann, & Albert, 2006).

2.2. Microarray DNA Data Analysis

Many biologists use Microarray technology to monitor genome-wide expression levels of genes in a given organism (Babu, 2004). A microarray is typically referred to a glass slide that contains thousands of spots, and each spot may contain a few million copies of identical DNA molecules fixed in an orderly manner that uniquely corresponds to a gene (Babu, 2004). One of the most popular applications of microarray is to compare the expression of a set of genes from a cell maintained in a particular condition to the same set of genes from a reference cell maintained under normal conditions (Babu, 2004).

The DNA microarray technology, including the design of experiments to extract mRNA samples, has been applied to analyse human cancers, such as breast, prostate and leukaemia (Russo, Zegar, & Giordano, 2003). mRNA samples are hybridised using a gene chip, which contained a strand of all genes in the human genome, such as HG-U133 Plus 2. Raw gene data are extracted from image analysis by measuring the level of hybridisation on the chip. Complementary DNA (cDNA) microarray and oligonucleotide chips are the two approaches for manufacturing the microarrays. cDNA arrays are fabricated by robotic spotting on glass slides, and oligonucleotide arrays are developed by photolithographic chemistry and light-directed chemical synthesis on small glass plates (Taub, DeLeo, & Thompson, 1983).

Gene expression matrices are the product of microarray data analysis, where rows denote genes and columns denote samples. Microarray data analysis can be

conducted by generating cell intensity (.CEL) files using the Affymetrix GeneChip Operating System Software (GCOS). The generated cell intensity files can then be converted into gene expression matrices using the R package, *affy* (Gautier, Cope, Bolstad, & Irizarry, 2004).

Emergent microarray technology using Affymetrix® GeneChip arrays paved a significant way to develop a better understanding of cancer prediction and diagnosis, and a better method to discover target drugs for the treatment of malignant tumours. Affymetrix® GeneChip arrays (<http://www.affymetrix.com/index.affx>) are high-density oligonucleotide gene expression arrays that been mainly used in biomedical research. Oligonucleotide expression array technology is referred to a system that uses oligonucleotides with a length of 25 base pairs (probe genes), in which each gene will be represented by 16–20 pairs of oligonucleotides (probe sets) (Irizarry et al., 2003). Oligonucleotide gene expression arrays treat each gene as 11-20 different probe pairs called a 'probe set'. Each probe consists of 25 nucleotide bases, and each probe set has two components: perfect match (PM), which refers to the specific sequence, and mismatch (MM), which is used to measure noise caused by non-specific binding. The expression level of a gene (*i*) is then calculated by the average of the difference between PM and MM for all probe pairs of the gene, as shown below:

$$E_i = \sum_{j=1}^p (PM_j - MM_j) \quad (2.2.1)$$

where *i* represents the gene *i*, *p* is the total number of the probe pairs of the gene *i*. *j* is an incremental value and presents as one probe pair.

Commonly, an experiment may involve extracting gene expression matrices at different time points with the same set of samples. If we reorganise the extracted gene expression matrices based on the order of time points, we will yield time series expression data. Time series expression data typically contain a series of m microarray expression measurements in the order of time points, involving n genes. The gene expression data is used to represent an $m \times n$ table (\check{T}) where m served as columns and n as rows (Silvescu & Honavar, 2001). There might be multiple samples, and each sample contains the same number of m and n but with different measurements. Combining all samples, it becomes a three-dimensional sample data space, \check{S} . Hence, the entry, e_{ij}^s , in row t and column i of the table \check{T}_s denotes the expression level of gene i in the j th measurement of the sample, s . Most data analysis is undertaken with a straightforward table, \check{T}_s (matrix) such as using cell cycle analyses. However, data analysis on a three-dimensional sample data space (\check{S}_s) might become more popular (Silvescu & Honavar, 2001).

Meaningful temporal gene expression patterns can be extracted from the time series data and genes can be associated with each pattern (gene groups). The relationships of gene groups can be modelled and depicted by GRN methods, such as Boolean network modelling. Based on the availability of time points that can be extracted from experiments, time series data can be categorised into two main groups: short time series with the number of time points fewer than eight, and long-time series with the number of time points more than eight (Ernst & Bar-Joseph, 2006; Ernst, Nau, & Bar-Joseph, 2005).

Because the expense involved in acquiring genetic data are not economical, about 80% of published experimental data are short time series (Chaiboonchoe, 2010; Ernst et al., 2005). Besides, the period of a patient's treatment is usually either too short or fatal (Z. Wang et al., 2008). Even if the expense is dropped, short time series experiments are still important because obtaining large quantities of biological material is prohibitive (Ernst et al., 2005).

Traditional algorithms do not perform well with short time series data due to the lack of the required length of the time series, i.e., the required time points are too short to fit these algorithms (Tchagang et al., 2012; Z. Wang et al., 2008). The construction and validation of traditional models are also complicated (Siebert, 2011). Short time series data typically contain an enormous number of genes but only a few observations. Knowledge of the kinetic parameters and mechanistic details are unable to be inferred consistently from short time series data because the data are very noisy and contain various lengths of temporal observational gaps. Valuable information may be missing between the sparse observation gaps and; hence, this may lead to incorrect conclusions.

How to choose the most suitable and dependable method to address a particular biological question from a specific dataset is a significant research question. One criterion is the capability to detect differentially expressed genes in terms of precision (specificity/variance) and accuracy (sensitivity/bias) (Irizarry, Wu, & Jaffee, 2006).

Differentially expressed genes are highly dependent on the normalisation methods that alter how the correction structure from the data impact on the accuracy of the inference of cellular networks. Microarray normalisation, typically, involves three main steps, and there is a background correction that removes background noise from the signal intensities; data normalisation that eliminates non-biological variability between arrays and makes distributions across arrays; and summarisation, that provides a single expression measure to each probe set in the array. The most common normalisation methods are MAS5.0, Robust Multichip Average (RMA) and GeneChip RMA (GCRMA). MAS5.0 applies MM probes to adjust the PM probes for probe-specific non-specific binding for background correction. MAS5.0 uses a baseline array and scales all the other arrays to have the same mean intensity for normalisation and uses Tukey's biweight function for summarisation (Affymetrix, 2002). RMA (Irizarry et al., 2003) applies a global correction, quantile normalisation and a median polish summarisation. The GCRMA (Z. Wu, Irizarry, Gentleman, Martinez-Murillo, & Spencer, 2004) is a method to convert background adjusted probe intensities to expression measures using the same normalisation and summarisation approaches as RMA and is bias-corrected.

Gene expression microarray data, which are quantitative and semi-quantitative for cell status on a particular condition and time, are the most current data for biologists to use (Bansal, Belcastro, Ambesi-Impiombato, & di Bernardo, 2007). A new discipline of introducing computer technologies into biology using gene expression microarray data has been developed in recent years (Tušek & Kurtanjek, 2012). It is

now referred to as the foundation of systems biology or computational biology (Tušek & Kurtanjek, 2012). The progress in systems biology has led to the development of complete genetic regulatory networks of genomes of many organisms (Tušek & Kurtanjek, 2012).

The reconstruction of the dynamics, represented by time and the discrete state transition systems to gain insights into the functioning of cell systems, is attracting more and more attention (Ay & Arnosti, 2011; Hood, 2013; Lee & Tzou, 2009; Y. Wang, Zhang, & Chen, 2011). These dynamics can be used to simulate the perturbations of new drugs *in silico* to reduce the potential risk of applying drugs to human beings. For example, Chaiboonchoe (2010) identified new glucocorticoid-regulated genes through the inferred GC-regulation networks. The newly identified genes may help pharmacists to develop new drugs that contain fewer side effects when applying chemotherapy.

2.3. Cancer

Molecular species and their cellular circuitries comprise sophisticated cellular machinery, while many parts of the machinery are still mysterious. For example, the secret of how the modification of one gene affects other genes at the expression level is still unknown (Bruce et al., 2012). Besides, whether a particular heritable aberration is due to an alteration in the cell's DNA sequence or a constant change in the pattern of gene expression without modification in the DNA sequence, is still an area of ongoing dispute (Bruce et al., 2012). Furthermore, it now appears that the abnormal

epigenetic silencing of genes is no less critical than mutations in DNA sequences for the development of cancers (Bruce et al., 2012).

Cancer is a significant kind of, often, fatal genetic disease and the primary cause is commonly due to aberrant gene regulation, such as mutations in single somatic cells.

The aberrant gene regulations interrupt the standard control of proliferation that continually unregulates the proliferation of cancer cells that invade adjacent healthy tissues and organs from other sites (Bruce et al., 2012; Cooper, 2000; Fumia & Martins,

2013; Hanahan & Weinberg, 2000, 2011; Hodgson & Maher, 1999; Hornberg, Bruggeman, Westerhoff, & Lankelma, 2006; Martinez, Taylor Parker, Fultz, Ignatenko, & Gerner, 2003; Siegel, Ma, Zou, & Jemal, 2014; Weinberg, 2007). In other words, the unexpected changes in gene regulation cause healthy cells to undertake abnormal

functions and, as a consequence, develop cancers (Frederick, Nolan, Scott, & Slade, 2003). These changes are commonly attributed to inherited genetic mutations or are

induced by chronic exposure to carcinogenic environmental factors, such as UV light, X-rays, chemicals, tobacco products and viruses (Cooper, 2000; Frederick et al., 2003).

The abnormal functions, which are usually inhibited in healthy cells, are in cancer cells. The abnormal functions are usually caused by the nonfunctioning protein-encoding

genes that regulate cell division due to genetic mutations. If the cancer cells persist in their original site, they are considered benign, such as a common skin wart that

remains confined to its original location, neither invading the surrounding normal tissue nor spreading to a distant body site (Cooper, 2000; Frederick et al., 2003). In

contrast, the malignant cells can metastasise themselves into a different location in

the body and may form new tumours by invading the surrounding healthy tissue via the circulatory or lymphatic systems (Cooper, 2000; Frederick et al., 2003).

The number of genes that are associated with cancer in the human genome is approximately 35,000 and alternations in these genes are typically associated with a variety of cancers (Frederick et al., 2003). These malfunctioning genes are usually divided into three groups: proto-oncogenes, tumour suppressors and DNA repair genes. The genes of proto-oncogenes produce protein products to either enhance cell division or repress cell death, and the mutated forms of these genes are named oncogenes; in contrast, the genes of tumour suppressors develop protein products repressing cell growth or causing cell apoptosis. DNA repair genes make protein products to prevent carcinomatous mutations (Frederick et al., 2003).

The accumulated abnormalities in multiple cell regulatory systems result in the generalised loss of growth control as affected by cancer cells (Cooper, 2000). Growth control is a domain in the cell cycle.

2.3.1. Cell Cycle

The cancer cells *in vivo* have different proliferating behaviour in cell cultures from healthy cells. Healthy cells show density-dependent inhibition of cell proliferation. The availability of growth factors in the culture medium (in the form of serum) determines a finite cell density that limits the healthy cells' proliferation (Cooper, 2000). When a healthy cell reaches its finite cell density, the cell then ceases proliferating and

become quiescent and are arrested in the G0 stage of the cell cycle, where the G0 stage (also named as resting phase) designates a cellular state outside of the replicative cell cycle (Cooper, 2000). In contrast, tumor cells are not sensitive to density-dependent inhibition and their proliferation is uncontrollable *in vivo*. As a consequence, tumour cells usually remain growing to high cell densities when in culture (Cooper, 2000).

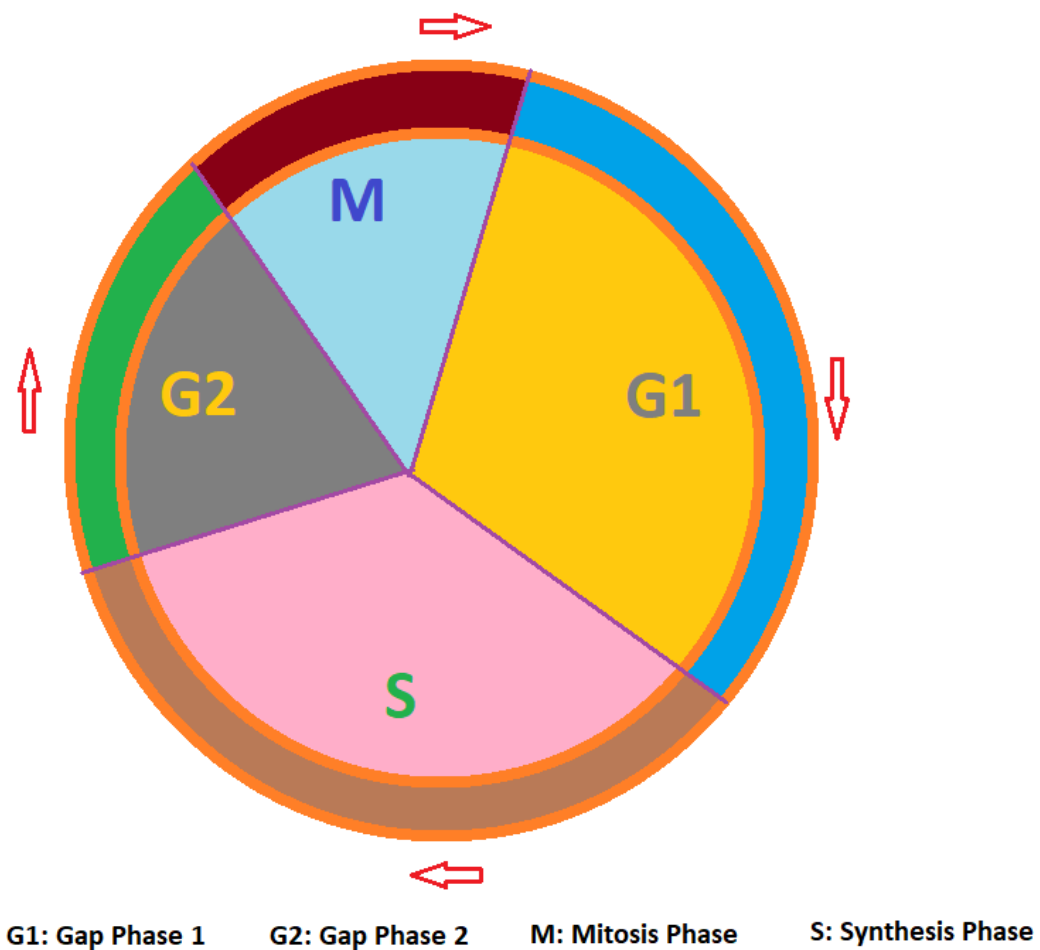


Figure 2-1 Cell Cycle.

Cells produce and split in an orderly fashion; namely, by the cell cycle. As shown in Figure 2-1, the cell cycle occurs in four stages: G1, G2, S (synthesis) and M (mitosis) phases. G1 and G2 are the two-gap stages, and in the two stages, no cell division happens but is it actively metabolising (Frederick et al., 2003). In the S phase, the chromosomes duplicate due to DNA replication. In the M phase, the chromosomes separate in the nucleus and the division of the cytoplasm (cytokinesis) occurs, and in the M phase itself contains four different sub-phases (prophase, metaphase, anaphase, and telophase). There are two checkpoints at the end of G1 and G2 that prevent the cell from entering the S or M phases of the cycle, respectively. Cells that are not in the process of dividing are in the G0 stage (Frederick et al., 2003).

2.3.2. Leukaemia

Blood cells come from the bone marrow, which is a kind of spongy material in found bones, as outlined in Figure 2-2. White blood cells are a type of blood cell and act as an immune system to defend against certain types of infection in different ways (Maton et al., 1997). There are two types of white blood cells based on where they developed from lymphocytes and granulocytes (LaFleur-Brooks, 2008). Lymphocytic white blood cells develop from lymphoid stem cells, and granulocytes develop from myeloid stem cells (LaFleur-Brooks, 2008). The myeloid cells usually fight against widespread infection, whereas lymphoid cells are more specific for certain types of infection (Stegelmeier et al., 2019).

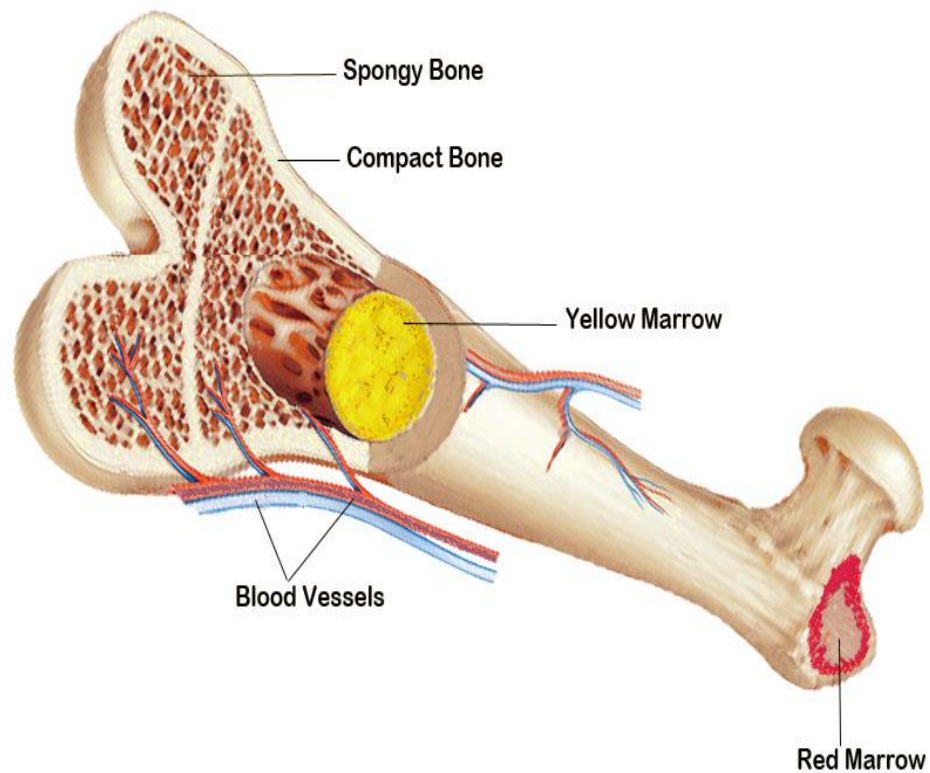


Figure 2-2 Inner details of bone structure, which is modified from (FBRadmin, 2013).

Leukaemia is white blood cell-related disease driven by cumulative mutations in immature white blood cells from the bone marrow that reduce the number of red cells, healthy white cells and platelets (Banjar, Adelson, Brown, & Chaudhri, 2017; Hanahan & Weinberg, 2000; Hornberg et al., 2006; Martinez et al., 2003; Weinberg, 2007). The causes of leukaemia are arguably due to radiation, chemicals/genetic problems, and smoke (Banjar et al., 2017; Hanahan & Weinberg, 2000; Hornberg et al., 2006; Martinez et al., 2003; Weinberg, 2007).

As a consequence, the redundant and unhealthy white blood cells enter the bloodstream and accumulate in organs, for example, the liver or spleen that may

cause many problems (Banjar et al., 2017). Figure 2-3 outlines a sample of a healthy vessel (down right) and a leukaemia vessel (up left). The leukaemia vessel accumulates too many abnormal lymphocytic white cells and hence, contains fewer erythrocytes than healthy ones.

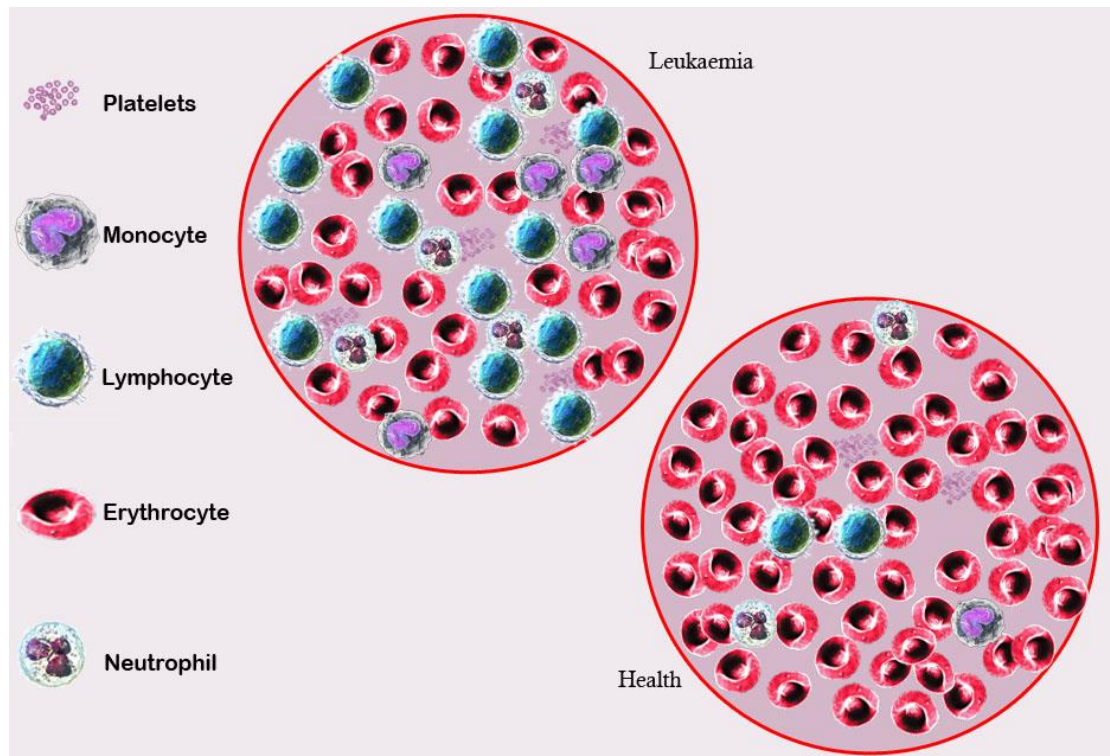


Figure 2-3 Healthy vessels and leukemic vessels

Nowell and Hungerford (Hodgson & Maher, 1999) identified the first consistent Philadelphia chromosome abnormality in chronic myeloid leukaemia in 1960. Since then, leukaemia has been categorised into two main groups, childhood and adult. Childhood leukaemia can be divided further into two types: acute or chronic. Most childhood leukaemia is acute. In this study, we focus on acute childhood leukaemia. Acute childhood leukaemia can be divided into two groups: acute lymphoblastic leukaemia (ALL) if lymphocytic cells were affected, and acute myelogenous leukaemia

(AML) if granulocytic cells were affected. Both groups have two subgroups of ALL: T-lineage (T-ALL) and B-lineage (B-ALL) (Tissing, Meijerink, den Boer, & Pieters, 2003).

Currently, there are three phases to treat the ALL of children: induction, consolidation/intensification, and maintenance (St. Jude Children's Research Hospital, 2019). The first phase is trying to kill the leukaemic cells in the blood and bone marrow, and the second phase is getting rid of any remaining cells that could cause the leukaemia to return. The last phase is to destroy any cancer cells that might have escaped from the previous two phases (St. Jude Children's Research Hospital, 2019).

Four types of treatments may be applied to cure childhood ALL during the three steps mentioned above. There is chemotherapy (“chemo”), stem cell transplants, radiation therapy, and targeted therapy (St. Jude Children's Research Hospital, 2019).

Chemotherapy, which can be applied by mouth or injected into the bloodstream (St. Jude Children's Research Hospital, 2019), applies strong medicine to introduce apoptosis in cancer cells and is the most common treatment for children with ALL. A stem cell transplant can replace the damaged blood-forming cells that caused by chemotherapy and/or radiation therapy, in the bone marrow with the new blood cells coming from a donor’s blood or bone marrow (bone marrow transplants) (St. Jude Children's Research Hospital, 2019). Radiation therapy applies powerful X-rays or other types of radiation to introduce apoptosis in cancer cells or block them from growing (St. Jude Children's Research Hospital, 2019). Targeted therapy focuses on specific cancer cells and tries to avoid harming healthy cells and uses medicines or other treatments (St. Jude Children's Research Hospital, 2019).

Apoptosis is a programmed cell death (PCD) process, also named ordered cellular suicide process, which may happen in a multicellular organism as a controlled mechanism to maintain the balance of cell multiplication (Green, 2011; Lakna, 2017; Schmidt et al., 2004). Another form of cell death is necrosis or accidental cell death and this is mainly caused by massive cellular damages or cellular distress (Schmidt et al., 2004). There are two significant types of signalling pathways for apoptosis: extrinsic, which is initiated directly through the ligand-mediated activation of membrane death receptors, and intrinsic, which is controlled by members of the Bcl2 family and mitochondria-derived proteins (Schmidt et al., 2004).

Introducing apoptosis in the aberrant white blood cells is a common approach to stop cumulative mutations (Green, 2011). The process of apoptosis in cells involves multiple biochemical events that lead to characteristic cell changes, such as cell shrinkage, blebbing, chromatin condensation, nuclear fragmentation, chromosomal DNA fragmentation and death (Green, 2011). Drugs like glucocorticoids are commonly applied in chemotherapy. Glucocorticoids are a family of steroid hormones containing synthetic products like dexamethasone (Dex), and prednisolone (PRD). Dex is a synthetic steroid, which is therapeutically used instead of the natural human glucocorticoid, cortisol (Thompson & Johnson, 2003). Glucocorticoids are essential steroid types of drugs commonly used to induce apoptosis in the malignant cells of childhood acute lymphoblastic leukaemia during the process of chemotherapy, due to the ability of these steroids to repress the growth and to cause the apoptotic death of these cells (Planey, Abrams, Robertson, & Litwack, 2003; Thompson & Johnson, 2003).

However, prolonged use of chemotherapy to introduce apoptosis may result in severe short-term or long-term side effects, such as osteoporosis, hypertension, psychosis, Cushing's syndrome and leucopenia (Chaiboonchoe, 2010; Rhen & Cidlowski, 2005; Schmidt et al., 2004; Smith & Cidlowski, 2010). An immune overreaction, namely cytokine release syndrome, can trigger high fevers, plummeting blood pressure and, in severe cases, also causes organ damage.

The GC enter into the leukaemia cell via a functional glucocorticoid receptor (GR), i.e., NR3C1 (Rainer et al., 2012), which is a ligand-activated transcription factor that exerts a pivotal role in inducing apoptosis in malignant lymphoid cells. The steroids as located in the cytosolic compartment in the absence of ligands (Thompson & Johnson, 2003). When GRs bind with ligands on their high-affinity site in the carboxy-terminal portion, the GRs translocates to the nucleus and are associated them with other transcription factors, to regulate specific sets of genes (Thompson & Johnson, 2003). However, GR alone is not sufficient for producing apoptosis. Accumulating evidence suggests that many leukaemic cells, which contain abundant quantities of normal GRs, are still unaffected by glucocorticoid-evoked apoptosis. For example, the steroid ligands could be blocked from passage through the plasma membrane and; hence, are destroyed biochemically-conjugated with GRs (Thompson & Johnson, 2003). Besides, the resistant cells may have genetically or phenotypically altered the response systems to glucocorticoids to resist their lethal effects, such as critical reductions in the quantity of one or more transcription factors, development of a dominant-negative form of such a factor or improper post-translational modifications of GR or an interactive

factor (Thompson & Johnson, 2003). The changes that affect the general pathways for apoptosis, such as alterations in the balance of pro- and anti-apoptotic members of the Bcl2 family of proteins; the loss of or inactivating mutations in caspases or other lethal proteases; changes in one or more critical protease substrates rendering them. Resistant; and alternating in specific genes' abilities to be regulated by ligand-driven GR (Thompson & Johnson, 2003).

Moreover, cellular gene transcription and translation are essential for GR-evoked apoptosis of lymphoid cells. There is evidence that blocking cellular transcription or translation prevents the advent of the classic morphological and biochemical events in the apoptotic pathway (Thompson & Johnson, 2003).

Currently, the transactivation or transrepression of target genes by GC is still not well understood, i.e., the clinical effects of GCs are poorly understood (Yoshida et al., 2002). For example, the mechanisms of glucocorticoid resistance in the clinical setting remain largely unresolved because the findings from the cell line model of glucocorticoid resistance in childhood acute lymphoblastic leukaemia (ALL) almost invariably exhibit altered glucocorticoid receptor (GR) function are incongruous with those using specimens derived directly from a leukaemia patient (Bachmann et al., 2007). Besides, GC signalling exerts a wide range of physiological actions because of the broad distribution of the GR. The actions include positive regulation of metabolism in the liver, adipose tissue or the induction of apoptosis and cell cycle arrest, and antiinflammatory effects in the immune compartment (Rainer et al., 2012).

Alterations in glucose metabolism contribute to cell death and survival decisions, especially in the lymphoid lineage (Carlet et al., 2010) in which lymphocyte-dependent extracellular signals are transmitted via surface receptors and survival kinases like PKB/Akt into cells and influence glucose metabolism. Glucose metabolism can be influenced by increasing the expression of glucose transporters and, as a consequence, this increases the productivity of glycolysis and ATP (Carlet et al., 2010). Reducing the production of glycolytic and ATP/ADP ratios causes a loss of integrity in the mitochondria, and this results in Bax-bak dependent cytochrome-C release and cell death (Carlet et al., 2010).

Glucocorticoids (GC) can suppress glucose utilisation by reducing both glucose uptake and glucose oxidation in two significant tissues, i.e., skeletal muscle and WAT (Kuo, McQueen, Chen, & Wang, 2015). The ability of GC has been connected to GC induced apoptosis in leukaemia cells, in which GC down-regulates the expression of glucose transporter 1 (GLUT1). The down-regulated expression of GLUT1 results in reduced glucose uptake into leukaemia cells (Kuo et al., 2015). Hence, GC induced apoptosis has physiologic and therapeutic significance in the treatment of lymphoid malignancies, particularly childhood acute lymphoblastic leukaemia (ALL) (Carlet et al., 2010).

The cognate receptor (GR) is a ligand-activated transcription factor in the massive nuclear transcription factor family and was critically essential for GC induced apoptosis (Carlet et al., 2010). The levels of GR and the subsequent alterations in gene

expression affected the effectiveness of GC treatment. Gene NR3C1 is a glucocorticoid receptor (GR) and is a member of the nuclear receptor subfamily 3, group C. Hence, NR3C1 was a critical gene to induce apoptosis of leukaemia cells.

Although the process of how GC-induced lipolysis affected glucose homeostasis was not transparent, the fatty acids generated from lipolysis were likely mobilised to skeletal muscle and liver and converted into lipid mediators, such as diacylglycerol (DAG) and ceramides, that resulted in insulin resistance (Carlet et al., 2010).

FKFB2 (6-phosphofructo-2-kinase/fructose-2,6-biphosphatase-2) was another crucial regulator of glycolysis that was induced more than 4-fold in all three T-ALL cases as well as in the T-ALL cell line CCRF-CEM (Carlet et al., 2010).

Research conducted by (Carlet et al., 2010) suggested that the GC response gene, PFKFB2 (6-phosphofructo-2-kinase/fructose-2,6-biphosphatase-2) was a kinase controlling glucose metabolism, was not a critical upstream regulator of the anti-leukaemic effects of GC. FKFB2 was induced more than 4-fold in all three T-ALL cases as well as in the T-ALL cell line CCRF-CEM (Carlet et al., 2010).

To understand these drug-related genetic problems scientists try to reconstruct the dynamics represented by time and the discrete state transition systems to gain insights into the functioning of cell systems (Ay & Arnosti, 2011; Hood, 2013; Lee & Tzou, 2009; Y. Wang et al., 2011). These dynamics can be used to simulate the perturbations of new drugs *in silico* to reduce the potential risks of applying drugs to human beings. Two common research issues are emerging for GCs: GC regulated

genes and the glucocorticoid receptor gene network. Signalling pathways and gene networks can be inferred from gene expression data grouped in a time series format.

2.4. Summary

In chapter 1, the gaps in the current Boolean modelling has been well discussed. In general, a fragment of DNA (gene) will transcribe into mRNA and then translate into protein. The fundamental concept of activator and inhibitor are the two critical elements in this study. However, the roles of gene activation and inhibition in traditional Boolean modelling cannot be intuitively understood. For example, the traditional Boolean rule of the cyclin gene *CycA* depends on a complex Boolean rule; in which the roles of activator and inhibitor cannot be identified easily. Besides, a gene may remain activated within a period of decay time when there are no activators present. To solve the gaps, we reviewed the historical development of Boolean modelling and discussed the variants of the conventional Boolean modelling, including synchronous, asynchronous and hybrid models in section 2.1 in details.

The objectives of this research mainly focus on developing a better understanding of the fundamental mechanisms in genetic regulatory pathways about cancer-related treatments. Hence, we reviewed the current DNA microarray technology, including the design of experiments to extract mRNA samples, that has been applied to analyse human cancers using the Affymetrix GeneChip Operating System Software (GCOS), in section 2.2. The GCOS technology paved a significant way to develop a better understanding of cancer prediction and diagnosis for the treatment of malignant

tumours. The technology generates microarray gene expression data (CEL files) for extracting differentially expressed genes. Differentially expressed genes are highly dependent on the normalisation methods, and hence, we reviewed the most common normalisation methods, including MAS5.0, RMA and GCRMA.

The data we used to explore are cell cycle and acute childhood leukaemia, and hence we reviewed the background knowledge of cancer, cell cycle and acute childhood leukaemia in section 2.3. Cancer is a significant kind of fatal genetic disease due to aberrant gene regulation, such as mutations in single somatic cells. The induction of apoptosis and cell cycle arrest on cancer cells are common ways for cancer-related treatments.

The following chapters propose a novel data-driven Boolean modelling and its extension for genetic regulatory pathways to address the gaps of Boolean modelling.

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Chapter 3: Fundamental Boolean Modelling

As discussed in chapter 1 and reviewed in chapter 2, the hypotheses of conventional Boolean models do not provide an intuitive technique to separate the individual activation and inhibition pathways. The processes of gene activation and inhibition are the two fundamental processes of genetic regulation. For example, activation may result in substantial drug regulatory effects, such as modifications in the metabolism of *in vivo* substances and vitamins (Barry & Feely, 1990). Likewise, inhibition may result in crucial clinical drug interactions being formed by a wide range of drugs (Barry & Feely, 1990). Inhibition can be classified into two groups: reversible inhibitors that can be easily inverted by dilution or dialysis since the interactions of this group are non-covalent with the enzyme surface (Saboury, 2009); and irreversible inhibitors that usually persist even during complete protein breakdown due to their sturdy covalent bonds on the enzyme surface (Saboury, 2009).

Hypothetically, under the theory of an enzyme reaction exposed to the action of a reversible inhibitor, the degree of inhibition may be modelled as the decreased rate of reaction divided by the uninhibited reaction rate (Saboury, 2009):

$$i = \frac{V_0 - V}{V_0} \quad (3.1)$$

where V and V_0 represent the rates of the inhibited and uninhibited reactions, respectively (Saboury, 2009). The degree of inhibition (i) may present uncertainty into the target gene if the value of i is lower than 1. Similarly, enzyme activation contains the same concept as a reversible type of reaction. Hence, the degree of inhibition can

be upgraded to the degree of the enzyme reaction; thus, encapsulating the degrees of inhibition and activation. For that reason, we could redefine the degree of the enzyme reaction to a conditional probability measure to represent the propensity rate of an enzyme reaction towards the target gene. A conditional probability measure is the probability of an event that occurs given another event has happened. If the conditional probability measure of an inhibitor is 1, the inhibitor is irreversible; otherwise, it is reversible.

Conventional Boolean models do not consider the reversible and irreversible behaviour of enzyme reactions. In biology, the disappearance of an activator does not preclude the emergence of an inhibitor because the proteins transcribed by a pre-activated gene might be still in the status of activation. The way we judge whether a gene activates or inhibits based solely on the concentration rate of the proteins produced by the gene. Therefore, there are logical reasons to separate the general Boolean function into the domains of gene activation and inhibition.

To analyse the Boolean networks for gene activation and inhibition, we propose a novel Boolean model for constructing the dynamic activation and inhibition networks, based on the abstraction of the features of enzyme activation, inhibition and the long-term degradation of a specific protein. Chapters 3 and 4 explain the published novel model and the related network inference methodology in detail.

3.1. Fundamental Boolean Model

Extending the original definition of a Boolean model, we defined a novel Boolean network as a graph $G(X, E_a, E_d)$, where the node collection, $V = \{v_1, v_2, \dots, v_n\}$, corresponds to a group of states, $X = \{x_i | i = 1, \dots, n\}$ of size n . Each node is a variable that is only in one of two states: *On* (1) or *Off* (0). The general edge set, E , is divided into two sets of fundamental Boolean functions, E_a and E_d , based on their regulatory functions, i.e., activation and inhibition, rather than a single function, as in all conventional Boolean models. The direction of the edges represents the propagation of their effectiveness on the target node, such as the signal flow between signalling molecules, genes or protein regulation.

We conceptualised this graph as a new type of Boolean network, namely the fundamental Boolean network (FBN). The two sets of fundamental Boolean functions are modelled as:

Fundamental Boolean functions of activation:

$$F_a^i = \{f_{a_j}^i | j = 1, \dots, l_a(i)\}, f_{a_j}^i: \{0, 1\} \rightarrow \{-, 1\} \quad (3.2.a)$$

Fundamental Boolean functions of inhibition:

$$F_d^i = \{f_{d_k}^i | k = 1, \dots, l_d(i)\}, f_{d_k}^i: \{0, 1\} \rightarrow \{-, 0\} \quad (3.2.b)$$

where F_a^i and F_d^i denote a set of fundamental Boolean activation and inhibition functions of gene i , respectively. Notably, $-$, here, refers to that the output of the function does not affect the target gene. $l_a(i)$ symbolises the total number of fundamental Boolean functions activating the target gene. $l_d(i)$ symbolises the total

number of fundamental Boolean functions deactivating the target gene. When the output of a Boolean activation function is *TRUE*, this means that the target gene is to be activated and *FALSE* means that the activation function does not influence the target gene, as denoted by $-$. Similarly, when the output of a Boolean inhibition function is *TRUE*, this means that the target gene is to be repressed and *FALSE* means that the inhibition function does not affect the target gene. The definition of the two types of Boolean functions set out the novelty of the proposed Boolean modelling.

The proposed fundamental Boolean functions have the general assumption that the production of each gene at every timestep is either completely activated or wholly inhibited when the output of the functions are determined. Based on the treatment time, the gene regulation time is embedded in the selected Boolean updating schema, i.e., as a synchronous or an asynchronous system.

The essential biological philosophies behind the fundamental Boolean functions are that a fundamental Boolean function can be treated as a simple transition rule. The rule takes a minimum required essential gene states as the input and then governs their regulation effects on the target gene. In general, a fundamental Boolean function is an atomic function that cannot be separated any further. Hereafter, we can treat the fundamental Boolean functions as conditions that constrain gene activity, a delegation of stereochemical reactions, and a transcription factor complex moulded by the transcription factor to proteins or protein to protein bindings.

The output of the proposed fundamental Boolean functions is only associated with the potential effectiveness of gene regulation on the target gene. For that reason, there is a need to calculate the level of confidence by what percentage we can trust the regulatory functions in affecting the target gene. As stated previously, the degree of enzyme reaction can be substituted by the conditional probability that an enzyme reaction can influence the target gene. Hereafter, the concept of conditional probability can be used to measure the confidence of the proposed functions. The following formulae called confidence measures, model the conditional probability of each fundamental Boolean function.

Confidence measure of activation:

$$C_{a_j}^i \left[f_{a_j}^i \left(A_i^j(t) \right) \right] = p(\sigma_i^{t+1} = 1 | A_i^j(t) = 1) = \frac{p(A_i^j(t)=1 \cap \sigma_i^{t+1}=1)}{p(A_i^j(t)=1)} \quad (3.3.a)$$

Confidence measure of inhibition:

$$C_{d_k}^i \left[f_{d_k}^i \left(D_i^k(t) \right) \right] = p(\sigma_i^{t+1} = 0 | D_i^k(t) = 1) = \frac{p(D_i^k(t)=1 \cap \sigma_i^{t+1}=0)}{p(D_i^k(t)=1)} \quad (3.3.b)$$

where σ_i^t denotes the Boolean state of gene i at time t , and σ_i^{t+1} denotes the Boolean state of gene i at time $t + 1$. \cap refers to a logical *And* connector. $C_{a_j}^i$ and $C_{d_k}^i$ delegate the confidence function with the input of the fundamental Boolean functions $f_{a_j}^i$ and $f_{d_k}^i$, respectively. A_i^j and D_i^k denote the set of inputs required or the state of the gene functions, $f_{a_j}^i$ and $f_{d_k}^i$, respectively. $A_i^j(t) = 1$ or $D_i^k(t) = 1$ mean the required gene input of $f_{a_j}^i$ or $f_{d_k}^i$ at time t is satisfied with the conditions of affecting the target gene, i .

The confidence measures of the activation and inhibition functions can simulate the stochastic epigenetic switches of gene regulation in nature, such as chromosomal rearrangements, by turning the confidence values up or down, compared with the standard example of *lac* operon (Edwards & Bestor, 2007). Examples of chromosomal rearrangements are the effect of variation in *Drosophila*, the telomere position effect in yeasts (Edwards & Bestor, 2007), competition between the OxyR repressor (a regulator of antioxidant genes). Dam (DNA adenine methyltransferase) controls the activity of the *agn43* promoter that causes neither OxyR or Dam to be 100% efficient (Edwards & Bestor, 2007).

There are various debates about mRNA/protein decay times in Boolean models. The decay time is the time that allows a gene to remain in the *On* state when there are no activators or inhibitors. R. Albert (2004) assumed that this decay might occur in two time steps. To capture the characteristics of protein decay, we induced a function f_{decay} (given below) to fulfil the requirements of protein degradation with input from the target gene i at time t :

$$f_{decay}(\sigma_i^t, \vartheta) = \neg(\tau \leq \vartheta) \times \sigma_i^t \quad (3.4)$$

where τ represents an incremental variable presenting the number of time steps processed. τ will be reset to 0 when there is any fundamental Boolean function affecting the target gene (i) at time $t + 1$. ϑ delegates the decay period to reflect the fact that the attenuation or enhancement of the expression of mRNA requires time. \neg represents a negation operator that changes a Boolean function from *TRUE* to

FALSE or vice versa. \times is a logical And operator. The output of the decay function f_{decay} is a Boolean state of On (1) at time $t + 1$ if the gene state of σ_i of time t is On (1) within the endured period or Off (0) at time $t + 1$ when the tolerated period is expired regardless of the gene state of σ_i of time t .

In this research, we assumed that the tolerated period for protein decay is one timestep, i.e., $\vartheta = 1$ because 80% of the microarray data are short time series data (Ernst et al., 2005). Short time series data contain only a few observations from which knowledge of the mechanical details and kinetic parameters cannot be mined consistently from the data (Chaiboonchoe, 2010; Ernst et al., 2005; Z. Wang et al., 2008).

By combining equations Eq.(3.2a), (3.2.b), (3.3.a), (3.3.b) and (3.4), we now define the novel Boolean model (FBM) as:

$$\sigma_i^{t+1} = (f_{decay}(\sigma_i^t, \vartheta) + \bigvee_{j=1}^{l_a(i)} \{P \llbracket C_{a_j}^i \llbracket f_{a_j}^i(A_i^j(t)) \rrbracket \rrbracket \}) \times \neg \bigvee_{k=1}^{l_d(i)} \{P \llbracket C_{d_k}^i \llbracket f_{d_k}^i(D_i^k(t)) \rrbracket \rrbracket \}$$

(3.5.a)

where $+$ is a logical *Or* operator and \times is a logical *And* operator. The decay function $f_{decay}(\sigma_i^t, \vartheta)$ in Eq.(3.4) is to ensure the gene state σ_i at time $t + 1$ depends on the pre-state of the gene at time t if no activators are present at time t and they are still tolerated by the parameter ϑ , a decay period. $P \llbracket x \rrbracket$ is a Boolean function that takes a uniform distributed random number, μ , and an output of 1 if $\mu < x$ and 0 otherwise.

$V\{x\}$ denotes the logical connective function of *Or*, i.e., $V_{j=1}^{l_a(i)} \{F_{a_j}^i\} = P \llbracket C_{a_1}^i (f_{a_1}^i) \rrbracket +$

$P \left[\left[C_{a_2}^i \left(f_{a_2}^i \right) \right] \dots + P \left[\left[C_{a_{i_a(i)}}^i \left(f_{a_{i_a(i)}}^i \right) \right] \right]$. For example, if the input gene states of $f_{a_1}^i$ at time t are satisfied, i.e., $A_i^j(t) = 1$, and $C_{a_1}^i$ has a confidence of 100% on the function $f_{a_1}^i$, the $P \left[\left[C_{a_1}^i \left(f_{a_1}^i \right) \right] \right]$ is $ON(1)$; and $V_{j=1}^{l_a(i)} \{F_a^i\}$ then is $ON(1)$, regardless of other activation functions. If $V_{j=1}^{l_a(i)}$ is $On(1)$ as well, according to the model defined in Eq(3.5a), it will turn the gene state σ_i at time $t+1$ $OFF(0)$, which means if an inhibitor is present and has an effect on the target gene (i), the inhibitor will inhibit the target gene, i .

Figure 3-1 shows an example of FBN. The left hand side outlines a wiring diagram of FBN; the top right hand side shows the Boolean functions in the conventional format; the bottom right hand side shows the Boolean functions in the format of a fundamental Boolean function. Both groups of functions have identical functionalities except that the second set displays the rules separated by the types of activators and inhibitors.

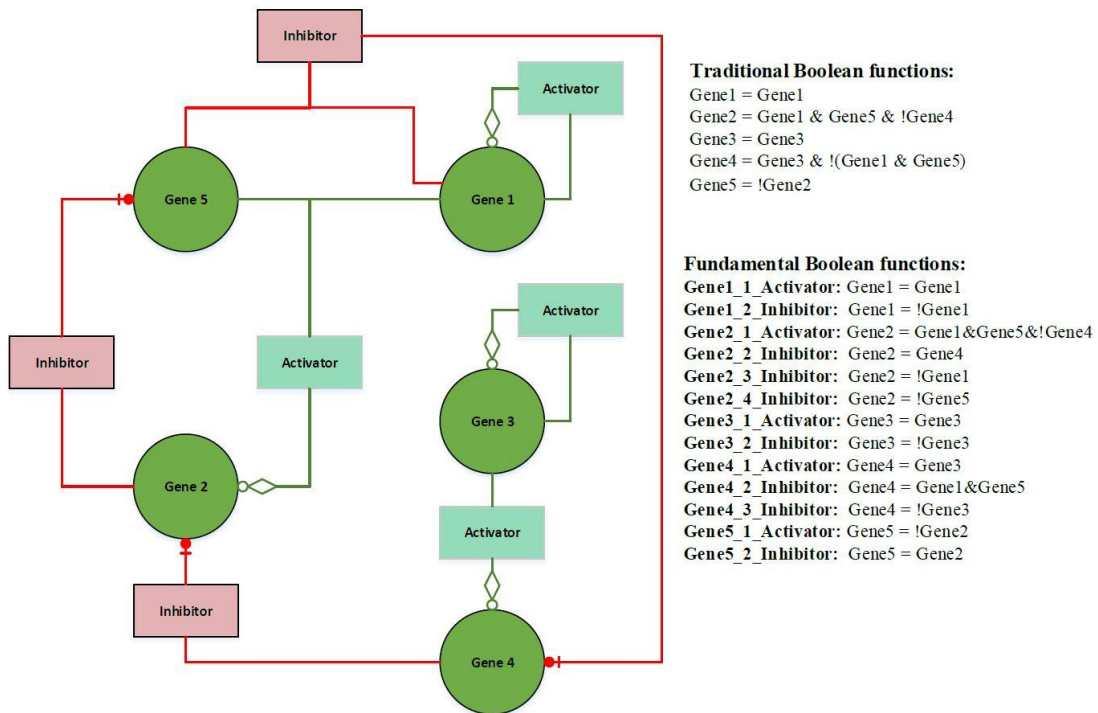


Figure 3-1 Example of a fundamental Boolean network. The icon box refers to a fundamental Boolean function. The red box refers to an inhibition function, and the light green box refers to an activation function. The green circle icon refers to a gene or a variable.

As given in Figure 3-1, the wiring diagram represents the dependencies between activation and inhibition, where the expression level of gene 4 at time $t + 1$ not only depends on the value of the activation rule associated with the expression level of gene 3 at time t but also depends on the value of the inhibition rule related to the expression level of genes 1 and 5 at time t . Hence, the model for gene 4 to be activated or inhibited at the next timestep is outlined, below:

$$\begin{aligned}
Gene_4^{t+1} &= (f_{decay}(gene_4^t, 1) \\
&+ P[[C_{a_1}^4 | f_{a_1}^4(gene_3^t)]]]) \times \neg(+ P[[C_{d_1}^4 | f_{d_1}^4(gene_1^t \& gene_5^t)]]]) \\
&+ P[[C_{d_2}^4 | f_{d_2}^4(!gene_3^t)]]])
\end{aligned}$$

If gene state at time t is: $gene_1=1$, $gene_3=1$, $gene_4=1$, $gene_5=1$ and the protein decay is 1, the above formula, then, is transferred to:

$$Gene_4^{t+1} = (1 + P[[C_{a_1}^4 | 0]]) \times \neg(P[[C_{d_1}^4 | 1]] + P[[C_{d_2}^4 | 0]])$$

Therefore, the final result of $Gene_4$ at time $t + 1$ can be calculated if $C_{d_1}^4 [1] = 1$ and, hence, $P[[1]] = 1$ ($P[[0]] = 0$):

$$Gene_4^{t+1} = (1 + 0) \times \neg(1 + 0) = 1 \times \neg(1) = 1 \times 0 = 0$$

The final result of the above formula is 0, which indicates that $gene_4$ at time $t + 1$ is inhibited.

Another sample, as shown in Figure 3-2, illustrates how the proposed model (FBM) simulates the dynamic equilibrium of gene regulation. There are only two rules in this example: Gene A is an activator of gene B, and gene B is an inhibitor of gene A. To demonstrate this, we first defined the FBM parameters as the timestep required by protein decay is 1; in the timestep where a gene completes its regulatory process it is 1; the confidence measure of each rule is 1 (100%), and we selected the synchronous scheme as the updating schema. The following illustrates four cases:

In case 1, Gene B activated by Gene A at timestep 2, then turned *Off* at timestep 3 due to the decay of the protein. Gene A was turned *Off* after protein decay at the timestep 2. After that, both genes reach the equilibrium state.

In case 2, Gene A was suppressed by Gene B at timestep 2. Gene B was repeatedly enhanced by Gene A at timestep 2 but decayed at timestep 3. Both genes now reach the equilibrium state at timestep 3.

In case 3, Gene A is repressed by Gene B at timestep 2, and Gene B decayed. Both genes now reach the equilibrium state at timestep 2. The gene states remain unchanged after timestep 2, and this means they are entrapped into a simple loop, i.e., an attractor, due to the lack of activators to perturb the equilibrium state.

In case 4, this is the same as case 3 at time step 2; hence, all the cases are entrapped by the same attractor, i.e., the gene state of {0, 0}.

Case 1

	Timestep 1	Timestep 2	Timestep 3
Gene A	1	0	0
Gene B	0	1	0

Case 2

	Timestep 1	Timestep 2	Timestep 3
Gene A	1	0	0
Gene B	1	1	0

Case 3

	Timestep 1	Timestep 2	Timestep 3
Gene A	0	0	0
Gene B	1	0	0

Case 4

	Timestep 1	Timestep 2	Timestep 3
Gene A	0	0	0
Gene B	0	0	0

Figure 3-2 Simulation of the dynamic equilibrium of gene regulation

3.2. Temporal Fundamental Boolean Model

The original FBM we defined in Eq(3.5.a) provides a mechanism to calculate gene state σ_i at time $t + 1$ based on the immediately before time t ; however, in reality, some gene regulations might require more time steps to complete than other genes. For the gene at $t + 1$, its state is not only dependent on the inputs of the immediately before t but also $t - 1, \dots, t - m$ ($m < t$ but ≥ 1). m here refers to the maximum temporal decrement value. We now defined another novel concept by extending the original FBN as graph $G(X, \mathbf{E}_a, \mathbf{E}_d, T)$, where T is the best temporal time step for an edge function to complete its biochemical reaction. A similar networked concept is the temporal Boolean networks, as discussed by Silvescu and Honavar (2001), but we use a different definition for the Boolean functions, i.e., $\mathbf{E}_a, \mathbf{E}_d$.

T is the time step, which has a minimum distance between the measurement matrix of $t - m$ and the perfect measurement matrix. A perfect measurement matrix contains all measures in the best states, such as a confidence value of 1. We denoted this extension model as the temporal fundamental Boolean model (TFBM), and its network as the temporal fundamental Boolean network (TFBN). TFBN may handle short time series better because it employs more timepoints than the initially proposed model. Furthermore, it reflects the reality that most biochemical reactions are asynchronous since each gene may be updated in different timescales.

TFBN assumes that the various durations of a fundamental Boolean function may have an impact on its target gene. The original FBN is a type of synchronous Boolean model

because it updates all gene states at the same time, while the extended FBN is asynchronous because its functions could have different time steps, which should be driven by data mining. Hence, we defined the extended model as:

$$\sigma_i^{t+1} = (f_{decay}(\sigma_i^t, \vartheta) + \bigvee_{j=1}^{l_a(i)} \{P \llbracket C_{a_j}^i \llbracket f_{a_j}^i(A_i^j(T_i^j)) \rrbracket \rrbracket\}) \times \neg \bigvee_{k=1}^{l_d(i)} \{P \llbracket C_{d_k}^i \llbracket f_{d_k}^i(D_i^k(T_i^k)) \rrbracket \rrbracket\}$$

(3.5.b)

where T_i^j and T_i^k are the best previous time step value for the activation function $f_{a_j}^i$ and inhibition function $f_{d_k}^i$, respectively, of gene i . The extended model respects the fact that a gene may be affected by a Boolean function at any previous time steps rather than its closest previous time step.

To calculate the best previous time step T_i^j or T_i^k for the extended model, we need to calculate all previous's measurement matrix that could derive the target gene i at $t + 1$ up to $t - m$ level. Let us define a measurement matrix as \tilde{A}_i for the activation function A_i^j , and a perfect matrix \ddot{A}_i , which contains the best value of each measurement in \tilde{A} :

If we define the measurement matrix as:

$$\tilde{A}_i = \begin{matrix} \text{measurement}_{i_1}^t & \text{measurement}_{i_1}^{t-1} & \dots & \text{measurement}_{i_1}^{t-m} \\ \text{measurement}_{i_2}^t & \text{measurement}_{i_2}^{t-1} & \dots & \text{measurement}_{i_2}^{t-m} \\ \dots & \dots & \dots & \dots \\ \text{measurement}_{i_e}^t & \text{measurement}_{i_e}^{t-1} & \dots & \text{measurement}_{i_e}^{t-m} \end{matrix}$$

Where e denoted as the element of the measurement matrix, which is a measure of how to infer the FBM networks discussed in the following chapter, and the perfect matrix as:

$\ddot{A}_i = \text{best value } (\ddot{A}_i)$

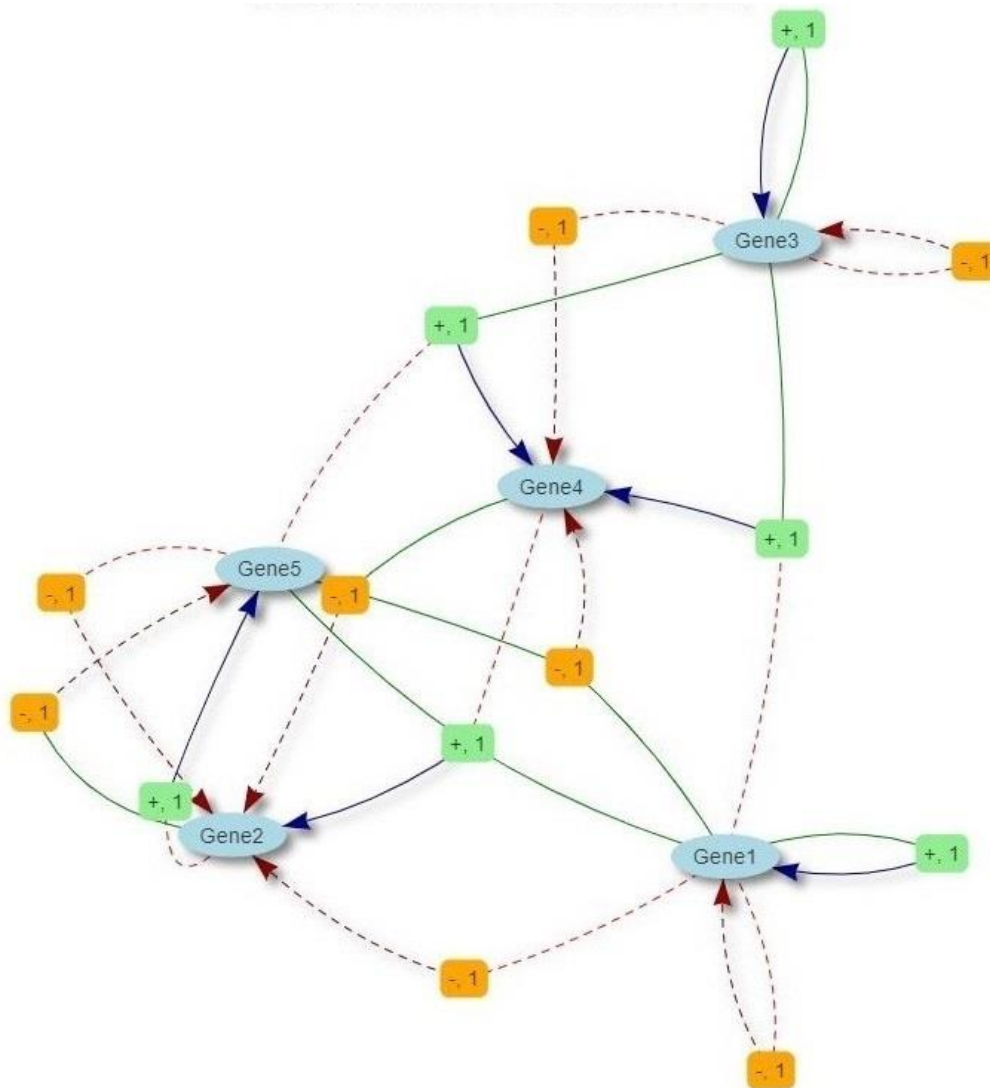
Then, the best previous time step value of the activation function $f_{a_j}^i$ is:

$$T_i^j = \min (\text{dist}_t^{t-m}(\ddot{A}_i, \ddot{A}_i)) \quad (3.6)$$

Eq(3.6) illustrates a simple method to find the best previous time step value where $\text{dist}()$ is a euclidean distance function, t denotes the current time step, and m is the maximum decrement value that can be supported by the previous time series data. However, it is not necessary to set m to the value of the total previous time points less one as the biological reaction might only need a few time steps to complete. It might be common to set the maximum decrement value (m) to two or three because about 80% of time series data are short time series data in which the sparse gap between each time step might not support the hypotheses that a regulation process of a gene might take more than two or three time points to complete.

3.3. Network Types of Fundamental Boolean Modelling

The proposed fundamental Boolean model splits the Boolean functions into the domain of gene activation and inhibition that facilitates us to analyse the Boolean regulatory pathways in different directions. The first FBN type is the general FBN type, namely FBNett_ALL (type 0), that contains up-regulatory and down-regulatory pathways, as shown in Figure 3-3.



Fundamental Boolean functions:

- Gene1_1_Activator: Gene1 = Gene1
- Gene1_2_Inhibitor: Gene1 = !Gene1
- Gene2_1_Activator: Gene2 = Gene1&Gene5&!Gene4
- Gene2_2_Inhibitor: Gene2 = Gene4
- Gene2_3_Inhibitor: Gene2 = !Gene1
- Gene2_4_Inhibitor: Gene2 = !Gene5
- Gene3_1_Activator: Gene3 = Gene3
- Gene3_2_Inhibitor: Gene3 = !Gene3
- Gene4_1_Activator: Gene4 = Gene3
- Gene4_2_Inhibitor: Gene4 = Gene1&Gene5
- Gene4_3_Inhibitor: Gene4 = !Gene3
- Gene5_1_Activator: Gene5 = !Gene2
- Gene5_2_Inhibitor: Gene5 = Gene2

Figure 3-3 An example of FBNNet_ALL (type 0). The legend refers to Figure 1.

The second FBN type is a forward network type, namely FBNNet_FAA (type 1), that shows the up-regulatory pathways of giving activated genes. The network type 1, shown in Figure 3-4, presents an example that the input gene Gene1 is activated, which causes the target gene Gene2 to be activated as a downstream effect.

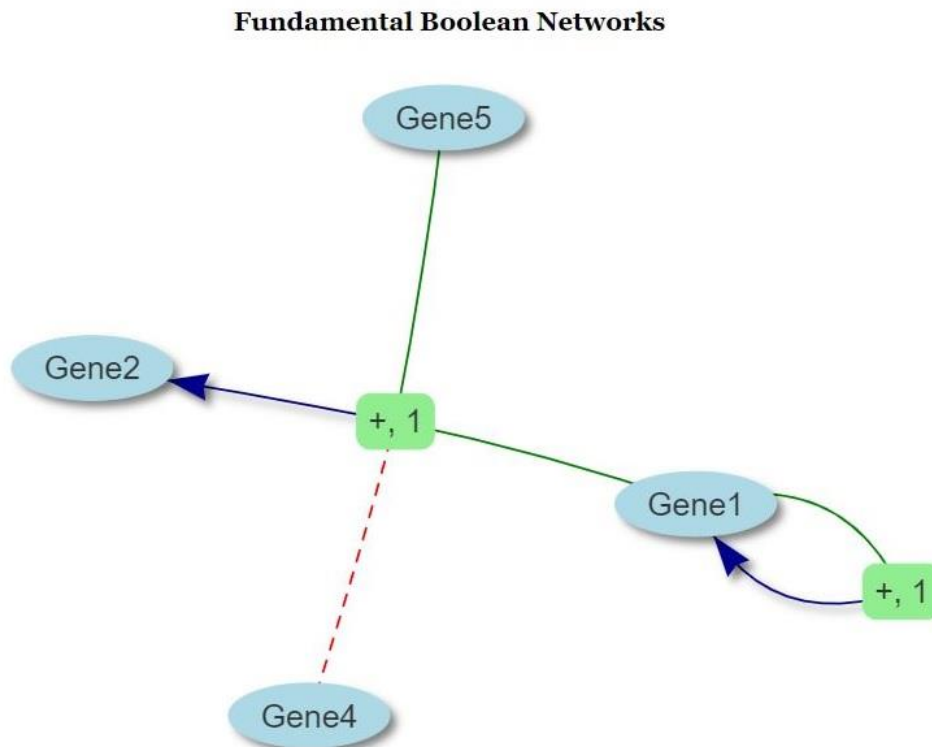


Figure 3-4 An example of FBNNet_FAA (type 1). The legend refers to Figure 1.

The third FBN type is also a forward network type, namely FBNNet_FAI (type 2), that shows the down-regulatory pathways of giving activated genes. The network type 2, shown in Figure 3-5, presents an example that the activation of the input genes Gene1 and Gene5 drives the target gene Gene4 to be inhibited as a downstream effect.

Fundamental Boolean Networks

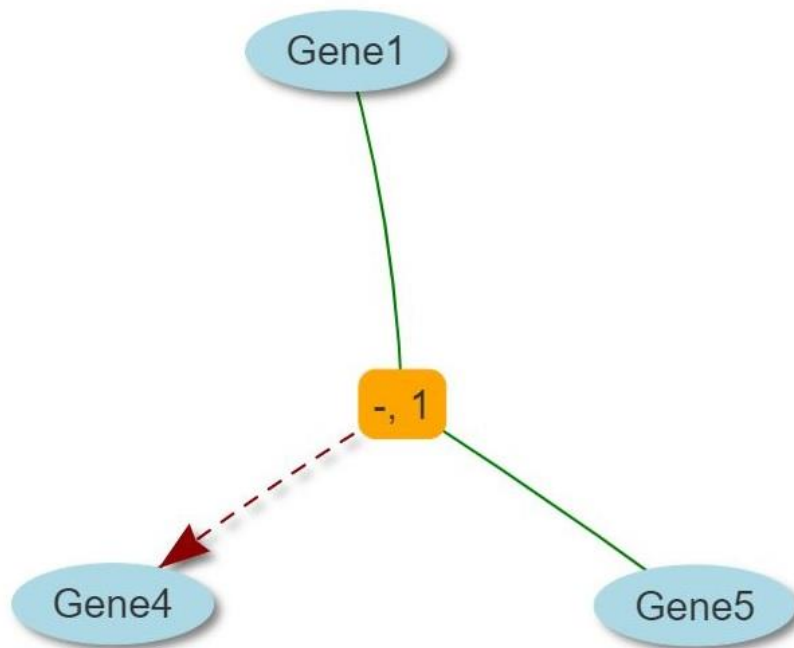


Figure 3-5 An example of FBNet_FAI (type 2). The legend refers to Figure 1.

The fourth FBN type is similarly a forward network type, namely FBNet_FIA (type 3), that shows the up-regulatory pathways of giving inhibited genes. The network type 3, shown in Figure 3-6, presents an example that the input gene Gene1 is inhibited that causes the target gene Gene4 to be activated as a downstream effect.

Fundamental Boolean Networks

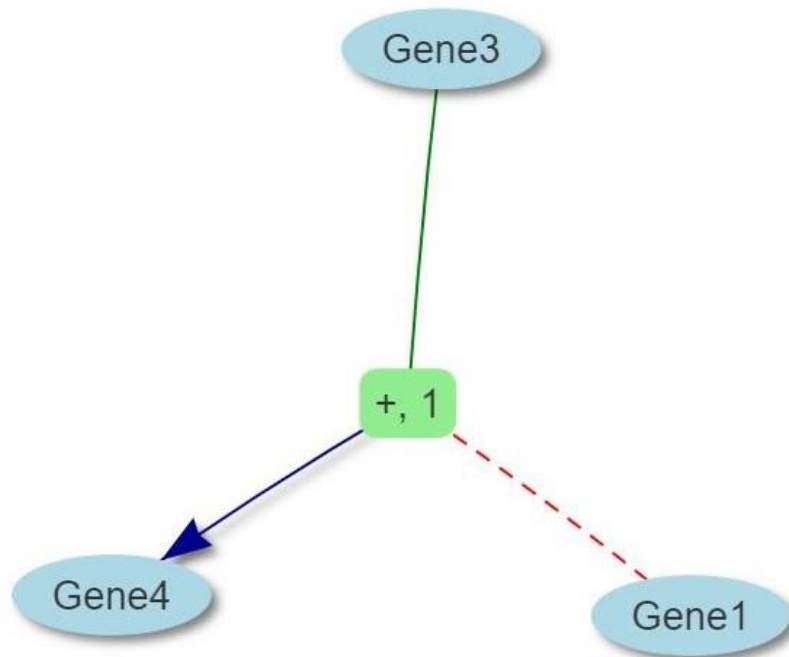


Figure 3-6 An example of FBNet_FIA (type 3). The legend refers to Figure 1.

The fifth FBN type is the last forward network type, namely FBNet_FII (type 4), that shows the down-regulatory pathways of giving inhibited genes. The network type 4, shown in Figure 3-7, presents an example that the input gene Gene1 is inhibited that causes the target gene Gene2 to be inhibited as a downstream effect.

Fundamental Boolean Networks

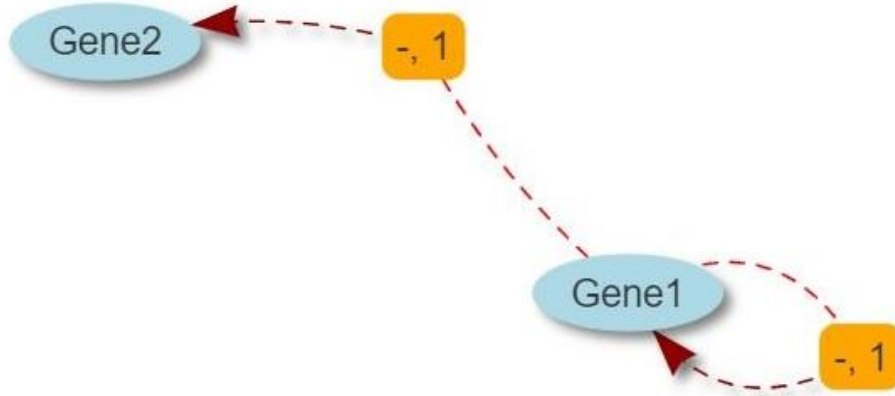


Figure 3-7 An example of FBNet_{FII} (type 4). The legend refers to Figure 1.

The sixth FBN type is the first backward network type, namely FBNet_{BA} (type 5), that shows the regulatory pathways that drives a target gene to be activated. The network type 5, shown in Figure 3-8, presents an example that the target gene Gene4 is activated as an upstream effect caused by either Gene3 & Not Gene1 or Gene3 & Not Gene5.

Fundamental Boolean Networks

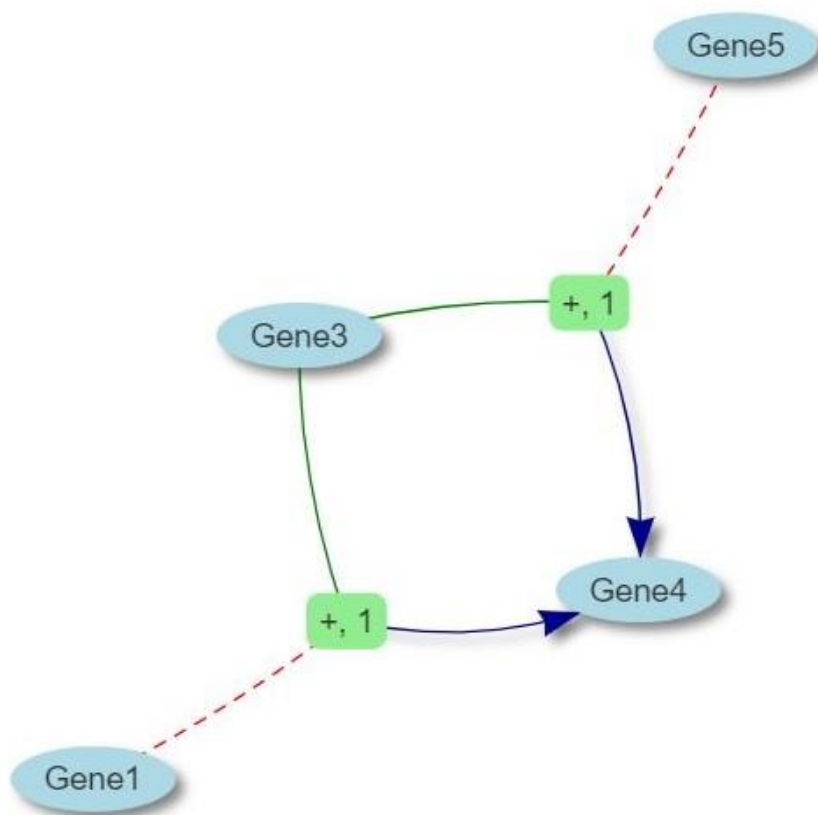


Figure 3-8 An example of FBNet_BA (type 5). The legend refers to Figure 1.

The seventh FBN type is the second backward network type, namely FBNet_BI (type 6), that shows the regulatory pathways that drive a target gene to be inhibited. The network type 6, shown in Figure 3-9, presents an example that the target gene Gene4 is inhibited as an upstream effect caused by either Gene5 & Gene1 or Not Gene 3.

Fundamental Boolean Networks

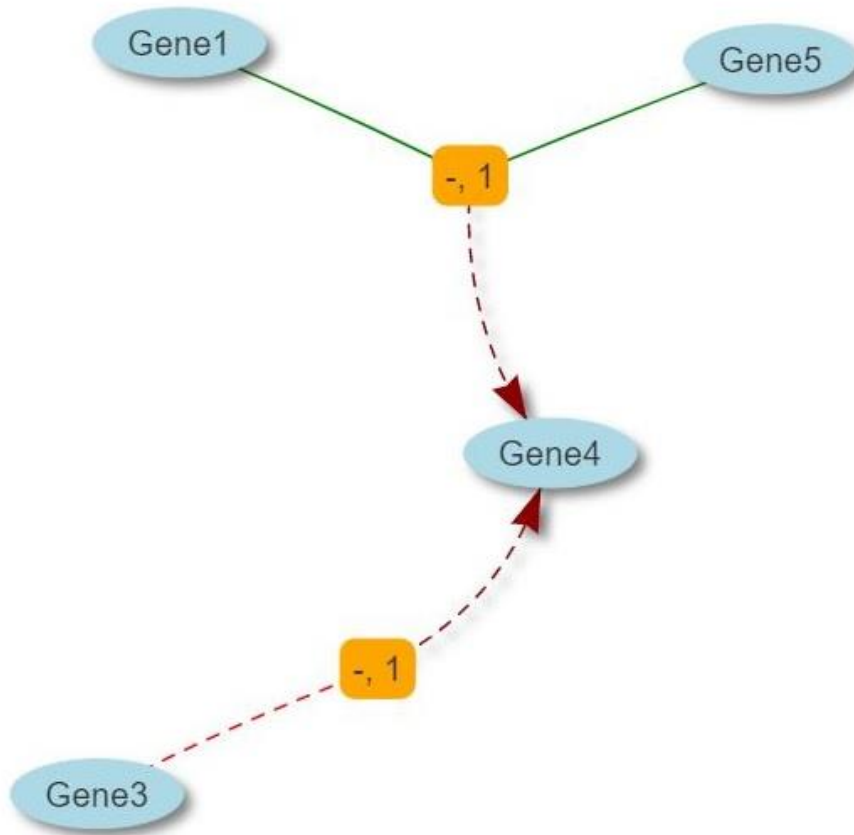


Figure 3-9 An example of FBNet_{BI} (type 6). The legend refers to Figure 1.

3.4. Summary

In this chapter, we proposed two novel Boolean models, i.e., the fundamental Boolean model and the temporal fundamental Boolean model, to intuitively analyse the activation, inhibition and protein decay networks. The type of the proposed models are an extension of Boolean network modelling, and seven FBN graphic types have been introduced. The possible applications of the mechanism can be applied to investigate the drug-related gene regulations because the inhibition pathway of a

novel drug can be exposed intuitively through an investigation of the drug-related fundamental Boolean networks. The critical challenge is how to extract the knowledge network (NK) from the drug-related dataset. The knowledge network, here, is typically referred to as the prior knowledge network (PNK) that encapsulates the biological knowledge, which is already known, for the main compounds being studied (Traynard et al., 2017). For most of the PNKs extracted from the literature, very few have been derived from data mining or related technologies.

The proposed Boolean model is novel, and there is no existing methodology to infer the related networks. Hence, we proposed a methodology documented in the following chapter to infer the related networks from time series data.

Chapter 4: Fundamental Boolean Network Inference

There are two main steps required to infer fundamental Boolean networks. The initial step is to construct a cube type database to store all critical precomputed measures, and the second step is to search for the best Boolean functions from the cube. Hence, the network inference process is separated from the process of constructing the cube and identifying the Boolean rules from the cube. The separation between the network extraction and construction of the cube enables further development of scalable methods to infer genetic networks effectively and efficiently because a cube has comparatively fewer updates although it can be consistently enhanced by feeding it more time series data.

4.1. Measure Matrix

To illustrate how to infer fundamental Boolean networks, we first explain the precomputed measures as follows:

- **Confidence Measures:**

As introduced in Eqs. (3.3.a) and (3.3.b), the confidence measures indicate a conditional probability of the input gene states at time t that regulates the target gene state at time $t + 1$.

- **Confidence Counter Measures:**

Similar to confidence measures, confidence counter measures are used to indicate the conditional probability of the target gene state at time t that regulates the conditional gene states at time $t + 1$. We denoted the confidence

counter measures as $CV_{a_j}^i$ and $CV_{d_k}^i$, respectively. a_j represents the j th fundamental Boolean function of activation and d_k represents the k th fundamental Boolean function of deactivation (inhibition):

Confidence counter measures of activation:

$$CV_{a_j}^i \left[f_{a_j}^i \left(A_i^j(t+1) \right) \right] = p(A_i^j(t+1) = 1 | \sigma_i^t = 1) = \frac{p(A_i^j(t+1)=1 \cap \sigma_i^t=1)}{p(\sigma_i^t=1)} \quad (4.1.a)$$

Confidence counter measures of inhibition:

$$CV_{d_k}^i \left[f_{d_k}^i \left(D_i^k(t+1) \right) \right] = p(D_i^k(t+1) = 1 | \sigma_i^t = 0) = \frac{p(D_i^k(t+1)=1 \cap \sigma_i^t=0)}{p(\sigma_i^t=0)} \quad (4.1.b)$$

Outputs, $CV_{a_j}^i$ and $CV_{d_k}^i$, are conditional probabilities, and the range of the value is between 0 and 1.

- **Support Measures:**

Support measures refer to the percentage of transactions that contain matched rules ($A_i^j(t) = 1 \cap \sigma_i^{t+1} = 1$ and $D_i^k(t) = 1 \cap \sigma_i^{t+1} = 0$) over all time steps across all samples.

If we denote the total number of time steps involved as \aleph :

$$\aleph = \sum_{i=1}^s (t_i - 1) \quad (4.2)$$

where t_i refers to the number of time steps of sample i and s refers to the total number of samples. The initial time step of sample i is not included because the calculation of the support measures requires at least a minimum of two time steps (t and $t+1$) and; hence, we remove 1 from t_i . We then represent the support measures as $S_{a_j}^i$ and $S_{d_k}^i$ for activation and inhibition, respectively:

Support measure of activation:

$$S_{a_j}^i = \frac{\text{count}(A_i^j(t)=1 \cap \sigma_i^{t+1}=1)}{N} \quad (4.3.a)$$

Support measure of inhibition:

$$S_{a_k}^i = \frac{\text{count}(D_i^k(t)=1 \cap \sigma_i^{t+1}=0)}{N} \quad (4.3.b)$$

- **Conditional Causality Test:**

Some researchers have argued that causality should not be a concept of statistics and not be statistically 'identifiable' since a secluded causal hypothesis cannot be verified using only observational data (Simcha, Younes, Aryee, & Geman, 2013). Nevertheless, we consider that the causality between the conditional gene and the target gene can be calculated using the following formulae:

$$\text{Conditional causality test} = \frac{\text{Confidence measure}}{\text{Confidence counter measure}} \quad (4.4)$$

These two types of measures have been deliberated in Eqs.(3.3.a), (3.3.b), (4.1.a) and (4.1.b). The proposed conditional causality test formulae in Eq.(4.4) is based on plausibility reasoning theory:

If gene B at time t has been observed to have induced gene A, at time $t + 1$, then the confidence of $p(A_{t+1} = 1|B_t = 1)$ is 100%. However, the reasoning that gene A at time t regulates gene B at time $t + 1$ may not be as strong as for gene A at time $t + 1$ due to a lack of information (observations) to support this reasoning. Hence, confidence $p(B_{t+1} = 1|A_t = 1)$ is less than or equal to $p(A_{t+1} = 1|B_t = 1)$. Hereafter, the ratio $p(A_{t+1} = 1|B_t = 1)$ divided by

$p(B_{t+1} = 1|A_t = 1)$, can then be used as a causality test to identify the direction of the regulation between genes A and B. This test can distinguish indirect regulators from direct ones because the indirect regulators usually have weaker observations than the direct ones.

The conditional causality test can be interpreted as follows:

- If the conditional causality test for target gene A, and conditional gene B, is greater than 1, we can conclude that gene A is regulated by gene B.
- If the conditional causality test is equal to 1, we can conclude that genes A and B are regulated by each other.
- If the conditional causality test is lower than 1, we can conclude that there is no causal relationship between genes A and B and so reject the hypothesis of that gene B regulates gene A.

4.2. Examples of Measure Matrix

Suppose we have three time series sample data for genes: CycD, p27, CycE, and E2F, as shown in the following table, Table 4-1. The time windows for time t are {1, 2, 3} and the time windows for time $t + 1$ are {2, 3, 4}.

Table 4-1 Sample discrete time series data for precomputed measures

	Sample 1				Sample 2				Sample 3			
Timepoint \ Genes	1	2	3	4	1	2	3	4	1	2	3	4
CycD	1	0	1	0	1	0	0	1	1	1	1	1
p27	1	1	1	0	1	1	1	0	0	1	1	1
CycE	1	0	1	0	0	0	0	0	0	0	0	0
E2F	1	1	1	1	1	1	1	1	1	1	1	1

Example of Confidence Measures

We use the data shown in Table 4-1 to calculate the confidence measure based on the hypothesis that p27 activates gene CycD, i.e., the regulatory function $f_{a_j}^i: p27^1 \rightarrow CycD^1$, so we use the formulae in Eq.(3.3.a). First, we calculate the value for $p(A_i^j(t) = 1)$, where $A_i^j(t): p27=1$ at time $t = \{1, 2, 3\}$. As shown in Table 4-1, the value of $p(A_i^j(t) = 1)$ is equal to 0.8889 (8 divided by 9). Now, we calculate the values for $p(A_i^j(t) = 1 \cap \sigma_i^{t+1} = 1)$. The value for $p(A_i^j(t) = 1 \cap \sigma_i^{t+1} = 1)$ is 0.4444 (4 divided by 9). Therefore, the confidence measure for the hypothesis is:

$$C_{a_j}^i \left[f_{a_j}^i(p27^1(t)) \right] = p(CycD^1(t+1) = 1 | p27^1(t) = 1) = \frac{0.4444}{0.8889} \approx 0.5$$

Examples of Confidence Counter Measures

Secondly, to calculate the confidence counter measure using the same hypothesis as the example above, we can use the formula in Eq. (4.1.a). As shown in Table 4-1, the value of $p(\sigma_i^t = 1)$ is equal to 0.6667 (6 divided by 9) and the value for $p(A_i^j(t+1) = 1 \cap \sigma_i^t = 1)$ is 0.5556 (5 divided by 9). Therefore, the confidence counter measure of the activation function is:

$$C\forall_{a_j}^i [f_{a_j}^i(p27^1(t+1))] = p(p27^1(t+1) = 1 | CycD^1(t) = 1) = \frac{0.5556}{0.6667} \approx 0.83$$

Examples of Support Measures

We use the same data, as shown in Table 4-1, to calculate the support measure for the same hypothesis, we use the formulae in Eq. (4.3.a). First, we calculate the value for \aleph , which is 9 in this case. Secondly, we count the event of which $(p27(t) = 1 \cap CycD(t+1) = 1)$ and the value is 4. Therefore, the support measure for the hypothesis is:

$$Support\ of\ activation = \frac{4}{9} \approx 0.4444$$

Examples of Conditional Causality Test

To calculate the conditional causality test measure with the same hypothesis as the examples, above, we can use the formulae in Eq.(4.4), and the values of the

confidence measures and confidence countermeasures are calculated from the examples above, to obtain the value of the conditional causality test.

$$\text{Conditional causality test} = \frac{0.5}{0.83} \approx 0.6$$

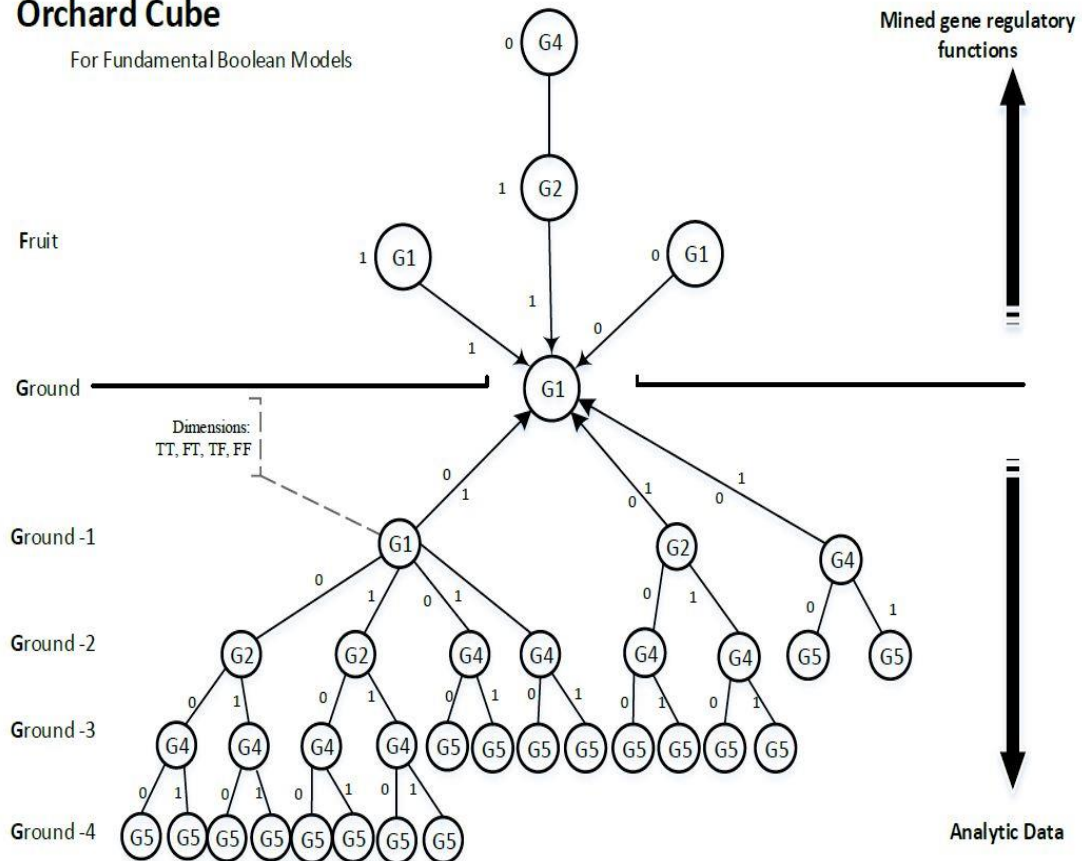
4.3. Orchard Cube

A data cube is a data abstraction providing a mechanism to analyse aggregated data from multiple dimensions. A data cube can also be regarded as a collection of identical 2-D tables stacked one upon the other. Many standard genetic time series data are multidimensional and involve the three main dimensions of genes, time steps, and samples. Researching multi-dimensional data could entrap performance bottlenecks. To release the performance bottlenecks, we can apply scalable mechanisms for quick access to the summarised data (Han, Kamber, & Pei, 2012).

To mine the fundamental Boolean networks, we extend the data mining technique of bottom-up computation (BUC) to a prefix tree type of cube; namely, Orchard cube, as shown in Figure 4-1. BUC is an algorithm designed for the computation of sparse cubes from the Apex cuboid downward (Han et al., 2012). We named this cube as Orchard cube because it looks like an orchard containing many fruit trees.

Orchard Cube

For Fundamental Boolean Models



Four major dimensions for each ground node and they are:
 TT: Target gene state is True and Conditional gene state is True.
 TF: Target gene state is True and conditional gene state is False.
 FT: Target gene state is False and conditional gene state is True.
 FF: Target gene state is False and conditional gene state is False

Figure 4-1 Illustration of an Orchard cube

Every branch or link of a tree above ground is referred to as a regulatory function. Each node under the first ground contains possible regulatory functions. Due to the regulatory functions being the information we are searching for, we call them *fruit*. The gene nodes on the ground are named *seeds*. The training data are called *fertilisers* as they aid the trees to grow better (more confident and; hence, more satisfied with the functions). This type of cube can distribute the computational costs to multiple computing nodes in a cloud computing environment because each branch can be

calculated independently. Moreover, the pre-computing cube can persist in any distributed database, so inferring networks from the cube is straightforward.

The underground nodes are analytical branches that contain all possible regulatory functions and measurements for the target genes. The nodes above ground contain the inferred regulatory functions mined from the branches underground.

Pairwise Dimensions

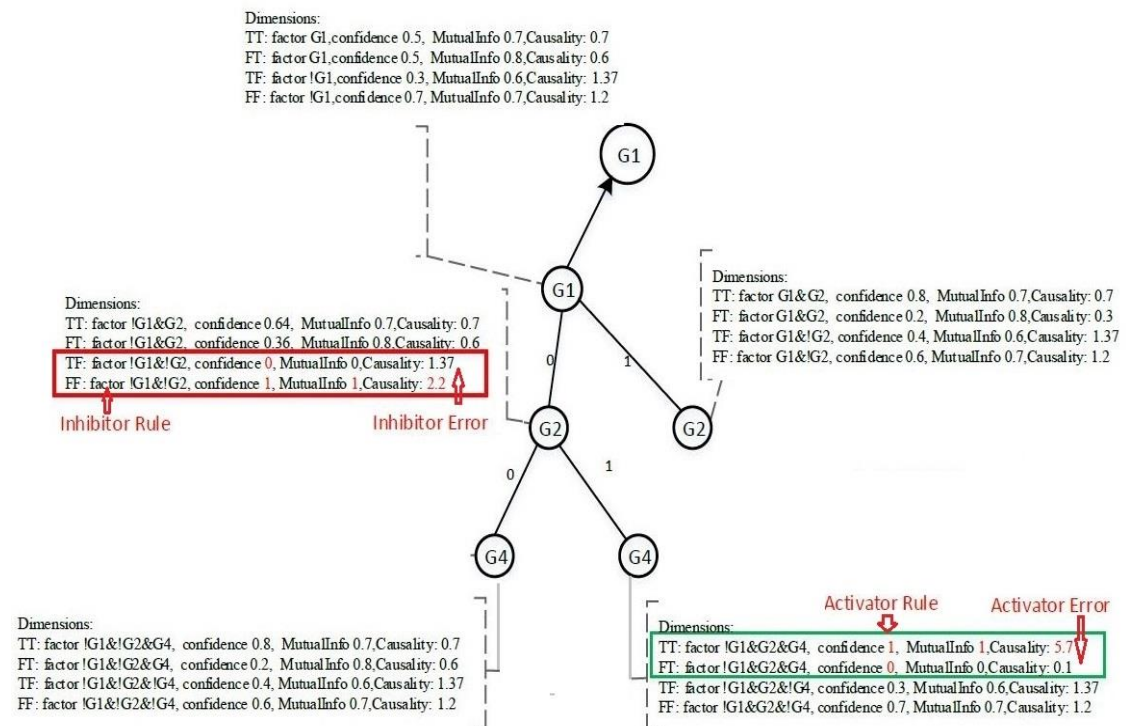


Figure 4-2 Sample nodes and measurement

As shown in Figure 4-2, a node comprises four dimensions, i.e., has four major groups of measures. Each dimension denotes a potential regulatory function of the target gene. The four dimensions are represented as TT, TF, FT and FF are outlined as follows:

- TT refers to a case when the target gene state at time $t + 1$ is *TRUE*. The current combination of the effectiveness of the input gene states at time t is *TRUE*, and all other upstream input gene states are fixed;
- TF refers to a case when the target gene state at time $t + 1$ is *TRUE*. The current combination of the effectiveness of the input gene states at time t is *FALSE*, and all other upstream conditional gene states are fixed;
- FT refers to a case when the target gene state at time $t + 1$ is *FALSE*. The current combination of the effectiveness of the input gene states at time t is *TRUE*, and all other upstream conditional gene states are fixed;
- FF refers to a case when the target gene state at time $t + 1$ is *FALSE*. The current combination of the effectiveness of the input gene states at time t is *FALSE*, and all upstream conditional gene states are static;

Notably, all nodes under Ground-2 will have prefixed gene states, as shown in Figure 4-1. For example, node $G4$ of the target gene, $G1$, at Ground-3 has its two upstream gene states, $G1$ and $G2$, fixed, i.e., $G1(0)$ and $G2(0)$. Hence, the four dimensions of the node $G4$ are outlined as TT ($G1|!G1\&!G2\&G4$), FT ($!G1|!G1\&!G2\&G4$), TF ($G1|!G1\&G2\&!G4$), and FF ($!G1|!G1\&G2\&!G4$).

As shown in Figure 4-2, each dimension has a factor that provides a function statement, which is the potential gene regulatory function. The dimensions TT and FT, FT and FF, are pairwise, respectively. The minimum confidences between TT and FT, FT and FF, are defined as error measures. The pairwise dimensions contain the features: $P(TT)=1-P(FT)$ and $P(TF)=1-P(FF)$.

For the pairwise dimensions, if we define one dimension to be the confidence measure, then, the other dimension is referred to as an error measure.

Orchard Cube Pruning

Building the cube entails the construction of an optimal tree-type data structure run for all likely combinations of the connected genes up to a maximum depth. Hence, the computational cost rises exponentially. This is endurable because all precomputed genes should not go to the next level to avoid redundant computations. For example, assume that there are three genes; A, B and C; and the conditional probability that gene A and gene B regulate the expression of C, is $p(C|A, B)$. The branch for pre-computing $p(C|B, A)$ is not processed from the main tree; therefore, $p(C|A, B)$ is equal to $p(C|B, A)$. Hence, the computational cost of constructing the full cube is reduced logarithmically.

Apart from the initial pruning, Pearson's Chi-square test (Plackett, 1983) could also be applied to exam the *NULL* hypothesis that a target gene is independent of the conditional gene. For example, all genes in GRNs will be tested using this criterion to remove unrelated genes from Ground-2 if the p-value is over 0.05. This procedure reduces unnecessary root branches. The Chi-square test does respond to the question of whether or not a conditional gene can be associated with the target gene. However, it fails to response the question of whether or not it has a direct or indirect association due to the principle of 'guilt-by-association' that does not distinguish gene regulation from an indirect association (Childs, Davidson, & Buell, 2011).

Algorithm to Construct the Orchard Cube

Initially, we treated all input genes as potential target genes; hence, we placed all the input genes as *seeds* on the level of the first ground and divided them into N trees. N is the total number of input genes. Because the construction of the N trees is partitioned and can be run in parallel, this algorithm is for one tree only. The output result is an Orchard cube type of data structure that contains all precomputed measures, as discussed previously. The following steps outline the main algorithm to construct an Orchard cube.

1. With the input target gene i , we first apply the Chi-square test to test the *NULL* hypothesis between the target gene and all potential input genes. Non-related genes will be rejected, as discussed previously, so this yields a subset of all potential regulatory genes denoted as \hat{G} .
2. We ran through all potential regulatory genes, \hat{G} , and calculated the measures of the four dimensions (TT, TF, FT and FF) based on the immediately previous time step t for FBN or based on the temporal time steps from t to $t-m$ for TFBN where m refers to the maximum temporal decrement value, as discussed previously.
3. If the current level is lower than value $maxK$, which refers to the maximum underground level that the tree can penetrate, all potential regulatory genes except for the current gene and the genes that are in higher levels, denoted as G , will go to the next lower level.
4. In the next level, we repeat steps 2 and 3 until the current level is equal to $maxK$ or all the related genes (\hat{G}) are handled.
5. If all relevant genes (\hat{G}) are processed, or the current level is equal to $maxK$, then the cube is entirely constructed.

4.4. Inferences of Fundamental Boolean Networks

The second stage is to mine the fundamental Boolean functions from a cube type database structure. Figure 4-3 presents a schematic diagram of the fundamental Boolean network inferences.

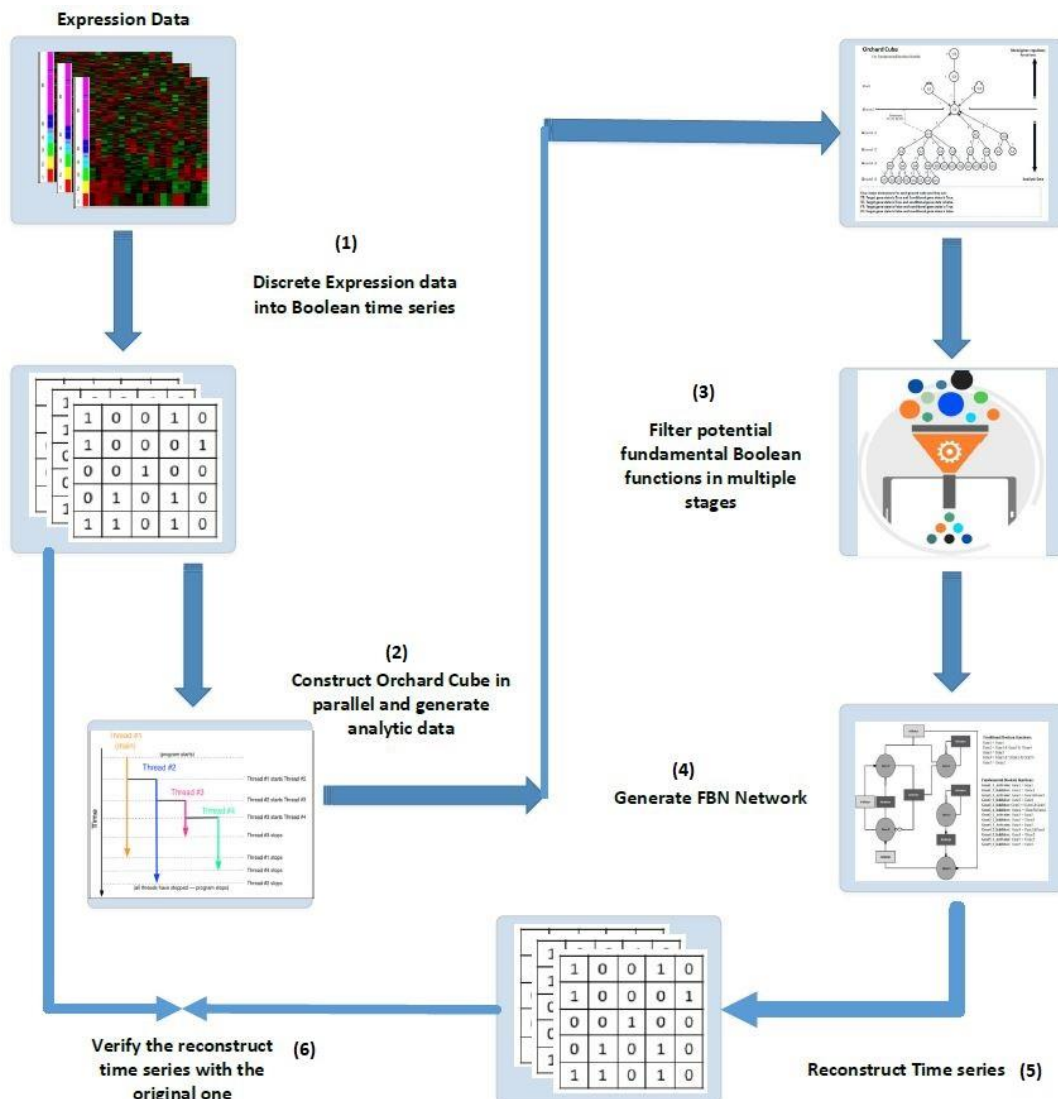


Figure 4-3 Schematic diagram of FBN modelling and network inferences: (1) separate expression data into Boolean time series; (2) build an Orchard cube in parallel to generate diagnostic data and store all precomputed measures; (3) infer potential regulatory rules for all target genes through the Orchard cube, based on some

criteria; (4) produce the fundamental Boolean network; (5) apply the generated network to renovate the input time series by providing the initial states of all original inputs; (6) validate the reconstructed time series with the original series to gain confidence in the results.

As outlined in Figure 4-3, to infer FBNs, all essential measures from a set of training data need to be calculated. The precomputed measures then persist in a cube type database structure so end users can mine fundamental Boolean functions from the cube. For TFBN, only the measures of the best temporal time step will be persisted. The excavated FBN / TFBN are then used to reconstruct a time series to validate the network by comparing the reconstructed time series data with the original training data.

By working with the proposed Orchard cube, we can extract fundamental Boolean functions in the GRNs based on the criteria listed below:

1. The conditional causality test value ought to be ≥ 1 .
2. Reject the functions if they are matched with the non-essential states, such as:

$$f_{a_j}^i(A\&B\&C) = f_{a_j}^i(A\&B\&!C) \text{ or } f_{a_j}^i(A\&B\&C) = f_{a_j}^i(A\&B\&!C)$$

where the definition of the essential Boolean state x_i must match the requirements of -

$$f(x_1, \dots, x_{i-1}, 0, x_{i+1}, \dots, x_n) \neq f(x_1, \dots, x_{i-1}, 1, x_{i+1}, \dots, x_n) \quad \text{for all}$$

$x_1, \dots, x_{i-1}, x_i, x_{i+1}, \dots, x_n$, where f is a Boolean function and x_i is a Boolean state (Faure et al., 2006) and the output of f is a Boolean value

3. The value of the confidence measure should be higher than a threshold ≤ 1 , e.g., 0.7 if the data contains noise.
4. Sort all Boolean functions remaining after step 4 by the error measure, the support measure and the number of genes input. The reason is to keep the rules that have more substantial support value with minimum errors.
5. The total number of fundamental Boolean functions for each type (activation or inhibition) of a gene should be limited to F_n based on different experimental requirements such as noise level, length of available time points etc. Nevertheless, in this study, we always take F_n to be 5.

4.5. Reconstruction of the Time Steps

The formulae of the proposed model and the theory of the Orchard cube represent a complete mechanism to compute the next gene state at time $t + 1$ by providing the gene state at time t . Consequently, we can reconstruct the time steps of any length by providing the initial gene state at time $t=0$. The following list gives the primary use of applying the model to reconstruct time steps:

1. Validate the reconstructed time series data against the original time series. If the functions are 100% accurate, we should be able to rebuild the time steps with the same initial states as in the original time series. If the functions are not 100% accurate, they might be caused by the model selection of fundamental Boolean modelling, i.e., synchronous (FBM) or asynchronous (TFBM), or the data are short time series.
2. Recreate the hidden layers between the observed time steps. The gaps between the observed time steps are quite significant in short time series data. We can apply the reconstructed time series to disclose the hidden layers by providing the initial Boolean state and continually generating the next time step based on the previous time step until the final generated time step is identical to the next time step observed. By providing the two observed states $S_{observed_1}$ and $S_{observed_2}$, we represent the time series data, as follows:

$$S_{reconstructed} = S_1, S_2 \dots S_k, S_{observed_1} = S_1 \text{ and } S_{observed_2} = S_k$$

The states between S_1 and S_k are then designated as missing time steps or hidden layers. This approach can be used to find attractors.

4.6. Summary

The concepts of the fundamental Boolean models (FBM and TFBM) discussed in chapter 3 is novel, and hence there is no existing methodology to infer the related networks. In this chapter, we proposed a novel methodology to infer the related networks. To achieve the goal, we studied the main measure matrix for network inference and then extended the concept of data mining technique of bottom-up computation (BUC) to a prefix tree type of cube; namely, Orchard cube to store all critical precomputed measures first and then search the best fundamental Boolean functions from the cube. This type of cube can distribute the computational costs to multiple computing nodes in a cloud computing environment because each branch can be calculated independently. Network inference from the proposed cube is a separate process that enables further development of scalable methods to infer genetic networks effectively and efficiently. An algorithm for searching fundamental Boolean functions from the proposed cube is provided and discussed in section 4.4. The following chapter demonstrates FBM with artificial mammalian cell cycle data.

Chapter 5: Cell Cycle Application

There are three main steps to validate the proposed Boolean model: (i) the selection of the updating schema for the model; (ii) the specification of the parameters of the model; and (iii) inferring the regulatory network. As deliberated briefly in the introduction, there is two main Boolean updating schema: synchronous and asynchronous, according to the treatment of time. For the sake of simplicity, we select the synchronous updating scheme to test the model. The parameter of the protein decay is set to 1 time step; the updating time step for each fundamental Boolean function is set to 1; the parameter of confidence of each subfunction is inferred using the method we proposed. To infer the fundamental Boolean network, we developed an unpublished R package, namely *FBNNet* (a prototype version of an R package for investigating the fundamental Boolean networks is available at https://github.com/clsdavid/FBNNet_Lincoln) to construct an orchard type cube and mine FBNs from the cube. The *FBNNet* tool can be used to mine the fundamental Boolean networks from the time series data, based on whether they are either FBM or TFBM models. The following paragraphs discuss the main experiment we conducted.

5.1. Experimental Design and Dataset

The experiments conducted and described here aim to demonstrate the concept of the new Boolean Model, i.e., the FBM. Figure 5-1 outlines the experimental design as a benchmark to compare the results generated via *BoolNet* (Mussel, Hopfensitz, &

Kestler, 2010) with those reconstructed from the new R package, *FBNet*. The *BoolNet* package was introduced in the tutorial by Hopfensitz et al. (2013) and the study of Ruz, Goles et al. (2014).

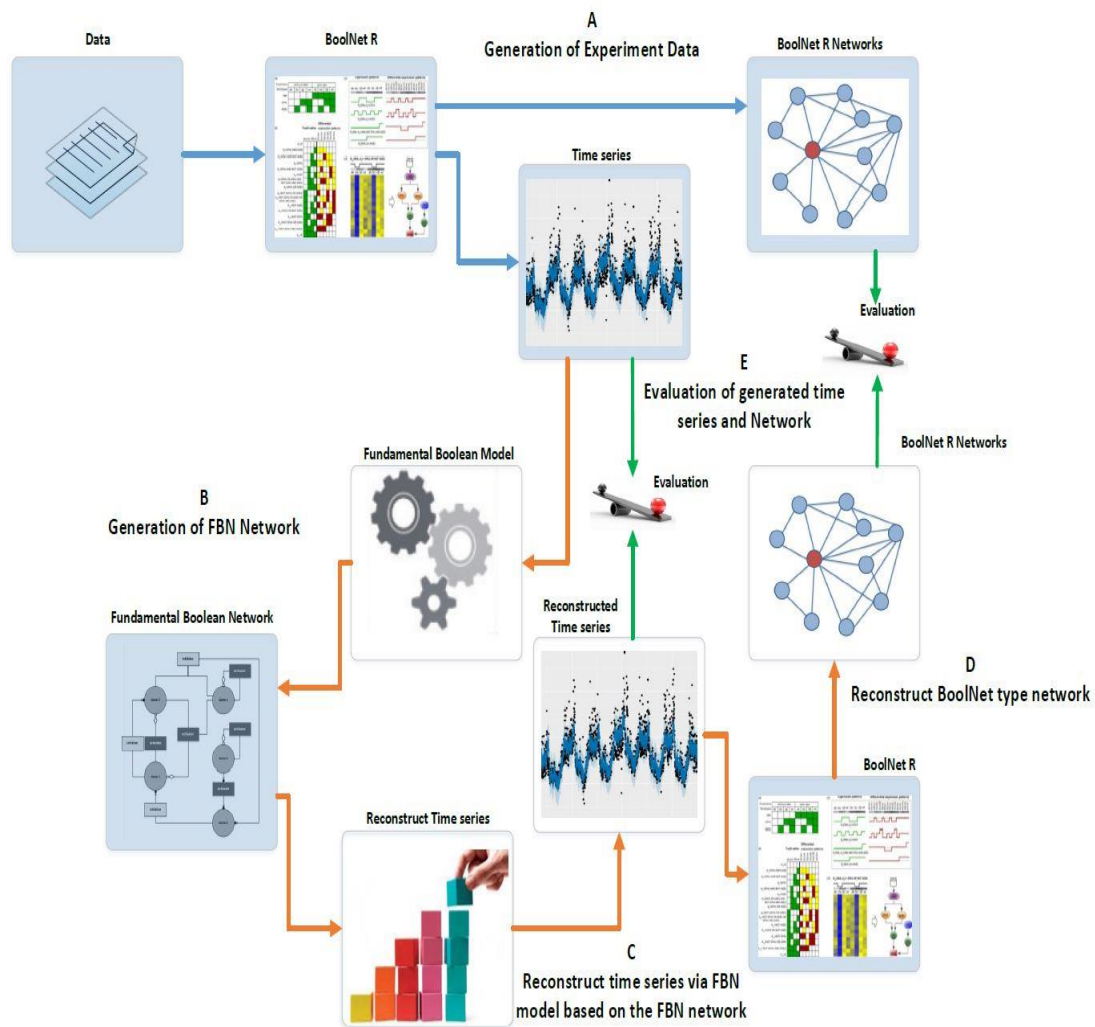


Figure 5-1 Design of the experiment to evaluate the fundamental Boolean network inference. The blue arrows denote the processes using BoolNet; the brown arrows denote the processes using our FBNet tools. The green arrows denote the evaluation process: (A) We apply the BoolNet script, loadNetwork.R, to load pre-defined networks from files and then produce time series and networks; (B) We apply the time series produced by BoolNet and the new R package, FBNet, to produce FBNs; (C) We rebuild the time series via the FBM; (D) To assess the FBM, we reconstruct the BoolNet type

network based on the rebuilt time series; and (E) We assess the FBN inference methods by comparing the generated time series with the original time series.

Many new algorithms can be tested using the simulated datasets resulting from the regulatory networks identified, and the outcomes can be compared with other known regulatory networks. In this thesis, we applied the mammalian cell cycle network, as listed in Figure 5-2, which was well established by Hopfensitz et al. (2013), to generate test data. The regulation of the cell cycle has been identified to lead to the replication of a cell, either in the synthesis or S phase. The cell division encompasses two daughter cells (mitosis, or M phase). The M phase itself comprises four different sub-phases (prophase, metaphase, anaphase, and telophase) (Faure et al., 2006). The S and M phases comprise two gap phases, specifically, G1 and G2 (Faure et al., 2006).

Initially, to create the experimental data, we used the command *loadNetwork*, provided by the R package *BoolNet*, to load the cell cycle network, which is specified in the text files: *cellcycle.txt*, as shown in Figure 5-2. Then we used the method *generateTimeSeries*, also provided by *BoolNet*, to produce 1024 noiseless sample data with 43 time steps, all using default settings, i.e., the parameter *type* is synchronous, the parameter *noiseLevel* is set to 0, and the parameter perturbations are 0. Each sample contains the same ten genes of the mammalian cell cycle: CycD, Rb, E2F, CycE, CycA, p27, Cdc20, Cdh1, UbcH10 and CycB; the same as the research led by Hopfensitz et al. (2013). This produced 1024 sample data (a combination of 2^{10} changes) that

are the dataset used for this experiment. Each sample comprises 43 time steps in sequence. Hence, the number of genes expressed in each sample is varied.

Boolean network with 10 genes

Involved genes:

CycD Rb E2F CycE CycA p27 Cdc20 Cdh1 Ubch10 CycB

Transition functions:

CycD = CycD

Rb = (! CycA & ! CycB & ! CycD & ! CycE) | (p27 & ! CycB & ! CycD)

E2F = (! Rb & ! CycA & ! CycB) | (p27 & ! Rb & ! CycB)

CycE = (E2F & ! Rb)

CycA = (E2F & ! Rb & ! Cdc20 & ! (Cdh1 & Ubch10)) | (CycA & ! Rb & ! Cdc20 & ! (Cdh1 & Ubch10))

p27 = (! CycD & ! CycE & ! CycA & ! CycB) | (p27 & ! (CycE & CycA) & ! CycB & ! CycD)

Cdc20 = CycB

Cdh1 = (! CycA & ! CycB) | (Cdc20) | (p27 & ! CycB)

Ubch10 = ! Cdh1 | (Cdh1 & Ubch10 & (Cdc20 | CycA | CycB))

CycB = ! Cdc20 & ! Cdh1

Figure 5-2 Known mammalian cell cycle networks provided by BoolNet

Since the proposed Boolean rule definition is novel and different from the conventional Boolean rules, i.e., instinctive rules vs compressed rules (not intuitive), as discussed in the introduction, this would not be a fair comparison with the traditional Boolean networks generated directly by other tools. Hence, the best way to estimate the generated FBNs is to apply them to rebuilding the time series data and then compare them with the training time series data. Under the synchronous model, if the inferred network is correct, it network should yield the same time series data if it is provided with the same initial states for each sample.

The assessment matrices for the time series comparison we adopted were: error rate (ER), accurate rate (AR), mismatched rate (MMR) and perfect matched rate (PMR).

The present definition of the matrix as follows:

$$ER = \frac{\sum_{i=1}^n \text{num of unmatched state per sample}(i)}{n}$$

$$AR = \frac{\sum_{i=1}^n \text{num of matched state per sample}(i)}{n} = 1 - ER$$

$$PMR = \frac{\text{Num of 100\% matched sample matrixes}}{n}$$

$$MMR = \frac{\text{Num of unmatched sample matrixes}}{n} = 1 - PMR$$

where n denotes the total number of samples. The time series data here refer to a list of matrices representing the states of samples, and each sample matrix contains Boolean gene states.

5.2. FBN of the Cell Cycle and its Validation

The extracted FBN for the cell cycle genes via the R package *FBNet* is shown in Table 5-1.

Table 5-1 Inferred FBN cell cycle network

<p>Fundamental Boolean Network with 10 genes</p> <p>Genes involved:</p> <p>CycD, Rb, E2F, CycE, CycA, p27, Cdc20, Cdh1, UbcH10, CycB</p> <p>Networks:</p> <p>Multiple Transition Functions for CycD with decay value=1:</p> <p>CycD_1_Activator: CycD = CycD (Confidence: 1, TimeStep: 1)</p> <p>CycD_1_Inhibitor: CycD = !CycD (Confidence: 1, TimeStep: 1)</p> <p>Multiple Transition Functions for Rb with decay value=1:</p> <p>Rb_1_Activator: Rb = !CycD&p27&!CycB (Confidence: 1, TimeStep: 1)</p> <p>Rb_2_Activator: Rb = !CycD&!CycE&!CycB&!CycA (Confidence: 1, TimeStep: 1)</p> <p>Rb_1_Inhibitor: Rb = CycD (Confidence: 1, TimeStep: 1)</p>
--

Rb_2_Inhibitor: $Rb = CycB$ (Confidence: 1, TimeStep: 1)

Rb_3_Inhibitor: $Rb = CycA \& !p27$ (Confidence: 1, TimeStep: 1)

Rb_4_Inhibitor: $Rb = CycE \& !p27$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for E2F with decay value=1:

E2F_1_Activator: $E2F = !Rb \& !CycA \& !CycB$ (Confidence: 1, TimeStep: 1)

E2F_2_Activator: $E2F = !Rb \& p27 \& !CycB$ (Confidence: 1, TimeStep: 1)

E2F_1_Inhibitor: $E2F = Rb$ (Confidence: 1, TimeStep: 1)

E2F_2_Inhibitor: $E2F = CycB$ (Confidence: 1, TimeStep: 1)

E2F_3_Inhibitor: $E2F = CycA \& !p27$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CycE with decay value=1:

CycE_1_Activator: $CycE = !Rb \& E2F$ (Confidence: 1, TimeStep: 1)

CycE_1_Inhibitor: $CycE = !E2F$ (Confidence: 1, TimeStep: 1)

CycE_2_Inhibitor: $CycE = Rb$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CycA with decay value=1:

CycA_1_Activator: $CycA = !Rb \& E2F \& !Cdc20 \& !UbcH10$ (Confidence: 1, TimeStep: 1)

CycA_2_Activator: $CycA = !Rb \& CycA \& !Cdc20 \& !UbcH10$ (Confidence: 1, TimeStep: 1)

CycA_3_Activator: $CycA = !Rb \& CycA \& !Cdc20 \& !Cdh1$ (Confidence: 1, TimeStep: 1)

CycA_4_Activator: $CycA = !Rb \& E2F \& !Cdc20 \& !Cdh1$ (Confidence: 1, TimeStep: 1)

CycA_1_Inhibitor: $CycA = Rb$ (Confidence: 1, TimeStep: 1)

CycA_2_Inhibitor: $CycA = Cdc20$ (Confidence: 1, TimeStep: 1)

CycA_3_Inhibitor: $CycA = !E2F \& !CycA$ (Confidence: 1, TimeStep: 1)

CycA_4_Inhibitor: $CycA = Cdh1 \& UbcH10$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for p27 with decay value=1:

p27_1_Activator: $p27 = !CycD \& !CycE \& !CycB \& !CycA$ (Confidence: 1, TimeStep: 1)

p27_2_Activator: $p27 = !CycD \& !CycA \& !CycB \& p27$ (Confidence: 1, TimeStep: 1)

p27_3_Activator: $p27 = !CycD \& !CycE \& !CycB \& p27$ (Confidence: 1, TimeStep: 1)

p27_1_Inhibitor: $p27 = CycD$ (Confidence: 1, TimeStep: 1)

p27_2_Inhibitor: $p27 = CycB$ (Confidence: 1, TimeStep: 1)

p27_3_Inhibitor: $p27 = \text{CycA} \& !p27$ (Confidence: 1, TimeStep: 1)

p27_4_Inhibitor: $p27 = \text{CycE} \& !p27$ (Confidence: 1, TimeStep: 1)

p27_5_Inhibitor: $p27 = \text{CycE} \& \text{CycA}$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for Cdc20 with decay value=1:

Cdc20_1_Activator: $\text{Cdc20} = \text{CycB}$ (Confidence: 1, TimeStep: 1)

Cdc20_2_Inhibitor: $\text{Cdc20} = !\text{CycB}$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for Cdh1 with decay value=1:

Cdh1_1_Activator: $\text{Cdh1} = \text{Cdc20}$ (Confidence: 1, TimeStep: 1)

Cdh1_2_Activator: $\text{Cdh1} = !\text{CycA} \& !\text{CycB}$ (Confidence: 1, TimeStep: 1)

Cdh1_3_Activator: $\text{Cdh1} = p27 \& !\text{CycB}$ (Confidence: 1, TimeStep: 1)

Cdh1_1_Inhibitor: $\text{Cdh1} = !\text{Cdc20} \& \text{CycB}$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for UbcH10 with decay value=1:

UbcH10_1_Activator: $\text{UbcH10} = !\text{Cdh1}$ (Confidence: 1, TimeStep: 1)

UbcH10_2_Activator: $\text{UbcH10} = \text{Cdc20} \& \text{UbcH10}$ (Confidence: 1, TimeStep: 1)

UbcH10_3_Activator: $\text{UbcH10} = \text{UbcH10} \& \text{CycB}$ (Confidence: 1, TimeStep: 1)

UbcH10_4_Activator: $\text{UbcH10} = \text{CycA} \& \text{UbcH10}$ (Confidence: 1, TimeStep: 1)

UbcH10_1_Inhibitor: $\text{UbcH10} = \text{Cdh1} \& !\text{UbcH10}$ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CycB with decay value=1:

CycB_1_Activator: $\text{CycB} = !\text{Cdc20} \& !\text{Cdh1}$ (Confidence: 1, TimeStep: 1)

CycB_1_Inhibitor: $\text{CycB} = \text{Cdh1}$ (Confidence: 1, TimeStep: 1)

CycB_2_Inhibitor: $\text{CycB} = \text{Cdc20}$ (Confidence: 1, TimeStep: 1)

As shown in Table 5-1, three primary parameters are bound to this novel FBN: confidence, protein decay and time step. The time step was then mined through the method discussed in chapter 4 as the best temporal value for this experiment. As the test data were generated with time step 1, the value of the parameter *timestep*, as shown in Table 5-1, correctly reflected the test data. The protein decay was manually

configured to 1 to match the way we produced the test data via the method `generateTimeSeries` of *BoolNet*. This function does not offer a configurable parameter for protein decay, but it is suspected it might have a default value of 1 (time step) fixed inside its logic.

A noteworthy difference from the existing Boolean models is that the inferred FBN intuitively separates the Boolean rules of the mammalian cell cycle into the fields of activation and inhibition, as shown in Table 5-1. At the same time, every gene could be regulated by multiple activations or inhibition rules. The results in Table 5-1 illustrate that the uncertainty of the process can be incorporated into the model. All FBM functions have a confidence parameter of 1, and the results presented are incredibly accurate. To validate the results, we applied the initial states from the training time series dataset to rebuild the same sized data set using the novel concept of FBM under the synchronous updating schema, which is the same as the schema used to generate the training dataset. The redeveloped dataset was then compared with the training time series dataset.

As shown in Table 5-2, all reconstructed time series data from the mammalian cell cycle network are identical to the training time series data with a 100% match for both AR and PMR. This result indicates that all regenerated time series data are matched with the training time series dataset. Therefore, we have confidence that the proposed FBM is an alternative way to represent the mammalian cell cycle network

but with more information to draw insights into the activation and inhibition pathway of the mammalian cell cycle network.

Table 5-2 Experimental results for reconstructed time series data

Network	Number of samples	Number of time steps	ER	AR	PMR	MMR
Cell cycle	1024	43	0	100%	100%	0

Regarding FBNs, Figure 5-3 presents the regulatory network graph of the cell cycle.

The graph is generated by integrating the inferred FBNs with the R package visNetwork (<http://datascorm-open.github.io/visNetwork/>).

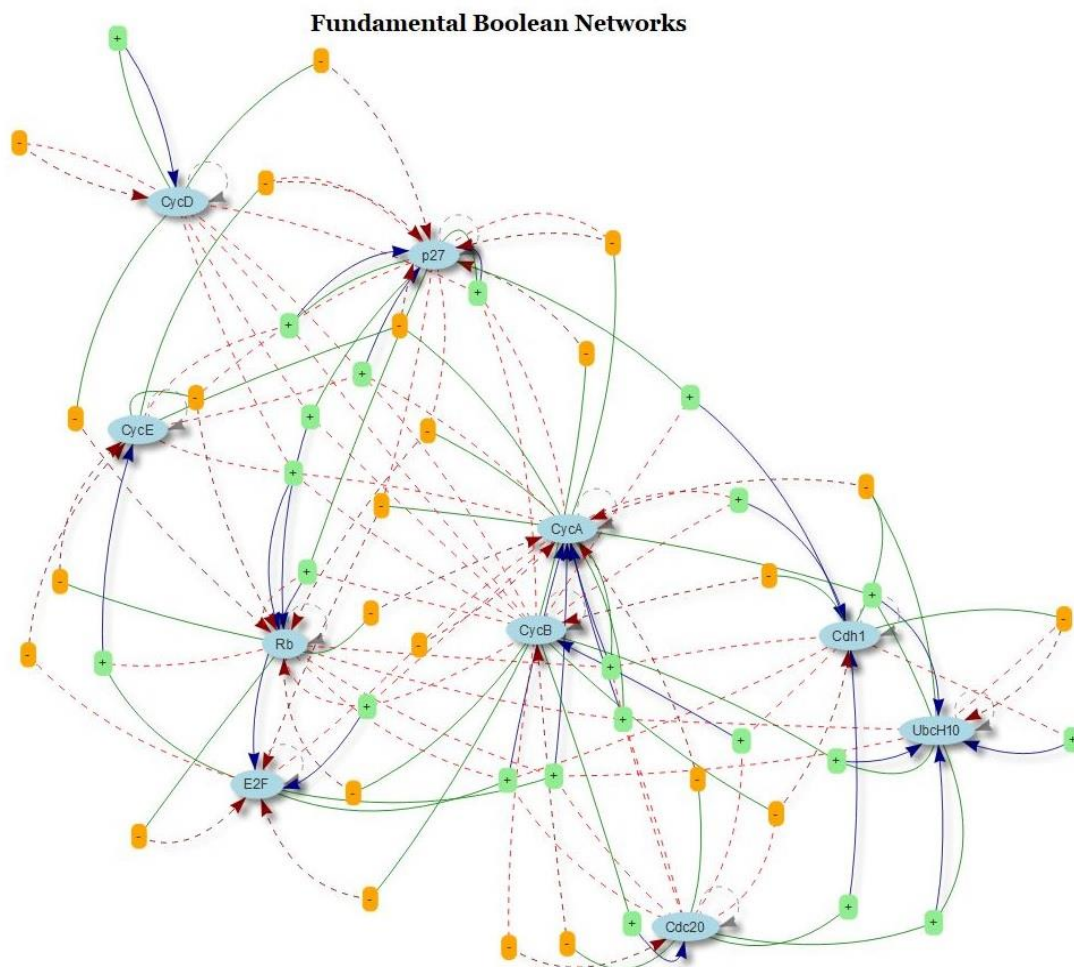


Figure 5-3 Cell Cycle FBN (FBNett_ALL), a static model. The legend refers to Figure

1.

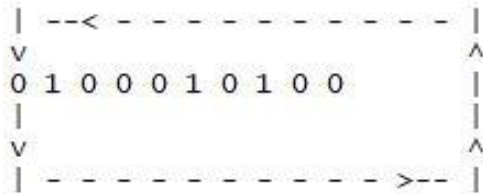
As shown in Figure 5-3, the internal relationships between the genes have been demonstrated. Hence, we can discover how these genes activated and inhibited other genes dynamically by drawing connections between the input genes and the target genes. The FBN of cell cycle comprises three types of network influences in the domains of gene activation, gene inhibition and protein decay.

5.3. Attractors

Attractors refer to the recurrent cycles of the states (Hopfensitz et al., 2013) and are of particular interest in Boolean modelling. Once a network reaches an attractor, it is entrapped in a cycle that repeats until an external perturbation happens to change some of the production of the essential genes of the attractor to let the network come out from entrapment. Using the most straightforward synchronous updating schema, yielded two attractors, as shown in Figure 5-4. One is a simple attractor, and the other is a cycle attractor. The attractors correspond to the findings that have been reported by (Faure et al., 2006; Hopfensitz et al., 2013). Therefore, the FBN of the cell cycle can construct the same attractors as in the other Boolean models.

Discovered Attractors via Fundamental Boolean Model :
 Genes are encoded in the following order::
 CycD Rb E2F Cyce Cyca p27 Cdc20 Cdh1 Ubch10 CycB:

Attractor 1 is a simple attractor consisting of 1 state(s):



Attractor 2 is a complex attractor consisting of 7 state(s):

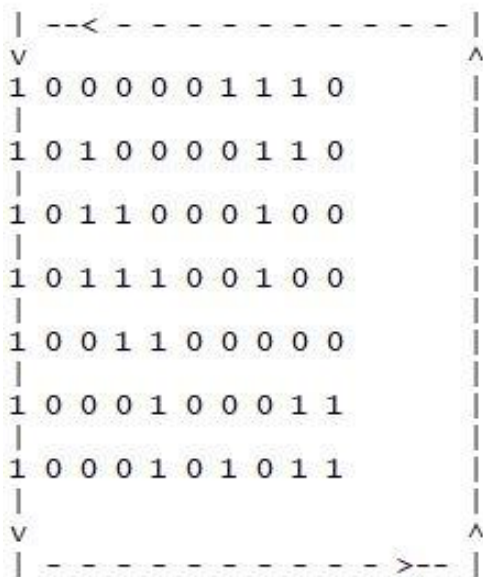


Figure 5-4 Synchronous attractors of the cell cycle fundamental Boolean model

The first attractor, as shown in Figure 5-4, is a simple attractor with only Rb, p27 and Cdh1 active and this is related to phase G0 or cell quiescence (Faure et al., 2006). CycD characterises the whole cdk4/6-Cyclin D complex, and cdk4/6 refers to cyclin-dependent kinase (cdk) partners.

One advantage of the proposed model was its dynamic networks, which provided a complete trajectory of gene activation, inhibition and protein decay. Figure 5-5 demonstrates the dynamic trajectories of attractor 2, which explicitly display the

internal mechanisms of gene regulation in the domains of gene activation, inhibition and protein decay. The existence of CycD leads to seven other stable dynamical cycles. Each cycle is constructed from a sequence of seven successive states (attractor 2). The secreted pathways of attractor 2 were not explicit in any conventional Boolean model or related networks.

Nevertheless, with the proposed model and type of networks, the pathways of attractor 2 have been explicitly demonstrated. For example, in Figure 5-5, CycD suppressed Rb and p27. Rb, in turn, then induced E2F, a family of dimeric transcription factors. As a result, E2F enhanced CycE and CycA. Activated CycE and CycA then continually maintained the inhibition of Rb and p27.

Furthermore, Cdh1 was another crucial element, it an activator delegating APC, a viral E3 ubiquitin ligase. The activated Cdh1 dissociated CycB directly and kept inhibiting Ubch10.

Moreover, Rb phosphorylated E2F, CycA, and CycE, and the promotion of p27 activated Cdh1. Rb continued to be enhanced without interruption by CycA, CycB, CycE, and CycD. The Ubch10 at time step 3 was repressed because of protein decay (see the dashed grey arrow line) because none of its activation and inhibition functions have an impact on it.

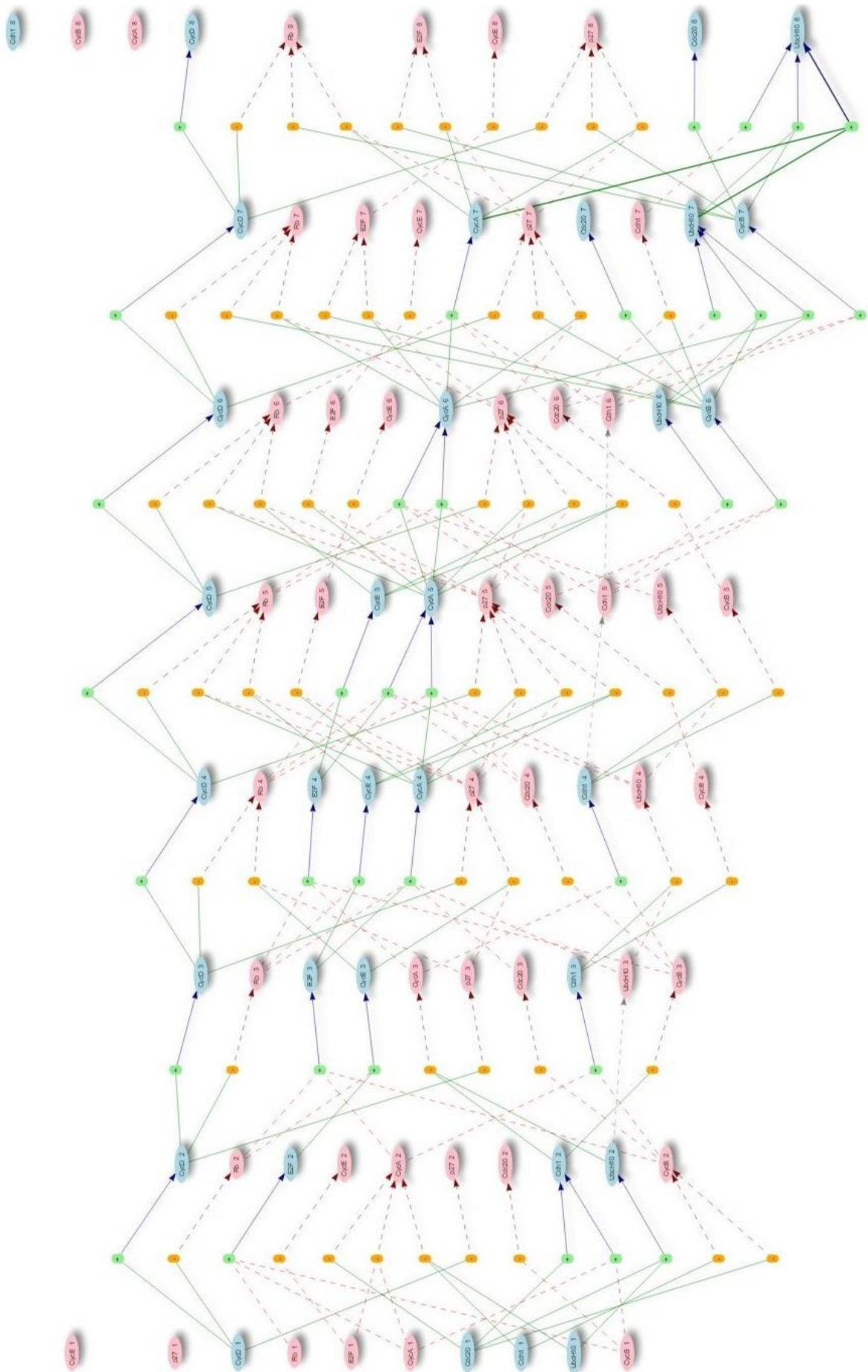


Figure 5-5 Dynamic trajectories of attractor 2. The numbers designate the time step that the gene was located. The light blue elliptical icons denote genes; the orange icons denote inhibition functions, and the light green icons denote activation functions. The dark blue arrows denote activation; the dark red arrows denote inhibition, and the grey arrows denote protein decay. The legend refers to Figure 1.

5.4. Conclusion and Summary

As revealed by the demonstration of the proposed model with the mammalian cell cycle, we have proved that we can apply the proposed Orchard cube to infer the GRNs of the mammalian cell cycle. This outcome confirmed the hypothesis that if the network inferred was 100% correct; the reconstructed time series should be identical to the original training dataset. Nevertheless, this assumption was based on the degree of completeness of the initial training dataset under the synchronous Boolean schema.

The cell cycle FBN disclosed the internal gene activation, inhibition and protein decay mechanisms. The generated new Boolean cell cycle network was very different from any other Boolean network, but it still demonstrated the same attractors as documented in the study of Faure et al. (2006). This means that the proposed Boolean model is a novel extension of other Boolean models. When compared with the traditional Boolean cell cycle network as in the study of Hopfensitz et al. (2013), our network divides a complex rule into multiple rules under the domains of activation and inhibition. It delivers more insights into the dynamics of the pathways. Under the

fundamental Boolean model, a node was connected with multiple functions divided into the two main kinds of gene regulation (activation and inhibition type). Besides, protein decay was also considered as a connection of gene transitions; hence, the FBN contained three types of connections: activation, inhibition and protein decay (as shown in Figure 5-5). This new feature can enable scientists to develop pharmaceutical agents by examining the related fundamental Boolean network and simulating perturbations due to drugs.

We also demonstrated the dynamic trajectories for attractor 2. The main benefit of the proposed novel Boolean model was that it revealed connections in the fields of activation, inhibition, and protein decay pathways that can facilitate scientists in understanding intrinsic genetic regulations. One disadvantage was that the FBN might contain too many links. The novel Boolean network might contain abundant rules, but all these functions were data-driven with outstanding confidence values. Therefore, the FBN of the cell cycle was fine-grained, and the original Boolean network, as shown in Figure 5-2, was coarse-grained. We could also limit the number of rules per type (activator and inhibitor). However, reducing the number of fundamental Boolean functions per type might decrease the correctness of the inferred network.

The current version of *FBNet* was developed as a prototype for the proposed FBM using pure R language without any performance optimisation improvements. Hence, to generate the experimental results required approximately 200 seconds with parallel computing and 530 seconds without parallel computing. However, these

figures were recorded from using an earlier version of *FBNNet*, and its performance might have been improved. The machine we used for the experiment was a laptop Acer™ Aspire V 17 Nitro model. Notably, the performance of these packages was well understood, and they may not provide real parallel power for computing as good as C or C++, although they were good enough to demonstrate the idea of the proposed novel Boolean model. *BoolNet* was implemented in C to improve its performance in constructing the cell cycle network and, hence, it was faster than the current version of *FBNNet*.

Moreover, our method can be used to derive the intuitive activation and inhibition pathways using the proposed Orchard cube methodology and; hence, may require more computational time, i.e. the total time mentioned included the time to construct the cube and the time to infer the networks from the cube. The planned Orchard cube was also used to maintain all precomputed measures in a database for all possible fundamental Boolean functions in case we needed to mine FBN from short time series data. Hence, it used a different technology from *BoolNet* and; therefore, it was not necessary to compare the performance between *BoolNet* and *FBNNet*.

Finally, we proposed a method to reconstruct any missing time steps by estimating all fundamental Boolean functions' *TRUE* or *FALSE* values that affected the target genes by verifying the input states to be corresponding to the requirements of the functions. The time interval between time steps is a parameter of the proposed model. It should reflect the assumption that all related genes should have finished their biological

reactions; for instance, transcription from DNA to mRNA, and translation from mRNA to protein. If we set the time interval for all genes, then the FBM, was a synchronic Boolean model. When all genes have their time interval defined, the FBM was then an asynchronous Boolean model. The proposed Boolean model then can be used to reconstruct the missing time steps under synchronic Boolean modelling. However, we are not able to use the reconstructed time series to validate the generated output correctly under the asynchronous Boolean modelling because the results from the asynchronous Boolean model were non-deterministic.

Chapter 6: Application for Leukaemia

To understand normal genetic problems scientists reconstruct the dynamics, as represented by time and discrete state transition systems, to gain insights into the functioning of cell systems (Ay & Arnosti, 2011; Hood, 2013; Lee & Tzou, 2009; Y. Wang et al., 2011). These dynamics can be used to simulate the perturbations of new drugs *in silico* to reduce the potential risks of administering drugs to humans. Hence, inferring gene networks from time series data in clinic settings using discrete modelling is a promising approach in cancer research.

In the previous chapter, we demonstrated how to apply FBM to artificial cell cycle data and presented the dynamics of cell cycle networks. This demonstration proved that the concept of the novel Boolean model (FBM) could be applied to genetic related time series data. The data used in the previous chapter were artificial and produced by the R function, provided by *BoolNet*, based on previous biological knowledge of cell cycle networks (Hopfensitz et al., 2013).

In this chapter, we demonstrate how to apply FBM, primarily TFBM, on a real dataset (leukaemia data), which were downloaded from the GenBank. The link of the GenBank website is <https://www.ncbi.nlm.nih.gov/genbank/>. The previous biological knowledge of leukaemia is not required for this demonstration, although previous biological knowledge still played an essential role in inferring gene networks (Chaiboonchoe, 2010).

6.1. Introduction

Before the experiment conducted by Schmidt et al. (2006), investigations had led to some conflicting attractive hypotheses, which have not yet been tested in a clinical setting (Schmidt et al., 2006). Hereafter, Schmidt et al. (2006) generated 13 comparative whole-genome expression profiles (purified at three time points) using lymphoblasts from 13 GC-sensitive children all under therapy for acute lymphoblastic leukaemia (ALL) (Schmidt et al., 2006). As a consequence, a substantially complete list of GC-regulated candidate genes in clinical settings and experimental systems has been generated to facilitate immediate analysis of any gene for its potential significance to GC-induced apoptosis (Schmidt et al., 2006). Schmidt's (2006) study identified a small number of novel candidate genes, including a key regulator of glucose metabolism (PFKFB2), a putative transcription factor (ZBTB16) and a protein kinase implicated in cell cycle regulation (SNF1LK); however, this study was inconsistent with most model-based hypotheses such as expression profiling studies with sub-genome microarrays in model systems (Schmidt et al., 2006).

The data generated by Schmidt (2006) in a clinical setting were a short time series, but they were still valuable. Researchers have continually analysed the data and proposed novel hypotheses or questions, such as the study conducted by (Chaiboonchoe, Samarasinghe, & Kulasiri, 2009), in which more novel glucocorticoid-regulated genes were identified through inferred GC-regulation networks (Chaiboonchoe, 2010; Chaiboonchoe et al., 2009). The newly identified genes may pave the way to develop

new chemotherapy drugs that have fewer side effects. However, the models Chaiboonchoe (2009) applied were based on the emerging clustering methods, such as self-organising maps (SOMs) (Haykin, 1999; Jaakko, 1996; Yin, 2008), emergent self-organising maps (ESOM) (Ultsch & Herrmann, 2005), the short time series expression miner (STEM) (Ernst & Bar-Joseph, 2006) and fuzzy clustering by local approximation of membership (FLAME) (Cruz, Vieira, & Vinga, 2015) and the networks Chaiboonchoe (2009) presented were based on previous network knowledge. In this chapter, we proposed to reanalyse this valuable data with the fundamental Boolean modelling, a data-driven model that we discussed in chapters 3 and 4.

6.2. Methods

6.2.1 Dataset and Pre-processes

Schmidt et al. (2006) generated 13 comparative whole-genome expression profiles (purified at three time points) using lymphoblasts from 13 GC-sensitive children under therapy for glucocorticoid-resistant ALL (Carlet et al., 2010; Schmidt et al., 2006). Raw data (GEO Assession code: GSE2677 and GSE2842) in the form of CELL files were downloaded online from the website of the National Centre for Biotechnology Information (NCBI) (<http://www.ncbi.nlm.nih.gov/geo/>) and were provided by Schmidt et al. (2006). The data contained gene expression measurements for 13 samples, and each sample has three time points: 0 hours (before GC treatment), 6/8 hours (after GC treatment) and 24 hours (after GC treatment). Among the 13 samples,

three samples from T-lineage (T-ALL) patients, and the rest were B-lineage (B-ALL) patients.

The common pre-process of analysing gene expression data involves data normalisation, selection of differentially expressed genes, and data discretisation. In section 2.3, we discuss the methods of normalisation briefly. Regarding gene expression, in general, there are two main basic patterns which are called underexpression (down-regulation) and overexpression (up-regulation). Overexpressed genes are genes that have higher expression values when two samples are compared; for example, cancer (target) and healthy. On the other hand, underexpressed genes have lower expression values in the target than in reference samples (Dubitzky, Granzow, Downes, & Berrar, 2003). Commonly, a gene with more than twofold changes is considered significant or differentially expressed.

This study selected R software as the platform to analyse genetic networks. R (<http://www.r-project.org>) is an open-source platform for statistical computing developed by the Bioconductor project (<http://www.bioconductor.org>) to analyse genomic data based on the R programming language. We applied the following tools to conduct the experiments described in this chapter.

- FBNet, unpublished R package, version 2.0. A package implemented explicitly for the fundamental Boolean model (FBM) and the temporal fundamental Boolean model (TFBM), as proposed and discussed in this thesis.

- BoolNET, an R package for analysing conventional Boolean networks, documented in (Mussel et al., 2010).
- Robust multi-array average (RMA) is a method that converts probe level data (CEL files) into a gene expression measure.
- GeneChip RMA (GCRMA) is an improvement from RMA that uses the probe sequence information for background correction and is a bias-corrected (Wu & Irizarry, 2004).
- Gene Annotation via DAVID Bioinformatic Database (Huang, Sherman, & Lempicki, 2009a, 2009b).

Inferring genetic networks from the whole genome is usually very time consuming and very difficult to achieve. The biologist or pharmacist needs a small subgroup of differentially expressed genes for further experiments. The original dataset contains more than 30000 genes/proteins; hence, we need to identify the differentially expressed genes and use these genes to construct FBN networks. Since the time and scope of this study is relatively limited, we only pick a few critical genes from the inferred fundamental Boolean networks to discuss.

6.2.2 Experimental Methods

First, we downloaded the CEL files, which are data files created by Affymetrix DNA microarray image analysis software, from the Genbank, which is the NIH genetic sequence database (Nucleic-Acids-Research, 2013), and normalised them using RMA

and GCRMA, respectively. Secondly, we computed the differentially expressed genes for the 13 samples (three T-All samples and 10 B-All samples) with the same criteria as Schmidt et al. (2006), i.e., the cutoff threshold is 0.7, and the majority value is set to 6 out of 13 samples. Three different methodologies on computing the differentially expressed genes have been conducted:

- Re-analyse the M-values and E-values provided by Schmidt (Schmidt et al., 2006) where the M-values were denoted as regulation values, and the E-values were denoted as normalised expression values.
- Normalise the data using RMA.
- Normalise the data using GCRMA.

The cutoff point represented the threshold for fold changes. For example, a cutoff of 1 means M values of ≥ 1 , which represent a two-fold regulation; a cutoff 0.7 means a 1.4 fold regulation; and a cutoff of 2 means a four-fold regulation. The majority value means the fold regulation happens in at least six out of 13 samples.

Apart from these experiments, we also computed the differentially expressed genes from all 10 B-All samples and all three T-All samples. The common genes from these three methods will be the input target genes for the FBNNet process. The training data for FBNNet process were the data normalised by GCRMA. The training data were filtered by the target genes. All duplicated genes were aggregated by *mean*, an R function to average the data. The training data for constructing an Orchard cube contained the data combined from the GEO Assession codes: GSE2677 and GSE2842.

The data for computing differentially expressed genes are from GEO Assession code: GSE2677 and contained data from 13 samples.

Section 2.2 discussed how to calculate the differentially expressed genes. It was straight forward to normalise the raw data using `affy.rma` and `affy.gcrma`, provided by the Bioconductor project (Bioconductor, 2020) via R. Constructing an Orchard cube based on the training data was similar to the way used in the previous chapter for the cell cycle data. Different from the experiments conducted on cell cycle data, where $maxK$ was 4, we choose $maxK = 3$ for the sake of simplicity, where $maxK$ defined the maximum level of the combinations of genes that could affect the target gene. The maximum temporal value (m), which is a value that indicated how many times were the steps required to complete the regulation process of a fundamental Boolean function, was 2.

6.3. Results and Discussion

Table 6-1 presents the results of differentially expressed genes that were identified by the three different methods. The figures of Re-analysis M-values were identical to the study conducted by (Chaiboonchoe, 2010) because we used the same criteria to select the differentially expressed genes.

Table 6-1 GC-regulated differentially expressed genes, $M \geq 0.7$ (1.4 fold regulation),

6/13 (majority)

	0 - 6/8 h		6/8 – 24 h		0 – 24 h		Total Unique
	Activated	Inhibited	Activated	Inhibited	Activated	Inhibited	
Re-analysis M-values	58(44)	66(52)	63(24)	61(49)	212(105)	258(193)	718 (348)
Analysis with R 3.6.1 affy::rma	59(46)	78(62)	61(22)	66(52)	223(115)	268(202)	755(378)
Analysis with R 3.6.1 affy::gcrma	117(93)	153(110)	156(59)	121(97)	427(220)	446(346)	1420(665)

Interestingly, the results from RMA under R version 3.6.2 were different from the Re-analysis M-value. The M-value was provided by (Schmidt et al., 2006) who also applied RMA (provided by an old version of R in 2006) to compute the M-values, as discussed in the literature review. The results from GCRMA contained more differentially expressed genes than the other two methods. Figure 6-1 visualises the common and different figures using a Venn diagram.

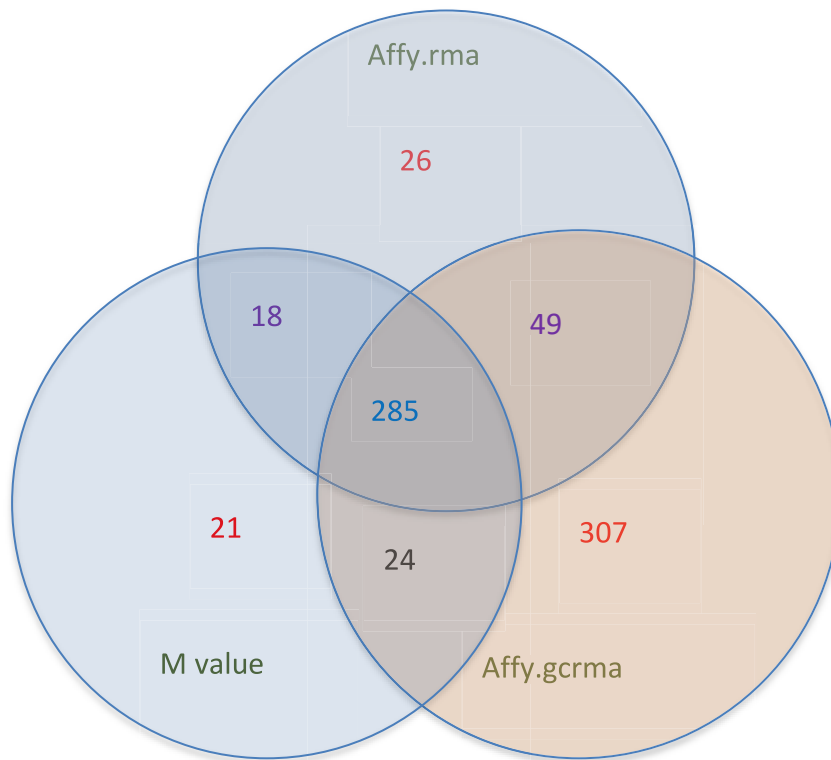


Figure 6-1 Venn diagram of the differentially expressed genes (glucocorticoid induced) across all time points.

We were interested in the genes set in the intersections of the results with the three methods. Hence, we constructed the FBM cube and extracted the fundamental Boolean networks based on the most common genes, i.e., 285 genes. Among the most common genes: 34 were induced for 0-6 h; 40 were repressed for 0-6 h; 87 were induced for 0-24 h; 180 were repressed for 0-24 h; 17 were induced for 6-24 h, and 34 were repressed for 6-24 h. Four of genes across all periods were induced, and five across all three time spans were repressed. Table 6-2 list the common genes split by periods and types of sample (T-ALL and B-ALL).

Table 6-2 Common glucocorticoid induced genes

Common Results	Genes
Common Genes (285)	<p>MS4A4A, CLN8, SNX10, RAB31, SERPINA1, LILRA1, LILRB2, PFKFB2, SOCS1, LGALS3, SIK1, SLA, FCGR3B, FGL2, BTNL9, RBMS3, DPEP1, MNDA, FKBP5, DDIT4, WFS1, S100A11, ZBTB16, P2RY14, GSN, EPPK1, ZFP36L2, FCER1G, FGR, IRAK3, PPBP, MYRIP, PIK3IP1, KIF26A, NUF2, ARPP21, ABHD17B, ASPM, PBK, KIF20A, CEP55, PDE4B, HMMR, CDKN3, FUS, AURKA, CENPF, POU4F1, PRR11, KIF14, CENPE, CENPA, NEK2, TTK, KIF11, GIMAP4, DLGAP5, PTTG1, UBE2C, CDC20, MKI67, CCNB2, BIRC5, FAM72C, GBP4, KIF23, NPCDR1, CCNL1, LOC728175, CLEC2B, BCL10, TOP2A, TENM4, CCNB1, SNORA21, MDM2, DEPDC1, DEPDC1B, GIMAP7, TBXA2R, STAB1, MIR4683, SMIM3, SLC22A23, TMEM100, LOC100130872, SESN1, TMEM2, IFNGR1, BIRC3, P2RX5, CDC42EP3, METTL7A, IL18RAP, CCR1, SNX29P2, LOC100505650, RHOBTB3, BCL2L11, SNTB2, MS4A1, SOS1, TNFSF8, RNASET2, CD53, LY96, SCML4, LOC285097, ITGAM, DEFA1, ISG20, DFNA5, MTSS1, IL6ST, MIR6845, NEAT1, KLF9, TXNIP, IL18R1, HBB, CELF2, HBG1, S100A8, MPV17L, NEDD9, FGD2, BTG1, TUBA4A, RPS6KA2, DENND3, IL27RA, MS4A7, SMAP2, RASSF4, BMF, ELL2, TARSL2, SEMA4D, ITGB2-AS1, PPP1R16B, CPM, ITPKB, IL1B, MCM10, CDCA3, UBE2T, TCF19, BRIP1, KIF18A, KCNK12, HELLS, E2F8, NUSAP1, ECT2, CENPN, SHCBP1, POLQ, KIF15, B3GNT2, TIPIN, DSCC1, CENPU, TNFRSF21, ATAD2, HJURP, NCAPG, DTL, KIF4A, CKAP2, MSH6, MCM7, LEF1, AKAP12, SKP2, TPX2, CDT1, RRM2, BUB1, KIF2C, MDK, ANP32E, CDK1, F13A1, BYSL, HIST4H4, CCNA2, MND1, EGR1, PRPS2, SNORD3B-1, HRK, CHEK1, WDR76, IGLL1, RAG1, RCC1, POLE2, MCM4, BCAT1, STIL, PSPH, PCNA, RAD51, PLK4, TRIP13, MELK, FEN1, WDHD1, BRCA1, SMC2, WASF1, CDC45, RFC3, ZWINT, CDC6, BUB1B, MAD2L1, CCDC86, TIMELESS, OIP5, TYMS, DHFR, FOXM1, KIAA0101, TK1, FABP5, TRIB1, CKAP2L, CKS1B, CENPH, MTHFD2, NME1, PPIF, EMP1, GGH, PAICS, GINS1, AURKB, ASF1B, GINS2, CENPK, IQGAP3, ZNF367, PTP4A1, ANLN, C5orf24, SUV39H2, SELENOI, RBM14,</p>

	HSP90AB1, FH, RAD51AP1, DHX9, TMEM97, NCAPH, FANCI, APITD1-CORT, RAG2, MCM5, KIF18B, CDCA5, ARRDC3, SQLE, LOC100996643, CCDC34, RMI2, CENPV, CDCA2, CENPW, PHF19, E2F7, KNL1, C4orf46, CDK6, CRNDE, SERPINB9, PRDM1, ID2, GVINP1, MIR8071-1, CCNE2, ID3, IGLC1, LYZ, IGH
Common Genes from B-ALL only via affy.rma (213/285), for 6/10 samples	FCER1G, WFS1, SCML4, GSN, SERPINA1, DDIT4, SLA, FCGR3B, MNDA, ZBTB16, DPEP1, P2RY14, LILRB2, SIK1, FGR, LGALS3, LILRA1, IRAK3, MYRIP, FGD2, RAB31, SNX10, CLN8, MS4A4A, PFKFB2, FGL2, BTNL9, KIF26A, EPPK1, RBMS3, TBXA2R, LOC728175, CDKN3, TOP2A, CCNB2, CDC20, UBE2C, CDK1, CCNA2, PDE4B, DLGAP5, KIF23, TTK, CENPA, CENPE, RAG1, POU4F1, HMMR, CENPF, AURKA, BUB1, MKI67, TENM4, CCNB1, MDM2, CEP55, NCAPG, KIF20A, GIMAP4, ASPM, DEPDC1, UBE2T, NUF2, FAM72C, GIMAP7, GBP4, NEDD9, ZFP36L2, IFNGR1, KLF9, STAB1, FKBP5, CCR1, ITGAM, LY96, IL18R1, IL18RAP, HBB, CDC42EP3, SOCS1, P2RX5, IL6ST, DENND3, SNX29P2, SESN1, LOC100130872, TMEM100, PIK3IP1, IL27RA, LOC100505650, SLC22A23, MS4A7, MIR4683, MIR6845, RASSF4, ELL2, TARSL2, ITGB2-AS1, RNASET2, PPP1R16B, NEAT1, CPM, TNFSF8, CELF2, ANLN, C5orf24, TK1, SUV39H2, TYMS, MAD2L1, HSP90AB1, PAICS, PCNA, PPIF, NME1, EGR1, RRM2, CKS1B, BIRC5, TRIB1, FABP5, KIAA0101, DHFR, PTTG1, GGH, BYSL, BUB1B, CDC6, ZWINT, TRIP13, CDC45, RFC3, RAD51AP1, WASF1, SMC2, KIF11, NEK2, WDHD1, MELK, PLK4, RAD51, IL1B, STIL, CHEK1, POLE2, HIST4H4, GINS1, KIF14, RCC1, IGLL1, MCM7, MDK, SQLE, KIF2C, AURKB, TPX2, LEF1, MSH6, MCM4, TMEM97, NCAPH, FANCI, APITD1-CORT, OIP5, BCAT1, TNFRSF21, RAG2, NUSAP1, ASF1B, CKAP2, KIF4A, DTL, HJURP, ATAD2, CENPU, DSCC1, PBK, TIPIN, KIF15, POLQ, CENPN, ECT2, E2F8, HELLS, MCM10, KIF18A, GINS2, BRIP1, KIF18B, CENPK, TCF19, CDCA3, MND1, CDCA5, CCDC34, CENPW, DEPDC1B, AKAP12, E2F7, PRR11, KNL1, IQGAP3, ZNF367, CKAP2L, CENPH, C4orf46, CDK6, HRK, WDR76, DEFA1, PRDM1, CCNE2, MIR8071-1
Common genes from B-ALL only via	CDC42EP3

affy.rma (1/285), for 10/10 samples	
Common genes from T-ALL only via affy.rma (23/285), for 3/3 samples	FKBP5, PFKFB2, MPV17L, SCML4, BTG1, ZFP36L2, DDIT4, RHOBTB3, SLA, ISG20, TNFSF8, SNX29P2, PIK3IP1, SMAP2, SNTB2, SEMA4D, CDKN3, BCL10, PTTG1, FEN1, MIR8071-1, MCM4, IGLC1
Common genes from B-ALL (6/10) and T-ALL (3/3)	SCML4, DDIT4, SLA, PFKFB2, CDKN3, ZFP36L2, FKBP5, SNX29P2, PIK3IP1, TNFSF8, PTTG1, MCM4, MIR8071-1
0-6 h common genes induced (34)	MS4A4A, CLN8, SNX10, RAB31, SERPINA1, LILRA1, LILRB2, PFKFB2, SOCS1, LGALS3, SIK1, SLA, FCGR3B, FGL2, BTNL9, RBMS3, DPEP1, MNDA, FKBP5, DDIT4, WFS1, S100A11, ZBTB16, P2RY14, GSN, EPPK1, ZFP36L2, FCER1G, FGR, IRAK3, PPBP, MYRIP, PIK3IP1, KIF26A
0-6 h common genes repressed (40)	NUF2, ARPP21, ABHD17B, ASPM, PBK, KIF20A, CEP55, PDE4B, HMMR, CDKN3, FUS, AURKA, CENPF, POU4F1, KIF14, CENPE, CENPA, TTK, GIMAP4, DLGAP5, PTTG1, UBE2C, CDC20, MKI67, CCNB2, BIRC5, FAM72C, GBP4, KIF23, NPCDR1, CCNL1, LOC728175, CLEC2B, TOP2A, TENM4, CCNB1, SNORA21, MDM2, DEPDC1, GIMAP7
0-24 h common genes induced (87)	TBXA2R, STAB1, MIR4683, SMIM3, SLC22A23, TMEM100, LOC100130872, SESN1, TMEM2, IFNGR1, BIRC3, P2RX5, LILRB2, PFKFB2, SOCS1, CDC42EP3, SIK1, METTL7A, IL18RAP, SLA, CCR1, SNX29P2, LOC100505650, RHOBTB3, BCL2L11, SNTB2, BTNL9, MS4A1, SOS1, TNFSF8, RNASET2, CD53, LY96, DPEP1, SCML4, LOC285097, ITGAM, DEFA1, MNDA, ISG20, FKBP5, DFNA5, MTSS1, DDIT4, WFS1, IL6ST, MIR6845, NEAT1, KLF9, TXNIP, IL18R1, P2RY14, GSN, HBB, CELF2, HBG1, S100A8, NETO1, ZFP36L2, MPV17L, CHPT1, NEDD9, LOC102723927, FGD2, EPPK1, BTG1, TUBA4A, RPS6KA2, DENND3, IRAK3, PIK3IP1, IL27RA, SNX9, SMAP2, RASSF4, BMF, ELL2, TARSL2, SEMA4D, ITGB2-AS1, CLN8, KIF26A, NR3C1, PPP1R16B, GGNBP2, CPM, ITPKB

<p>0-24 h common genes repressed (180)</p>	<p>IL1B, MCM10, NUF2, CDCA3, UBE2T, TCF19, BRIP1, HMGN5, KIF18A, KCNK12, HELLS, E2F8, NUSAP1, ASPM, ECT2, CENPN, SHCBP1, POLOQ, KIF15, B3GNT2, TIPIN, PBK, ARMC8, DSCC1, CENPU, TNFRSF21, ATAD2, HJURP, KIF20A, NCAPG, DTL, CEP55, KIF4A, CKAP2, PRC1, MSH6, PDE4B, MCM7, LEF1, DDX6, AKAP12, SKP2, BIRC5, TPX2, CDT1, RRM2, HMMR, CDKN3, BUB1, KIF2C, CENPF, MDK, ANP32E, AURKA, CDK1, BYSL, HIST4H4, CCNA2, MND1, EGR1, PRR11, PRPS2, SNORD3B-1, KIF14, HRK, CHEK1, WDR76, IGLL1, RAG1, RCC1, POLE2, MCM4, BCAT1, STIL, PSPH, PCNA, CENPE, RAD51, CENPA, PLK4, TRIP13, NEK2, TTK, MELK, POLA1, FEN1, WDHD1, BRCA1, KIF11, SMC2, WASF1, CDC45, RFC3, ZWINT, CDC6, BUB1B, DLGAP5, PTTG1, MAD2L1, CCDC86, TIMELESS, UBE2C, CDC20, MKI67, CCNB2, OIP5, TYMS, DHFR, FOXM1, KIAA0101, RGS1, TK1, FABP5, MTHFD1, TRIB1, FAM72C, CKAP2L, CKS1B, CENPH, NET1, GBP4, MTHFD2, NME1, SRM, PPIF, EMP1, GGH, KIF23, PAICS, GINS1, AURKB, ASF1B, GINS2, CENPK, IQGAP3, ZNF367, PTP4A1, ANLN, C5orf24, ZBTB24, SUV39H2, SELENOI, RBM14, ARPP21, GAB1, BCL10, HSP90AB1, TOP2A, FH, RAD51AP1, DHX9, TMEM97, NCAPH, FANCI, TENM4, APITD1-CORT, CCNB1, RAG2, MCM5, MDM2, KIF18B, DEPDC1, CDCA5, ARRDC3, KDELC2, SQLE, LOC100996643, CCDC34, RMI2, CENPV, CDCA2, CENPW, DEPDC1B, PHF19, E2F7, KNL1, LOC283454, C4orf46, CDK6, CRNDE</p>
<p>6-24 h common genes induced (17)</p>	<p>SESN1, PFKFB2, IL18RAP, CCR1, BCL2L11, BTNL9, TNFSF8, SERPINB9, DEFA1, FKBP5, PRDM1, ID2, P2RY14, S100A8, GVINP1, SCML4, CDC42EP3</p>
<p>6-24 h common genes repressed (34)</p>	<p>MCM10, HELLS, E2F8, DSCC1, ATAD2, DTL, MIR8071-1, RRM2, CDK1, F13A1, PRR11, BRIP1, IGLL1, CCNE2, MELK, FEN1, ID3, IGLC1, KIF11, CDC45, ZWINT, CDC6, SERPINA1, TYMS, KIAA0101, GGH, GINS1, ASF1B, ZNF367, MAD2L1, RAD51AP1, TMEM97, LYZ, IGH</p>
<p>Common genes for all-time span induced (4)</p>	<p>PFKFB2, BTNL9, FKBP5, P2RY14</p>

Common genes for all-time span repressed (5)	CEP55, MKI67, CCNB2, BIRC5, TOP2A
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As shown in Table 6-2, gene CDC42EP3 was the only one that applied in all B-ALL samples, and genes SCML4, DDIT4, SLA, PFKFB2, CDKN3, ZFP36L2, FKBP5, SNX29P2, PIK3IP1, TNFSF8, PTTG1, MCM4, MIR8071-1 were highly expressed in all T-ALL samples. The 13 genes may suggest that the T-ALL samples may be more sensitive to GC treatment than the B-ALL samples, although B-ALL had 212 common genes in six out of the 10 samples. Gene CDC42EP3 may be particularly associated with the GC treatment of B-ALL samples. Genes PFKFB2, BTNL9, FKBP5 and P2RY14, which were induced across all three time spans, were the set of genes documented in (Chaiboonchoe, 2010), and CEP55, MKI67, CCNB2, BIRC5 and TOP2A were repressed across all three time spans. This may indicate that GC introduced apoptosis started from inducing genes PFKFB2, BTNL9, FKBP5 and P2RY14 and inhibiting genes CEP55, MKI67, CCNB2, BIRC5 and TOP2A.

As discussed in previous chapters, the fundamental Boolean model splits the Boolean functions into the domains of gene activation and inhibition. That then facilitated analysing the Boolean regulatory network into seven main types of sub-networks, as discussed in chapter 3.

Hence, we started to explore the gene networks by filtering the extracted FBNet networks (Appendix B) and then plotted their network graphs within the six types (We skipped the general type as it contains too many nodes). Appendix A shows the

common annotated genes for further investigation on the FBN networks of the leukaemia data that could facilitate the interpretation of the 285 common genes and Appendix B shows fundamental Boolean networks extracted on the 285 common genes. Because of the limitation of the scope of the research, we only discussed a few genes in the following sections.

6.4. Networks of CDC42EP3

As shown in Table 6-2, gene CDC42EP3 was highly expressed in all B-All type samples and was a common gene at 0-24 h and 6-24 h. Hence, we are interested in finding out what activated this gene and what were the consequences of the activation.

Fundamental Boolean Networks

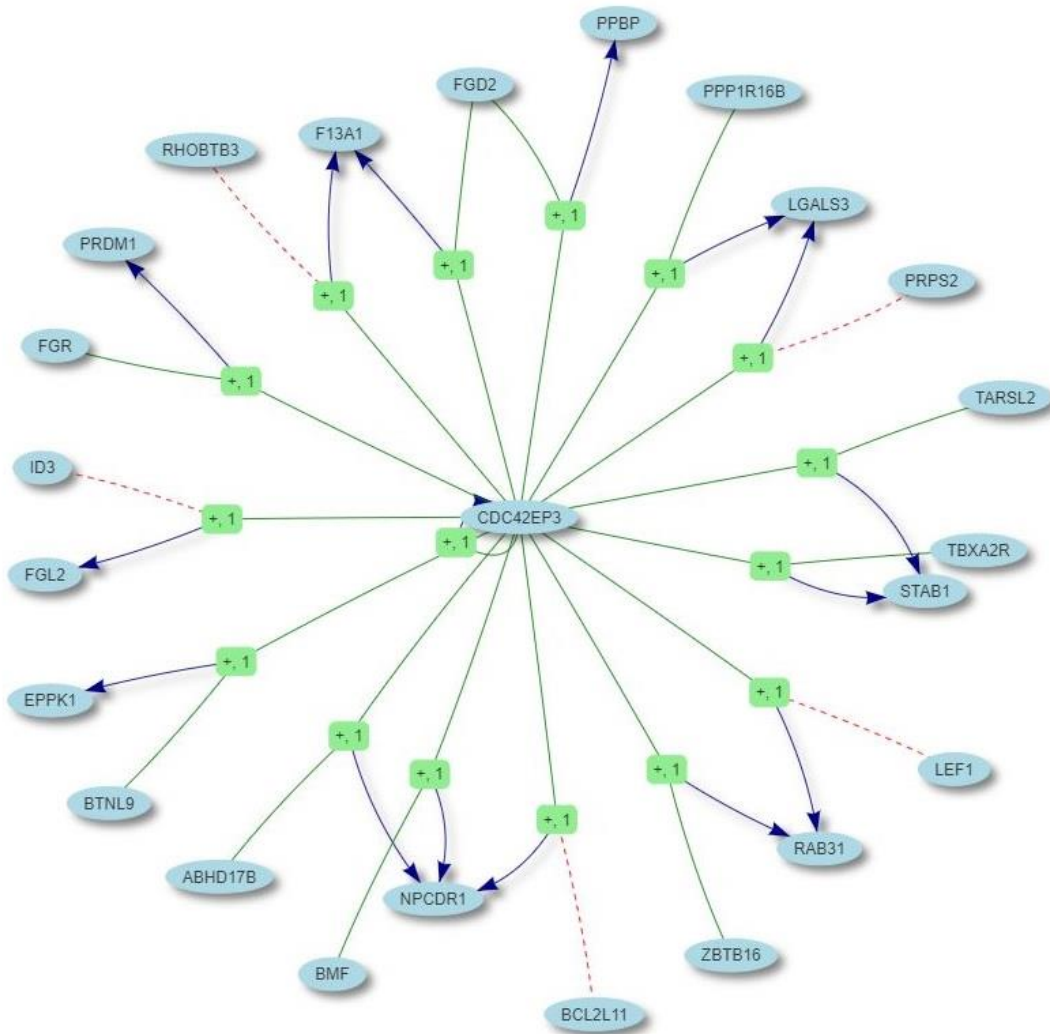


Figure 6-2 FBNet_FAA (type 1) type of fundamental Boolean networks of CDC42EP3. The legend refers to Figure 1.

Figure 6-2 shows that activated CDC42EP3 up-regulates genes EPPK1, F13A1, FGL2, LGALS3, NPCDR1, PPBP, PRDM1, RAB31 and STAB1. Five out of nine genes were documented in the list of the B-ALL gene network list from Chaiboonchoe (2010): EPPK1, FGL2, LGALS3, PRDM1 and STAB1. PPBP was documented in the list of T-ALL gene networks list from Chaiboonchoe (2010). The remaining three genes, F13A1,

NPCDR1 and RAB31, were new findings that had not been reported before that could be up-regulated by CDC42EP3 either in B-All samples or T-All samples. Five out of nine genes were associated with B-All samples, which meant that gene CDC42EP3 was mainly associated with B-ALL type of patients. F13A1 encoded a protein for coagulation factor XIII A chain, which was the last zymogen to become activated in the blood coagulation cascade (genecards.org, 2020a). Diseases associated with F13A1 included Factor Xiii (8), a subunit deficiency of Factor Xiii (8) (genecards.org, 2020a); NPCDR1, nasopharyngeal carcinoma, RNA gene and the diseases associated with NPCDR1, including nasopharyngeal carcinoma (genecards.org, 2020a); RAB31, a member of RAS oncogene family, was associated with diseases including estrogen-receptor-positive breast cancer (genecards.org, 2020a). Hence, we considered that the up-regulation of the three genes F13A1, NPCDR1 and RAB31 could cause side effects under GC induced apoptosis. To inhibit them, we may consider disabling their conditional genes, such as turning on RHOBTB3 and turning off FGD2, to prevent F13A1 from being activated.

Fundamental Boolean Networks

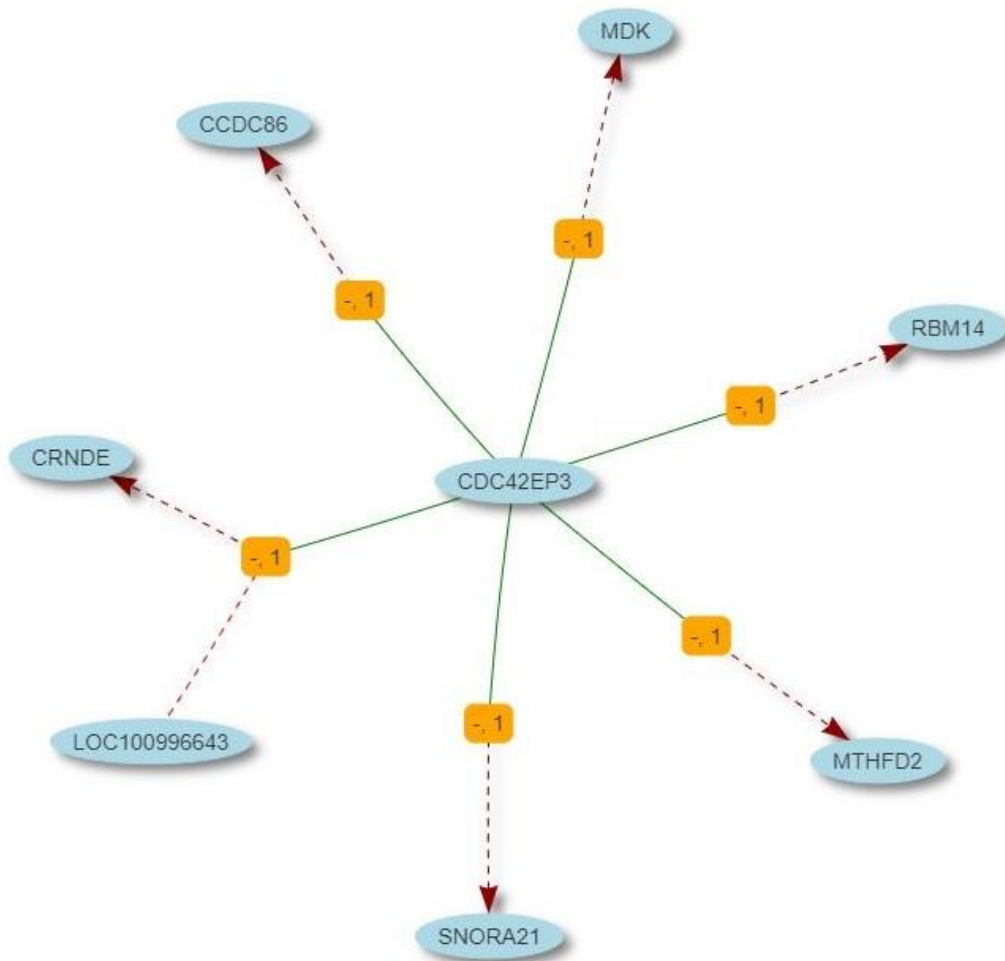


Figure 6-3 FBNet_FAI (type 2) type of fundamental Boolean networks of CDC42EP3.

The legend refers to Figure 1.

Figure 6-3 shows that activated CDC42EP3 down-regulates genes CCDC86, CRNDE, MDK, MTHFD2, RBM14, and SNORA21. These genes have not been reported in previous studies. This could be because down-regulation may be more challenging to detect than gene induction (Schmidt et al., 2006).

Fundamental Boolean Networks

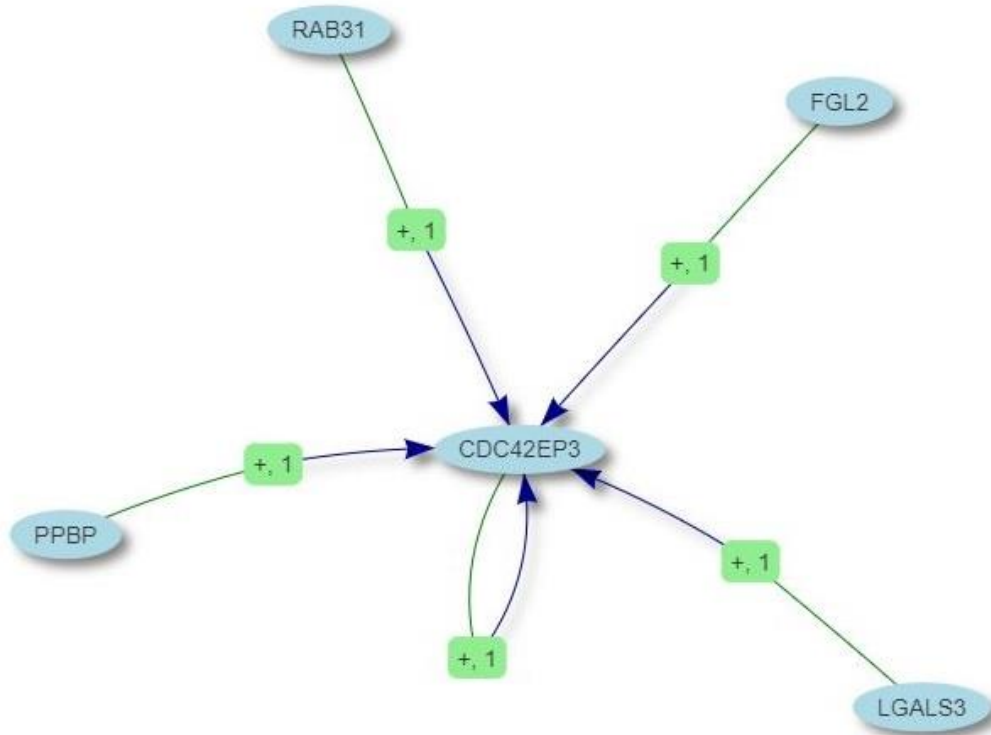


Figure 6-4 FBNNet_BA (type 5) type of fundamental Boolean networks of CDC42EP3.

The legend refers to Figure 1.

Figure 6-4 shows the genes that activate CDC42EP3: RAB31, PPBP, LGALS3, and FGL2.

LGALS3 and FGL2 have been documented in the list of B-ALL gene networks, and PPBP has been documented on the list of T-ALL gene networks (Chaiboonchoe, 2010).

RAB31 was a new finding that has not yet been reported to regulate CDC42EP3 in previous studies. New gene regulations, as well as potential side effects, have been identified through type 1, 2 and 5 regulatory pathways.

6.5. Networks of Genes Induced Across All Periods

Genes PFKFB2, BTNL9, FKBP5 and P2RY14 have been reported in Table 6-2 that have been induced across the three time spans of 0-6 h, 0-24 h and 6-24 h. Hence, types 1, 2, and 5 have been applied to analyse them.

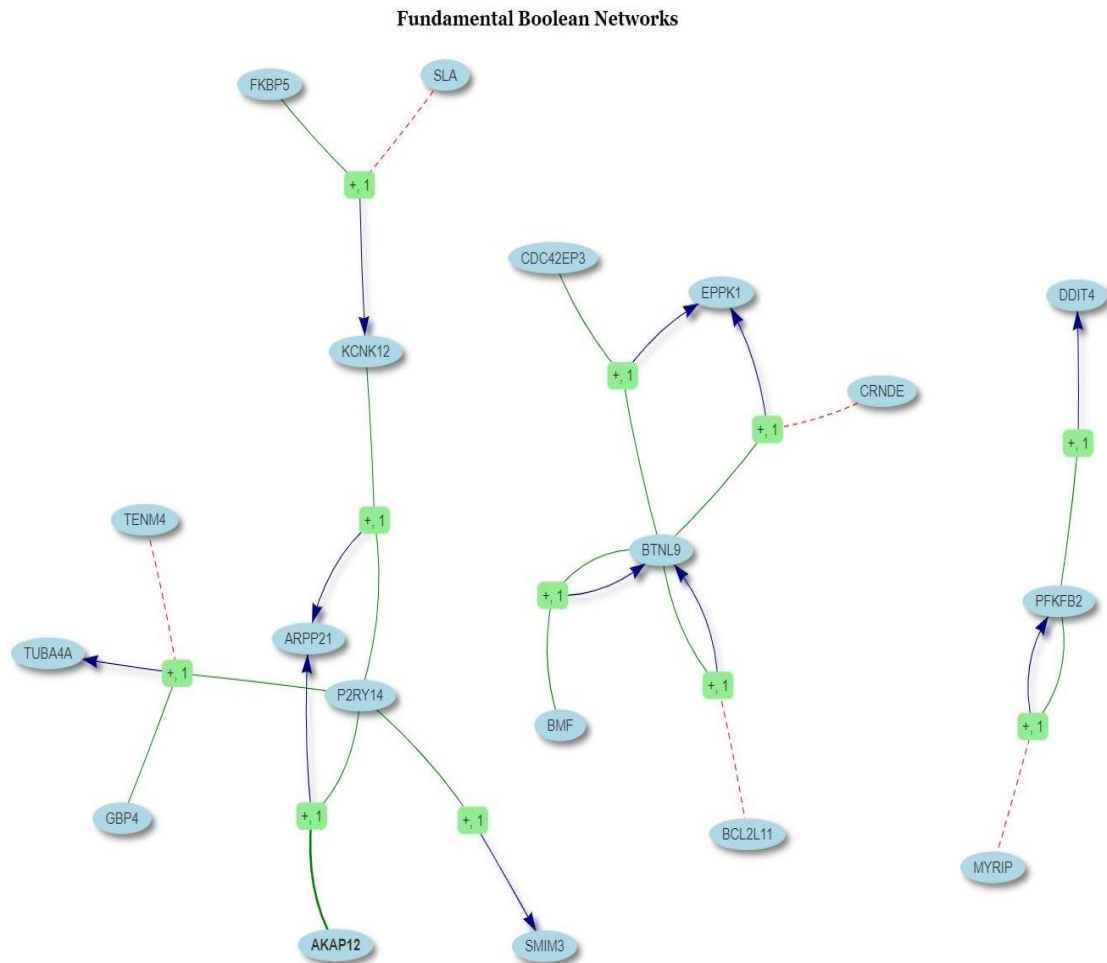


Figure 6-5 FBNNet_FAA (type 1) type of fundamental Boolean networks of all induced genes across the three time spans that up-regulated their target genes. The legend refers to Figure 1.

As shown in Figure 6-5, the GC related gene PFKFB2 (Carlet et al., 2010; Schmidt et al., 2006) activated the critical gene, DDIT4 (DNA damage-inducible transcript 4), which

was an essential candidate for GC-induced apoptosis. Without the expression of MYRIP, PFKFB2 will be self-activated that indicated that the GC treatment turned PFKFB2 on by inhibiting gene MYRIP, which coded the myosin VIIA and Rab interacting proteins. The MYRIP-related pathway was through peptide hormone metabolism. As discussed in the literature review, GC induced apoptosis by influencing hormone metabolism. BTNL9 (butyrophilin like 9) was found to be highly expressed in B-ALL samples, indicating that its pathway could only affect B-All type patients. BTNL9 activated EPPK1, and they were turned on by the overexpression of gene BMF and the underexpression of BCL2L11. BMF and BCL2L11 both belonged to the BCL2L11 family. As documented in Schmidt et al. (2006), genes LDHA, GPR65, MAP2K3, GZMA, MYC, NR3C1 and BCL2L11 were the top candidate genes. The BCL2L11 and Bcl-2 rheostat were proven to induce GC that led to cell death. The target gene of the activated BTNL9 was EPPK1 (its related pathway was cytoskeleton remodelling neurofilaments). EPPK1 could be associated with leukaemia healing because EPPK1 can accelerate keratinocyte migration during wound healing. Gene FKBP5 activated KCNK12 while the GC essential gene SLA was inhibited. The stimulated purinergic receptor (P2RY14) activated TUBA4A, which was connected to the diseases of amyotrophic lateral sclerosis 22 with or without frontotemporal dementia, Robinow syndrome and autosomal dominant 3 (Genecards.org, 2020c). The activated TUBA4A could be the side effect of GC-related treatment, but under two conditions where gene TENM4 must be inhibited and gene, GBP4 must be activated.

Fundamental Boolean Networks

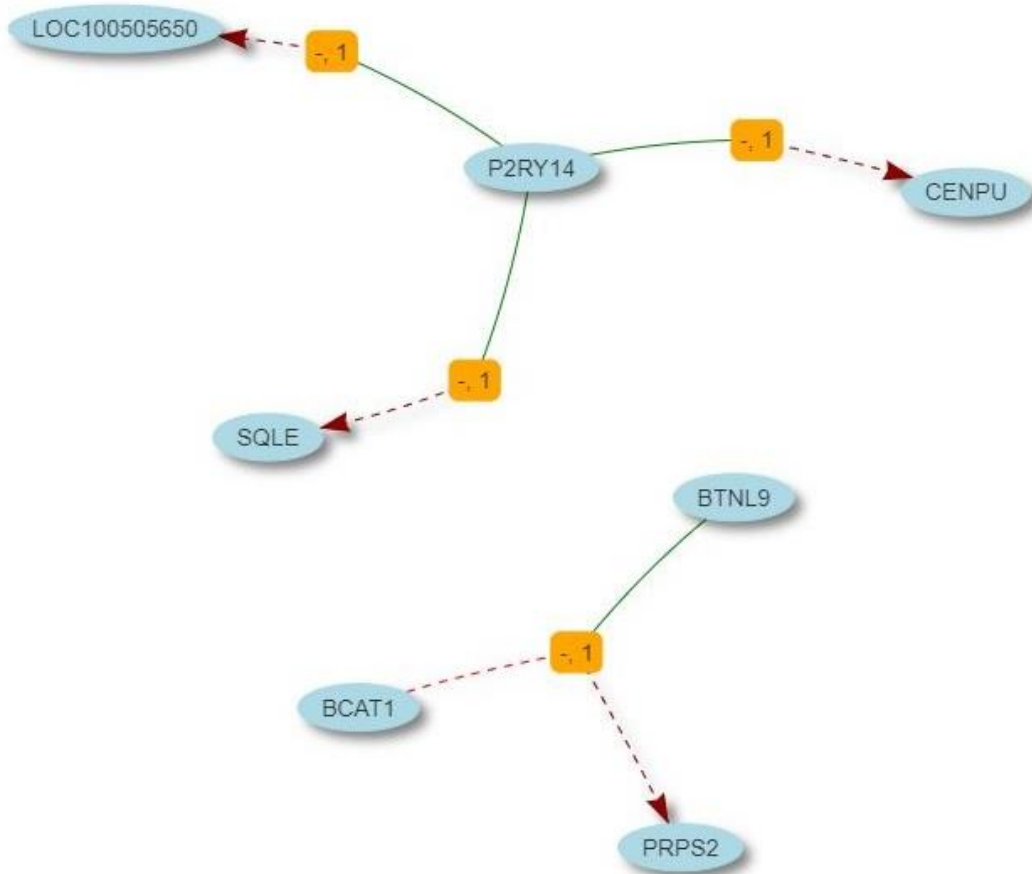


Figure 6-6 FBNet_FAI (type 2) type of fundamental Boolean networks of all induced genes across the three time spans, that down-regulated their target genes. The legend refers to Figure 1.

As shown in Figure 6-6, among the four induced genes, only P2RY14 and BTNL9 were found to inhibit their target genes when activated.

The networks demonstrated in Figures 6-2, 6-3, 6-4, 6-5 and 6-6, show that the fundamental Boolean networks can split into the domain of up-regulation and down-regulation easily without the need to apply other tools or previous knowledge about

networks. Besides, we can find what causes all the genes (induced and repressed) to be activated or inhibited by finding their backward regulation. Figure 6-7 shows that three out of four induced genes were regulated by genes in the set of common 285 genes. Only FKBP5 being missed indicated that it could be turned on by other genes rather than the common genes. The essential gene, PFKFB2, was turned on by gene TNFRSF21, which encoded a protein to activate nuclear factor kappa-B and mitogen-activated protein kinase 8 and induced cell apoptosis (genecards.org, 2020b).

Fundamental Boolean Networks

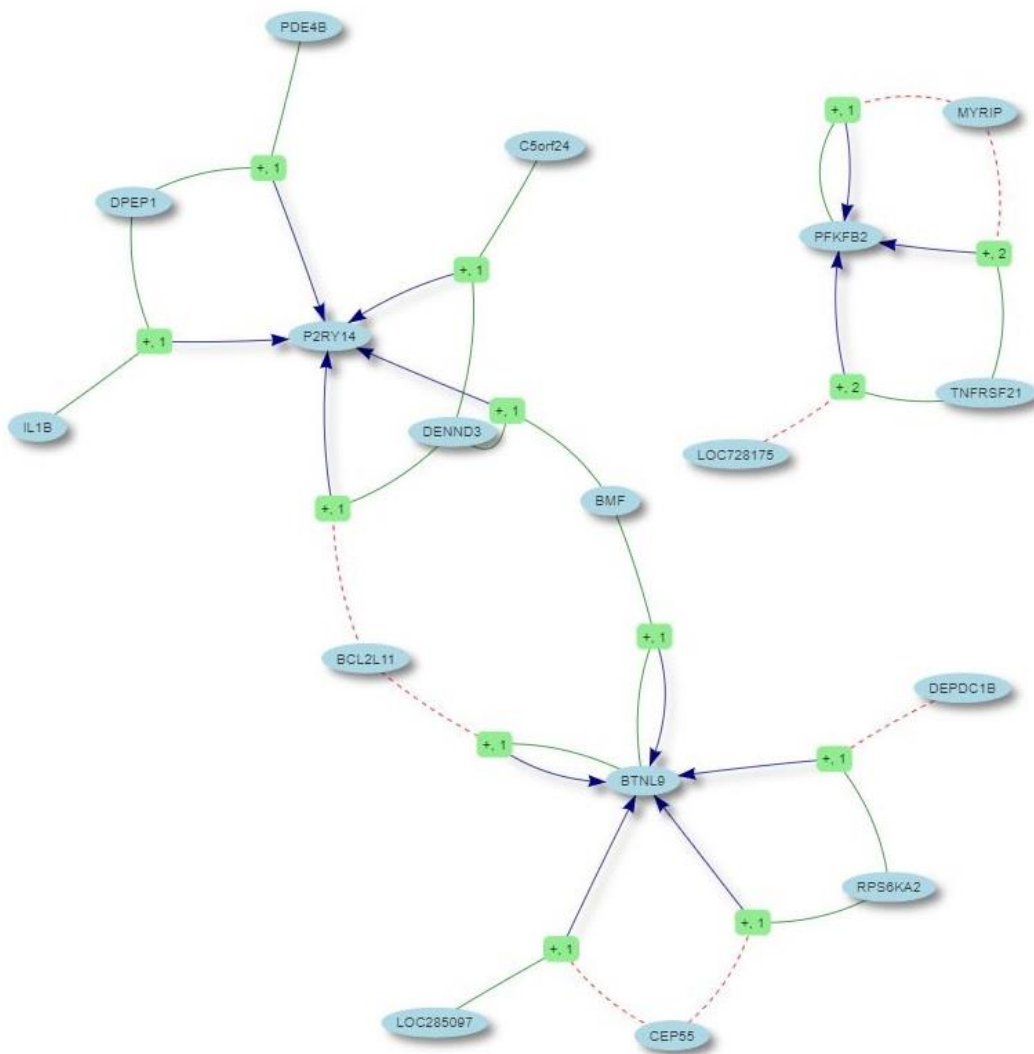


Figure 6-7 FBNett_BA (type 5) type of fundamental Boolean networks of the backward regulation of all induced genes. The legend refers to Figure 1.

Figure 6-7 shows the genes to be activated by tracing the backward regulation of the four induced genes.

6.6. Networks of Genes Repressed Across All Periods

Genes CEP55, MKI67, CCNB2, BIRC5, TOP2A, have been reported in Table 6-2 that have been repressed across three time spans; 0-6 h, 0-24 h and 6-24 h. Hence, types 3, 4, and 6 were applied to analyse them.

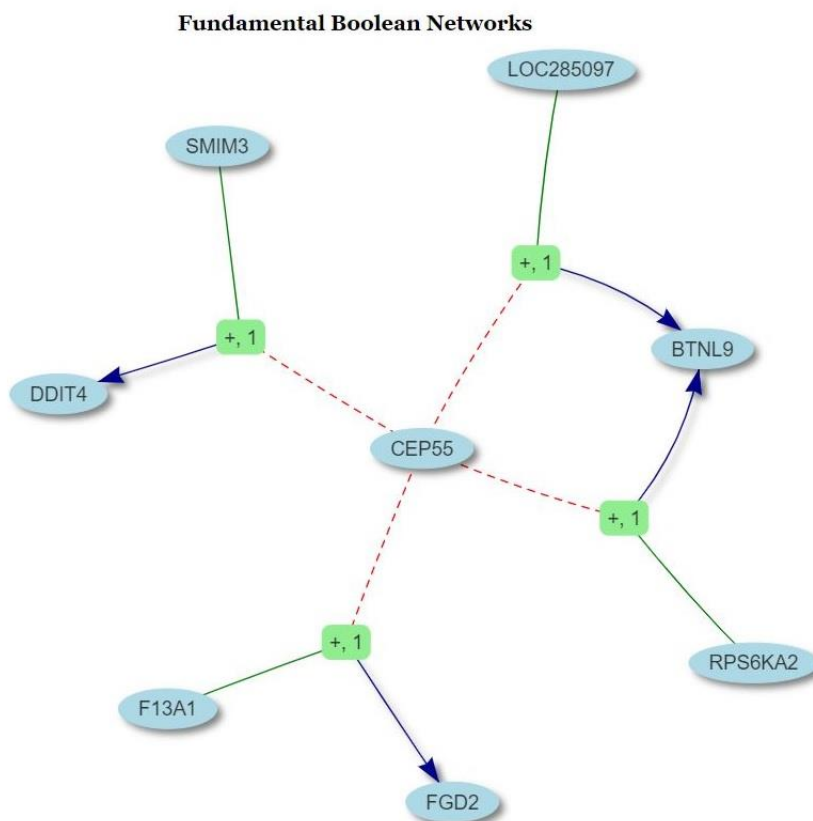


Figure 6-8 FBNet_FIA (type 3) type of fundamental Boolean networks of all repressed genes across the three time spans that up-regulated their target genes.

The legend refers to Figure 1.

Figure 6-8 shows the repressed gene CEP55, which was one of the five repressed genes that inhibited across all periods, up-regulates the critical genes of GC-regulated genes BTNL9 and DDIT4.

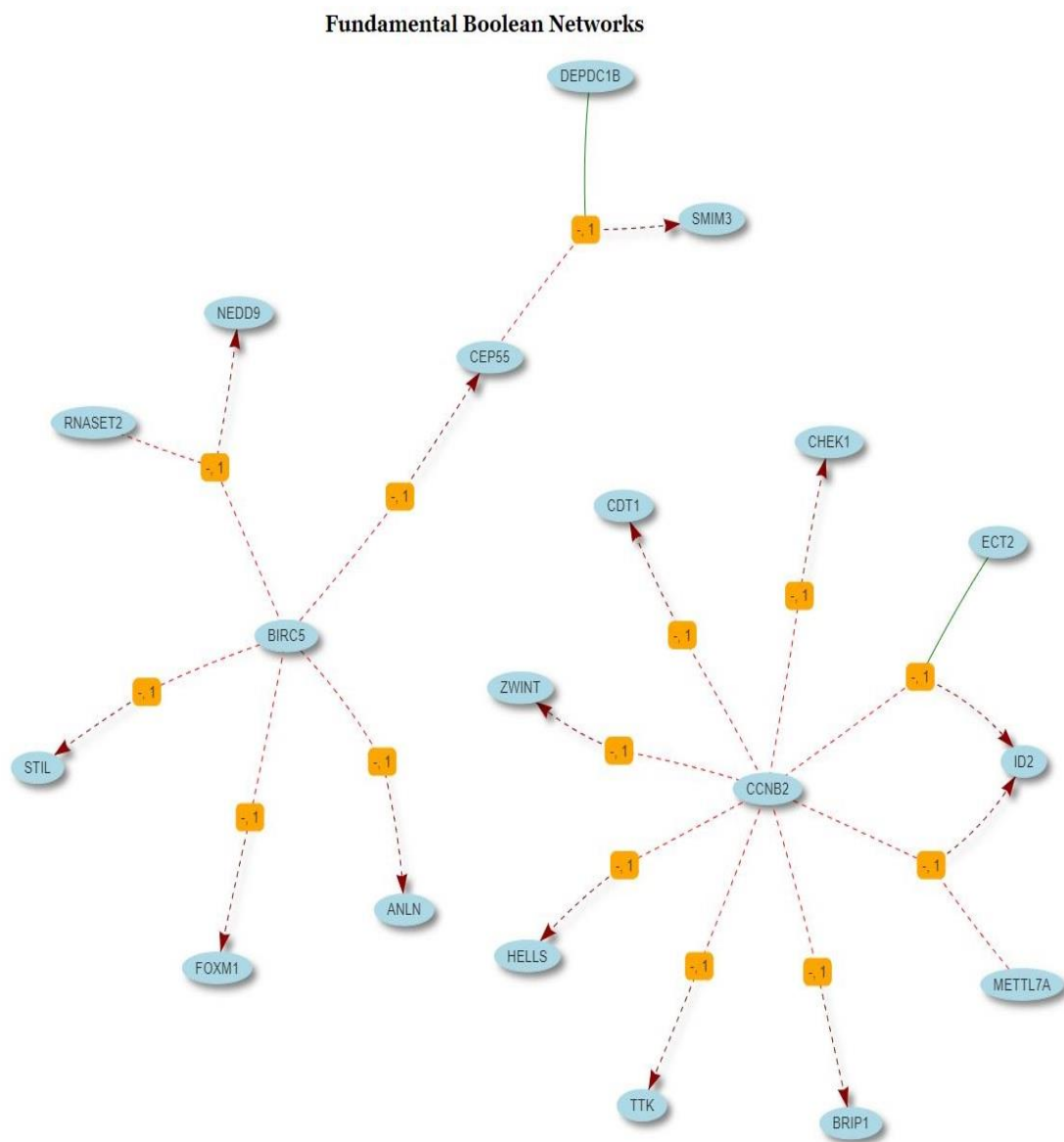


Figure 6-9 FBNet_FII (type 4) type of fundamental Boolean networks of all repressed genes across the three time spans, that up-regulated their target genes.

The legend refers to Figure 1.

Figure 6-10 shows the three repressed genes (CEP55, CCNB2 and CCNB2) among the FICE genes repressed across all periods (CEP55, MKI67, CCNB2, BIRC5 and TOP2Ac).

This may indicate that the target genes of MKI67 and TOP2Ac were not in the common 285 genes.

Fundamental Boolean Networks

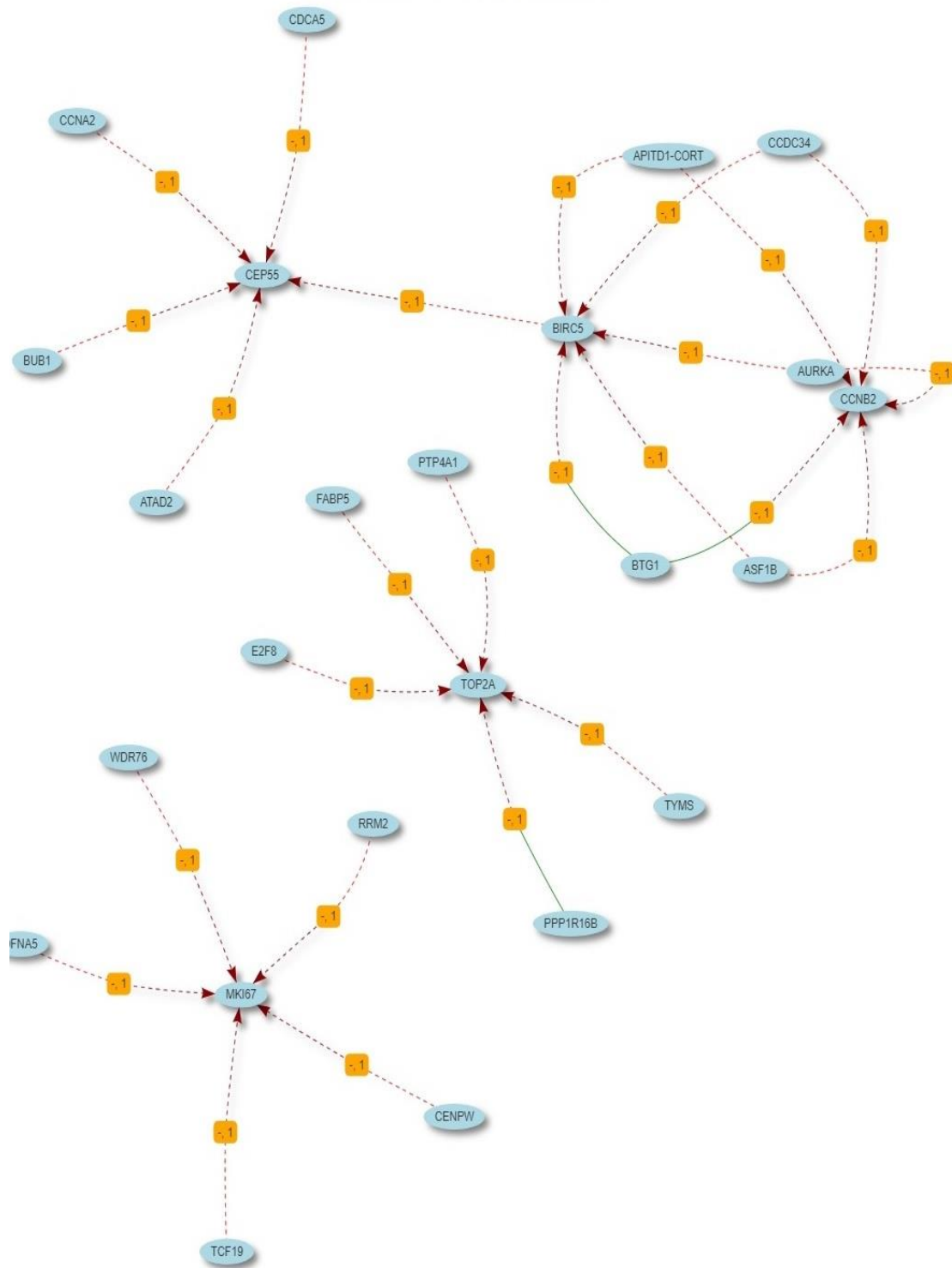


Figure 6-10 FBNNet_BI (type 6) type of fundamental Boolean networks of all the repressed genes backward regulation. The legend refers to Figure 1.

6.7. Primary Candidate Genes for GC-induced Apoptosis

In this section, we demonstrated how to reveal the nature of the GC-regulated genes responsible for the anti-leukemic GC effects via the three top candidates: BCL2L11, MYC and NR3C1. To understand BCL2L11, we explored the gene by looking at the forward and backward networks caused by the gene (intrinsic pathway) by directly regulating the expression of components of the cell death machinery (Schmidt et al., 2006). To fulfil this task, we reconstruct the Orchard cube based on the highly expressed genes with the cutoff ≥ 2 (4-fold), and the majority was two out of 13 samples.

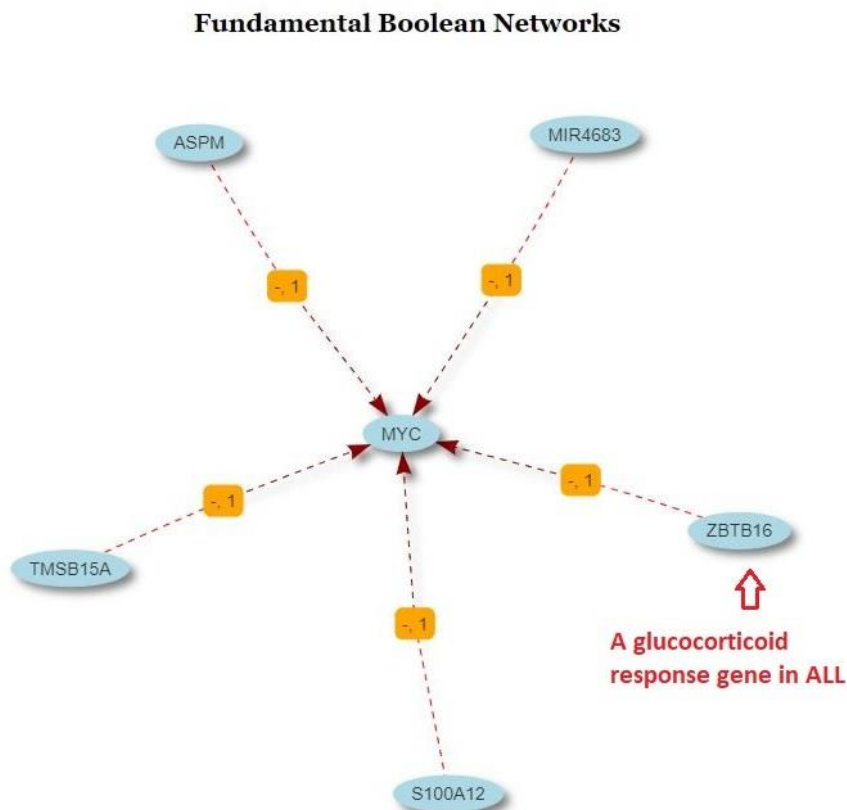


Figure 6-11 FBNett_BI type of fundamental Boolean networks reveals the direct repression on MYC. The legend refers to Figure 1.

As shown in Figure 6-11, the glucocorticoid response gene in ALL, ZBTB16/PLZF, was the critical component in the repression of MYC, which matched the evidence shown in Wasim et al. (2010).

Fundamental Boolean Networks

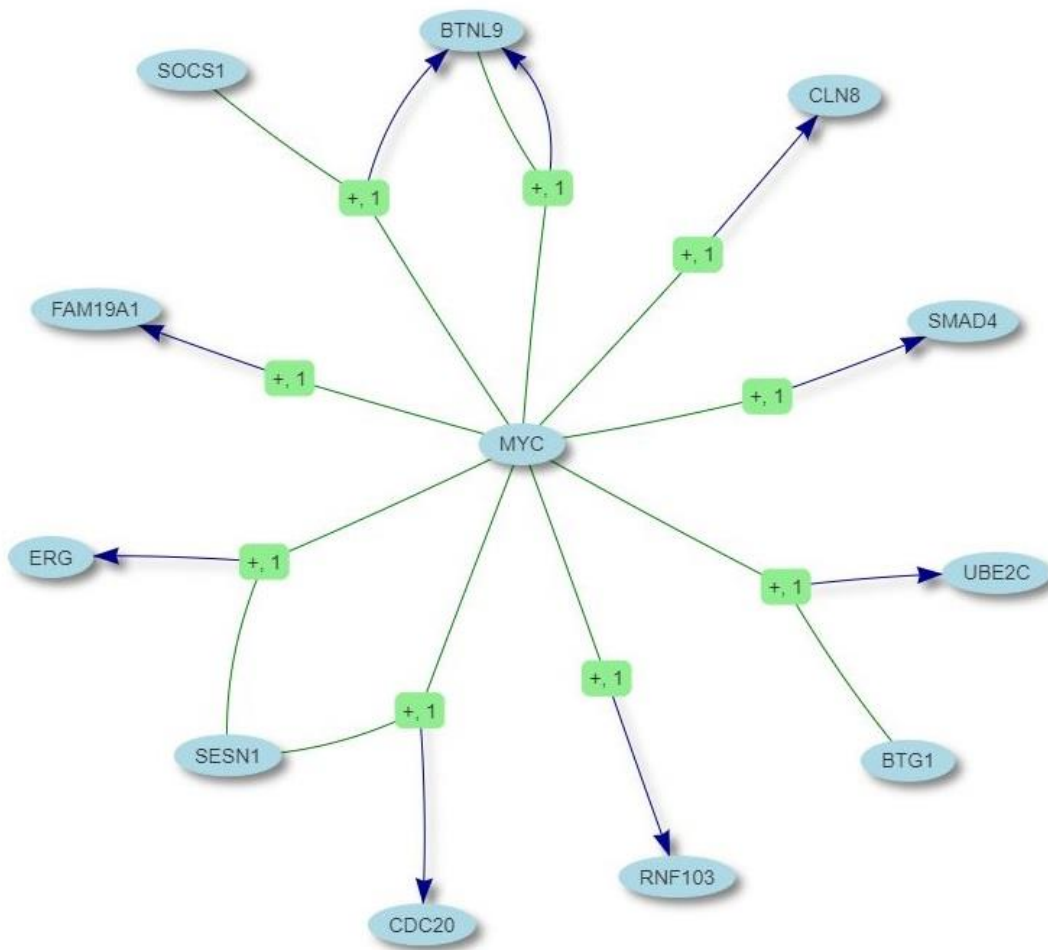


Figure 6-12 FBNNet_FAA type of fundamental Boolean networks reveals the direct regulation caused by the activated MYC. The legend refers to Figure 1.

Both *c-Myc* and *Cdc20* can induce the proliferation of primary glial progenitor cells (Ji et al., 2016); however, we now have evidence that *Cdc20* was driven by MYC, as shown

in Figure 6-12. In other words, MYC was the driver and Cdc20 the passenger, which has since been confirmed by the study (Ji et al., 2016). By combining the outputs of Figures 6-11 and 6-12, it was very straightforward to discover the intrinsic pathway of how GC repression of MYC (c-myc) generates a “conflicting signal”: GC binds with the glucocorticoid response gene, which in turn, inhibited MYC and hence, interrupted the induction of Cdc20.

UBE2C, whose alias name is Ubch10, is a protein-coding gene, from among the ubiquitin conjugating enzyme E2 gene family. The expression level of Ubch10 was deficient in many of the healthy tissues but prominent in the majority of cancerous cell lines (Okamoto et al., 2003). Ubch10 contributed to tumourigenesis or the progression of the tumor because Ubch10 was used to express at high levels in primary tumours, mainly derived from the lung, stomach, uterus, and bladder, compared with their corresponding healthy tissues (Okamoto et al., 2003).

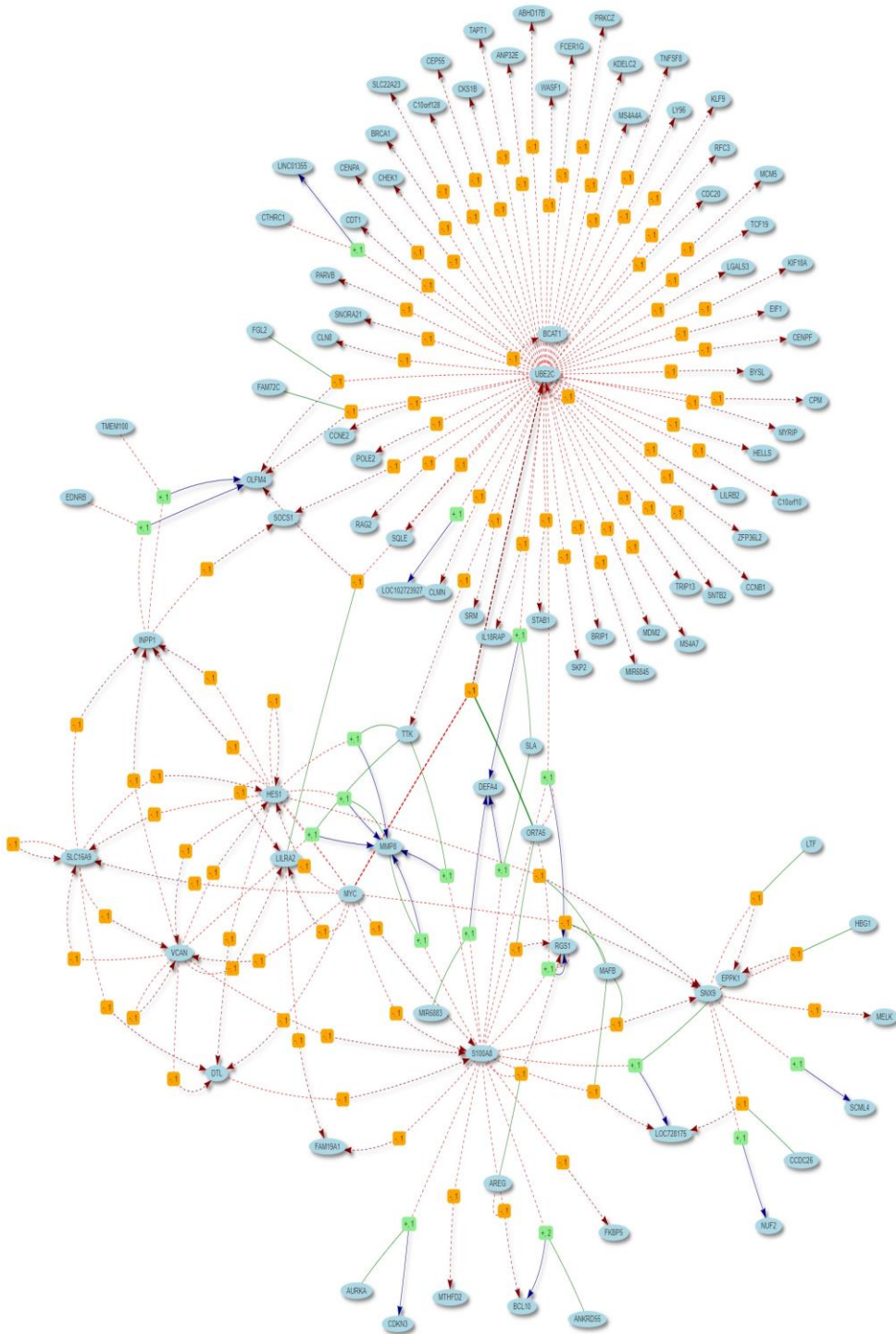


Figure 6-13 Downregulations of MYC in multiple levels when it has been inhibited.

The legend refers to Figure 1.

The repressed MYC also repressed UBE2C, suggesting the natural pathway of GC bound with ZBTB16 that then repressed MYC and inhibited MYC, along with the induction of ORA75, inhibited the ubiquitin conjugating enzyme E2 gene, UBE2C/Ubc10.

6.8. Summary

In summary, finding the down-regulatory pathway was difficult (Schmidt et al., 2006) but with the novel FBM/TFBM, it was very straightforward. The fundamental Boolean modelling concepts and related network inference technology enable scientists to analyse genetic regulatory pathways easily with different types of network visualisation. For example, potential side effects of GC treatment could be identified through the forward and backward regulatory pathways analysis.

In this chapter, we demonstrated how to apply the temporal fundamental Boolean modelling on acute childhood leukaemia data in clinical settings. The generated networks have been attached to Appendix B for further research. Due to the time constraint, we only discussed a few pathways of the networks in this chapter. However, we have found some potential new findings related to the pathways of CDC43EP3 such as the three genes, F13A1, NPCDR1 and RAB31, which considered to be the potential side effects of GC introduced apoptosis. This chapter provides evidence to confirm that the novel Boolean model could improve the understanding of genetic regulatory networks on the induction of leukaemia related treatments.

Chapter 7: Summary, Conclusions, Future Directions and Contributions

7.1. Summary

In this study, we investigated the characteristics of enzyme activation, inhibition and protein decay, then proposed two novel data-driven Boolean models, namely, the fundamental Boolean model (FBM) and temporal fundamental Boolean model (TFBM), to draw insights into gene regulatory pathways. The fundamental Boolean modelling can separate the activation and inhibition functions from conventional Boolean functions, and this separation could facilitate scientists in seeking answers in such as how an amendment of one gene distresses other genes at the expression level. We proposed a new data-driven method to infer networks for the proposed fundamental Boolean modelling. The new method comprises two different parts: the first part was to construct an Orchard cube to persist all precomputed measures for all potential fundamental Boolean functions; the second part was to infer FBNs/TFBNs from the Orchard cube, by filtering each tree's underground part. Dynamic FBNs show the significant trajectories of genes to reveal how genes regulated each other over a given period. This new feature could facilitate further research into drug interactions to detect the side effects from the use of a newly proposed drug. The protein decay issue was also a function of the proposed model (Eq(3.4)) to capture the characteristics of the time that allows a gene to remain in the *On* state when there are no activators or inhibitors.

Fundamental Boolean modelling is data-driven Boolean modelling, and the related Boolean functions are inferred from a particular type of data cube. Hence, knowledge about connectivity among the genes was not needed but can be used to verify the results generated. To demonstrate the concepts of the FBM and TFBM, we implemented an R package called *FBNNet*, which successfully demonstrated cell cycle and leukaemia pathways. The R package provided a tool to draw FBNs/TFBNs, either in the static model, as shown in Figure 5-3, or the dynamic model, as illustrated in Figure 5-5.

The dynamic trajectory of gene activation, inhibition and protein decay activities of attractor 2 were deciphered in chapter 5, and this confirmed that the proposed FBM could show the internal connections between genes separated by the assembly types of activation, inhibition, and protein decay.

It is a need to search all related genes and to calculate all relevant measures for all the associated gene combinations up to in some detail. This downside could end with the non-deterministic polynomial acceptable problems (NP-hard problem), i.e., there was no known polynomial algorithm to facilitate the time to find a solution grew exponentially with predefined problem size, as mentioned in Liu et al. (2016). Nevertheless, with the design of the Orchard cube, the cost was manageable because the design was embedded within parallel computations. Hence, even large GRNs can be derived from the method we proposed. Furthermore, the construction of the Orchard cube was detached from the inference of fundamental Boolean networks.

We can apply a different strategy to mine the regulatory networks without the need to reconstruct the cube. Consequently, the computational cost can be split into two parts: the construction of an Orchard cube and the network inference.

The proposed TFBM can be applied on short time series data because it has been designed to employ more previous time steps than FBM, i.e., using the best previous time step for its functions. To demonstrate this, we applied the network inference methodology on the leukaemia data, supplied by Schmidt et al. (2006). Chapter 6 documented the experiments and discussed the result. We highlighted the main findings below.

Findings

In chapter 6, we found there are three genes, F13A1, NPCDR1 and RAB31, which could be side effects of GC induced apoptosis, as activated by CDC42EP3, a gene induced across all B-All samples, from 6-24 h and remaining induced from 0-24 h. The activated CDC42EP3 down-regulates the genes CCDC86, CRNDE, MDK, MTHFD2, RBM14, and SNORA21, and these have not been reported in previous studies. RAB31 is a new finding that has not been reported that can regulate CDC42EP3 in previous studies. The extracted 285 common differentially expressed genes (Appendix A) were the results of the three experimental methods, i.e., re-analysing the M-value from Schmidt et al. (2006) research, computed the raw data normalised with RMA and GCRMA. The reported 285 genes could be the major gene set that regulated by GC induced apoptosis.

We also found that ZBTB16/PLZF was the critical component in the repression of MYC and confirmed that Cdc20 was driven by MYC.

7.2. Conclusions

In this thesis, we proposed the concepts of novel data-driven Boolean modelling (FBM and TFBM) to separate the traditional Boolean functions into the domain of gene activation and inhibition. As shown in the demonstration of the cell cycle and leukaemia networks, the down-regulatory pathways can be detected with the facilitation of the novel Boolean modelling easily, as well as up-regulatory pathways. With TFBM, the insights of short time series data can be revealed intuitively, as shown from the study of childhood leukaemia data.

In this thesis, the questions raised in chapter 1 have been addressed as follows:

Can we apply the Boolean model to gain meaningful insights into microarray time series data, even though the data are short time series data?

The applications of cell cycle and leukaemia documented in chapter 5 and 6 confirmed that we could apply the fundamental Boolean modelling, which is an extension of Boolean modelling, to gain meaningful insights into microarray time series data, even though the data are short time series data.

Can the novel Boolean model improve understanding of genetic regulatory networks on the induction of leukaemia related apoptosis?

Chapter 6 documented the experiments on leukaemia related apoptosis and provided evidence to confirm that the novel Boolean model can improve understanding of genetic regulatory networks on the induction of leukaemia related apoptosis.

Can we apply the Boolean model to identify down-regulatory pathways as well as up-regulatory pathways?

The answer has been addressed straightforwardly in previous chapters. The proposed Boolean models (FBM, TFBM) enable the related networks to be separated into seven subnetwork types, including up-regulatory pathways and down-regulatory pathways. Especially during the study, we produced the fundamental Boolean networks on the childhood acute lymphoblastic leukaemia gene expression data, which were produced in clinical settings. The pathways may be useful for pharmaceutical agents to identify any side effects through the subnetwork analysis.

To sum up, the proposed fundamental Boolean modelling are fine-grained Boolean modelling that can provide more insights into genetic regulatory pathways than the coarse-grained conventional Boolean modelling.

7.3. Future Directions

The proposed concepts of fundamental Boolean modelling (FBM and TFBM) and related networks (FBN and TFBN) are novel, and hence, they do need further research on how to apply them on clinical data. In this thesis, we only discuss a small set of critical genes for Leukaemia, and hence it requires further analysis of the networks

attached in Appendix B. We only documented a few genes in this thesis and hence, remain large regulatory pathways attached in Appendix B for future to analysis.

The unpublished R package FBNet (version 1.0, and 2.0) are developed as a prototype specifically for this study, and hence, it requires future work to improve the package to be publishable.

7.4. Main Contributions

In this thesis, we developed two novel theoretical Boolean network modelling concepts (FBM and TFBM) for analysing genetic regulatory pathways. The novel theoretical Boolean network modelling concepts could reveal significant trajectories in genes to explore how genes regulated each other over a given period. This new feature could facilitate further research on drug interventions to detect the potential side effects of a newly-proposed drug. With the novel modelling, we addressed the problem of investigating gene up-regulatory pathways as well as down-regulatory pathways under one model which drew insights into gene activation, inhibition and protein decay. Besides, we also provided a mechanism to infer the fundamental Boolean modelling related networks from time series data, including short time series data.

During the study, we produced the fundamental Boolean networks, as shown in Appendix B, on the childhood acute lymphoblastic leukaemia gene expression data, which were produced in clinic settings and are short time series data. The networks may be useful for pharmaceutical agents to identify any side effects when applying GC

induced apoptosis on children. We also pointed out some potential side effects in chapter 6 and discussed some new findings. The new findings could be useful for pharmaceutical agents to test their drug-related hypotheses.

Moreover, we developed an R package, namely *FBNet*, to facilitate the research on the genetic regulatory pathway analysis within the domain of fundamental Boolean modelling. The package produces all outcomes documented in this thesis. The pathways in Appendix B were the outcome extracted by the package and are valuable for further research.

Appendix A: Common Annotated Genes

Common annotated genes

Probeset	Symbol	Gene.Name
241765_at	CPM	carboxypeptidase M(CPM)
1565717_s_at	FUS	FUS RNA binding protein(FUS)
207216_at	TNFSF8	tumour necrosis factor superfamily member 8(TNFSF8)
222281_s_at	LOC100505650	uncharacterized LOC100505650(LOC100505650)
1559091_s_at	FGD2	FYVE, RhoGEF and PH domain containing 2(FGD2)
224847_at	CDK6	cyclin dependent kinase 6(CDK6)
232238_at	ASPM	abnormal spindle microtubule assembly(ASPM)
230499_at	BIRC3	baculoviral IAP repeat containing 3(BIRC3)
201577_at	NME1	NME/NM23 nucleoside diphosphate kinase 1(NME1)
205967_at	HIST4H4	histone cluster 4 H4(HIST4H4)
202156_s_at	CELF2	CUGBP, Elav-like family member 2(CELF2)
220046_s_at	CCNL1	cyclin L1(CCNL1)
218883_s_at	CENPU	centromere protein U(CENPU)
239635_at	RBM14	RNA binding motif protein 14(RBM14)
236641_at	KIF14	kinesin family member 14(KIF14)
1556473_at	LOC285097	uncharacterized FLJ38379(LOC285097)
203541_s_at	KLF9	Kruppel like factor 9(KLF9)
203401_at	PRPS2	phosphoribosyl pyrophosphate synthetase 2(PRPS2)
227062_at	NEAT1	nuclear paraspeckle assembly transcript 1 (non-protein coding)(NEAT1)
208079_s_at	AURKA	aurora kinase A(AURKA)
217763_s_at	RAB31	RAB31, member RAS oncogene family(RAB31)
1557910_at	HSP90AB1	heat shock protein 90 alpha family class B member 1(HSP90AB1)
218755_at	KIF20A	kinesin family member 20A(KIF20A)
210567_s_at	SKP2	S-phase kinase associated protein 2(SKP2)
1554768_a_at	MAD2L1	MAD2 mitotic arrest deficient-like 1 (yeast)(MAD2L1)
222962_s_at	MCM10	minichromosome maintenance 10 replication initiation factor(MCM10)

204150_at	STAB1	stabilin 1(STAB1)
204863_s_at	IL6ST	interleukin 6 signal transducer(IL6ST)
225160_x_at	MDM2	MDM2 proto-oncogene(MDM2)
1558143_a_at	BCL2L11	BCL2 like 11(BCL2L11)
213454_at	APITD1-CORT	APITD1-CORT readthrough(APITD1-CORT)
216277_at	BUB1	BUB1 mitotic checkpoint serine/threonine kinase(BUB1)
1568718_at	SLC22A23	solute carrier family 22 member 23(SLC22A23)
233969_at	IGLC1	immunoglobulin lambda constant 1(IGLC1)
1553106_at	C5orf24	chromosome 5 open reading frame 24(C5orf24)
217418_x_at	MS4A1	membrane spanning 4-domains A1(MS4A1)
202580_x_at	FOXM1	forkhead box M1(FOXM1)
219258_at	TIPIN	TIMELESS interacting protein(TIPIN)
219392_x_at	PRR11	proline rich 11(PRR11)
212912_at	RPS6KA2	ribosomal protein S6 kinase A2(RPS6KA2)
1562364_at	GVINP1	GTPase, very large interferon inducible pseudogene 1(GVINP1)
201013_s_at	PAICS	phosphoribosylaminoimidazole carboxylase; phosphoribosylaminoimidazolesuccinocarboxamide synthase(PAICS)
227265_at	FGL2	fibrinogen like 2(FGL2)
214581_x_at	TNFRSF21	TNF receptor superfamily member 21(TNFRSF21)
235574_at	GBP4	guanylate binding protein 4(GBP4)
213253_at	SMC2	structural maintenance of chromosomes 2(SMC2)
222874_s_at	CLN8	ceroid-lipofuscinosis, neuronal 8(CLN8)
238447_at	RBMS3	RNA binding motif single stranded interacting protein 3(RBMS3)
204026_s_at	ZWINT	ZW10 interacting kinetochore protein(ZWINT)
204560_at	FKBP5	FK506 binding protein 5(FKBP5)
219555_s_at	CENPN	centromere protein N(CENPN)
202975_s_at	RHOBTB3	Rho related BTB domain containing 3(RHOBTB3)
209116_x_at	HBB	hemoglobin subunit beta(HBB)
227957_at	GSN	gelsolin(GSN)
203612_at	BYSL	bystin like(BYSL)
226517_at	BCAT1	branched chain amino acid transaminase 1(BCAT1)

204698_at	ISG20	Interferon stimulated exonuclease gene 20(ISG20)
1556599_s_at	ARPP21	cAMP regulated phosphoprotein 21(ARPP21)
203033_x_at	FH	fumarate hydratase(FH)
212974_at	DENND3	DENN domain containing 3(DENND3)
221436_s_at	CDCA3	cell division cycle associated 3(CDCA3)
223381_at	NUF2	NUF2, NDC80 kinetochore complex component(NUF2)
207931_s_at	PFKFB2	6-phosphofructo-2-kinase/fructose-2,6-biphosphatase 2(PFKFB2)
1555728_a_at	MS4A4A	membrane spanning 4-domains A4A(MS4A4A)
235609_at	BRIP1	BRCA1 interacting protein C-terminal helicase 1(BRIP1)
237241_at	ECT2	epithelial cell transforming 2(ECT2)
228323_at	KNL1	kinetochore scaffold 1(KNL1)
203213_at	CDK1	cyclin dependent kinase 1(CDK1)
205048_s_at	PSPH	phosphoserine phosphatase(PSPH)
1556472_s_at	SCML4	sex comb on midleg-like 4 (Drosophila)(SCML4)
221557_s_at	LEF1	lymphoid enhancer binding factor 1(LEF1)
226661_at	CDCA2	cell division cycle associated 2(CDCA2)
203967_at	CDC6	cell division cycle 6(CDC6)
201897_s_at	CKS1B	CDC28 protein kinase regulatory subunit 1B(CKS1B)
204007_at	FCGR3B	Fc fragment of IgG receptor IIIb(FCGR3B)
206865_at	HRK	harakiri, BCL2 interacting protein(HRK)
227212_s_at	PHF19	PHD finger protein 19(PHF19)
1560706_at	NEDD9	neural precursor cell expressed, developmentally down-regulated 9(NEDD9)
235102_x_at	SNORD3B-1	small nucleolar RNA, C/D box 3B-1(SNORD3B-1)
226611_s_at	CENPV	centromere protein V(CENPV)
205883_at	ZBTB16	zinc finger and BTB domain containing 16(ZBTB16)
1564796_at	EMP1	epithelial membrane protein 1(EMP1)
218346_s_at	SESN1	sestrin 1(SESN1)
202533_s_at	DHFR	dihydrofolate reductase(DHFR)
210983_s_at	MCM7	minichromosome maintenance complex component 7(MCM7)
227404_s_at	EGR1	early growth response 1(EGR1)
205909_at	POLE2	DNA polymerase epsilon 2, accessory subunit(POLE2)

213931_at	ID2	inhibitor of DNA binding 2, HLH protein(ID2)
210052_s_at	TPX2	TPX2, microtubule nucleation factor(TPX2)
204727_at	WDHD1	WD repeat and HMG-box DNA binding protein 1(WDHD1)
205023_at	RAD51	RAD51 recombinase(RAD51)
224797_at	ARRDC3	arrestin domain containing 3(ARRDC3)
206940_s_at	POU4F1	POU class 4 homeobox 1(POU4F1)
211430_s_at	MIR8071-1	microRNA 8071-1(MIR8071-1)
209723_at	SERPINB9	serpin family B member 9(SERPINB9)
214710_s_at	CCNB1	cyclin B1(CCNB1)
1556209_at	CLEC2B	C-type lectin domain family 2 member B(CLEC2B)
211428_at	SERPINA1	serpin family A member 1(SERPINA1)
211590_x_at	TBXA2R	thromboxane A2 receptor(TBXA2R)
204959_at	MNDA	myeloid cell nuclear differentiation antigen(MNDA)
232278_s_at	DEPDC1	DEP domain containing 1(DEPDC1)
228071_at	GIMAP7	GTPase, IMAP family member 7(GIMAP7)
211646_at	IGH	immunoglobulin heavy locus(IGH)
227426_at	SOS1	SOS Ras/Rac guanine nucleotide exchange factor 1(SOS1)
200733_s_at	PTP4A1	protein tyrosine phosphatase type IVA, member 1(PTP4A1)
202870_s_at	CDC20	cell division cycle 20(CDC20)
239818_x_at	TRIB1	tribbles pseudokinase 1(TRIB1)
227312_at	SNTB2	syntrophin beta 2(SNTB2)
201009_s_at	TXNIP	thioredoxin interacting protein(TXNIP)
213008_at	FANCI	Fanconi anemia complementation group I(FANCI)
204531_s_at	BRCA1	BRCA1, DNA repair associated(BRCA1)
1553666_at	CCDC34	coiled-coil domain containing 34(CCDC34)
201761_at	MTHFD2	methylenetetrahydrofolate dehydrogenase (NADP+ dependent) 2, methenyltetrahydrofolate cyclohydrolase(MTHFD2)
1556201_at	RNASET2	ribonuclease T2(RNASET2)
221505_at	ANP32E	acidic nuclear phosphoprotein 32 family member E(ANP32E)
228868_x_at	CDT1	chromatin licensing and DNA replication factor 1(CDT1)
228033_at	E2F7	E2F transcription factor 7(E2F7)
202911_at	MSH6	mutS homolog 6(MSH6)

210660_at	LILRA1	leukocyte immunoglobulin like receptor A1(LILRA1)
213562_s_at	SQLE	squalene epoxidase(SQLE)
222848_at	CENPK	centromere protein K(CENPK)
222870_s_at	B3GNT2	UDP-GlcNAc:betaGal beta-1,3-N-acetylglucosaminyltransferase 2(B3GNT2)
203416_at	CD53	CD53 molecule(CD53)
219978_s_at	NUSAP1	nucleolar and spindle associated protein 1(NUSAP1)
223274_at	TCF19	transcription factor 19(TCF19)
229041_s_at	ITGB2-AS1	ITGB2 antisense RNA 1(ITGB2-AS1)
204962_s_at	CENPA	centromere protein A(CENPA)
218662_s_at	NCAPG	non-SMC condensin I complex subunit G(NCAPG)
1554899_s_at	FCER1G	Fc fragment of IgE receptor Ig(FCER1G)
207826_s_at	ID3	inhibitor of DNA binding 3, HLH protein(ID3)
202345_s_at	FABP5	fatty acid binding protein 5(FABP5)
203764_at	DLGAP5	DLG associated protein 5(DLGAP5)
234366_x_at	CKAP2	cytoskeleton associated protein 2(CKAP2)
203037_s_at	MTSS1	MTSS1, I-BAR domain containing(MTSS1)
211519_s_at	KIF2C	kinesin family member 2C(KIF2C)
219148_at	PBK	PDZ binding kinase(PBK)
234307_s_at	KIF26A	kinesin family member 26A(KIF26A)
204033_at	TRIP13	thyroid hormone receptor interactor 13(TRIP13)
223556_at	HELLS	helicase, lymphoid-specific(HELLS)
205263_at	BCL10	B-cell CLL/lymphoma 10(BCL10)
208949_s_at	LGALS3	galectin 3(LGALS3)
228964_at	PRDM1	PR/SET domain 1(PRDM1)
219493_at	SHCBP1	SHC binding and spindle associated 1(SHCBP1)
210448_s_at	P2RX5	purinergic receptor P2X 5(P2RX5)
209709_s_at	HMMR	hyaluronan mediated motility receptor(HMMR)
214146_s_at	PPBP	pro-platelet basic protein(PPBP)
202094_at	BIRC5	baculoviral IAP repeat containing 5(BIRC5)
238022_at	CRNDE	colorectal neoplasia differentially expressed (non-protein coding)(CRNDE)

212949_at	NCAPH	non-SMC condensin I complex subunit H(NCAPH)
206591_at	RAG1	recombination activating 1(RAG1)
1555274_a_at	SELENOI	selenoprotein I(SELENOI)
230992_at	BTNL9	butyrophilin like 9(BTNL9)
205046_at	CENPE	centromere protein E(CENPE)
204128_s_at	RFC3	replication factor C subunit 3(RFC3)
210517_s_at	AKAP12	A-kinase anchoring protein 12(AKAP12)
201369_s_at	ZFP36L2	ZFP36 ring finger protein like 2(ZFP36L2)
225282_at	SMAP2	small ArfGAP2(SMAP2)
204444_at	KIF11	kinesin family member 11(KIF11)
211713_x_at	KIAA0101	KIAA0101(KIAA0101)
224753_at	CDCA5	cell division cycle associated 5(CDCA5)
223344_s_at	MS4A7	membrane spanning 4-domains A7(MS4A7)
214445_at	ELL2	elongation factor for RNA polymerase II 2(ELL2)
204822_at	TTK	TTK protein kinase(TTK)
225949_at	MIR6845	microRNA 6845(MIR6845)
218355_at	KIF4A	kinesin family member 4A(KIF4A)
220285_at	ABHD17B	abhydrolase domain containing 17B(ABHD17B)
219306_at	KIF15	kinesin family member 15(KIF15)
201890_at	RRM2	ribonucleotide reductase regulatory subunit M2(RRM2)
206660_at	IGLL1	immunoglobulin lambda like polypeptide 1(IGLL1)
232164_s_at	EPPK1	epiplakin 1(EPPK1)
203723_at	ITPKB	inositol-trisphosphate 3-kinase B(ITPKB)
201292_at	TOP2A	topoisomerase (DNA) II alpha(TOP2A)
203560_at	GGH	gamma-glutamyl hydrolase(GGH)
215117_at	RAG2	recombination activating 2(RAG2)
225685_at	CDC42EP3	CDC42 effector protein 3(CDC42EP3)
205393_s_at	CHEK1	checkpoint kinase 1(CHEK1)
204768_s_at	FEN1	flap structure-specific endonuclease 1(FEN1)
39402_at	IL1B	interleukin 1 beta(IL1B)
238015_at	C4orf46	chromosome 4 open reading frame 46(C4orf46)
225834_at	FAM72C	family with sequence similarity 72 member C(FAM72C)

202887_s_at	DDIT4	DNA damage inducible transcript 4(DDIT4)
221521_s_at	GINS2	GINS complex subunit 2(GINS2)
207697_x_at	LILRB2	leukocyte immunoglobulin like receptor B2(LILRB2)
212141_at	MCM4	minichromosome maintenance complex component 4(MCM4)
204165_at	WASF1	WAS protein family member 1(WASF1)
209172_s_at	CENPF	centromere protein F(CENPF)
224140_at	NPCDR1	nasopharyngeal carcinoma, down-regulated 1(NPCDR1)
223276_at	SMIM3	small integral membrane protein 3(SMIM3)
203305_at	F13A1	coagulation factor XIII A chain(F13A1)
203418_at	CCNA2	cyclin A2(CCNA2)
218542_at	CEP55	centrosomal protein 55(CEP55)
218404_at	SNX10	sorting nexin 10(SNX10)
208078_s_at	SIK1	salt inducible kinase 1(SIK1)
219000_s_at	DSCC1	DNA replication and sister chromatid cohesion 1(DSCC1)
211088_s_at	PLK4	polo like kinase 4(PLK4)
1554732_at	LOC728175	uncharacterized LOC728175(LOC728175)
211424_x_at	METTL7A	methyltransferase like 7A(METTL7A)
202705_at	CCNB2	cyclin B2(CCNB2)
212021_s_at	MKI67	marker of proliferation Ki-67(MKI67)
221757_at	PIK3IP1	phosphoinositide-3-kinase interacting protein 1(PIK3IP1)
211814_s_at	CCNE2	cyclin E2(CCNE2)
215455_at	TIMELESS	timeless circadian clock(TIMELESS)
202954_at	UBE2C	ubiquitin conjugating enzyme E2 C(UBE2C)
205033_s_at	DEFA1	defensin alpha 1(DEFA1)
205339_at	STIL	SCL/TAL1 interrupting locus(STIL)
205098_at	CCR1	C-C motif chemokine receptor 1(CCR1)
209999_x_at	SOCS1	suppressor of cytokine signaling 1(SOCS1)
202338_at	TK1	thymidine kinase 1(TK1)
229610_at	CKAP2L	cytoskeleton associated protein 2 like(CKAP2L)
207746_at	POLQ	DNA polymerase theta(POLQ)
203708_at	PDE4B	phosphodiesterase 4B(PDE4B)
229551_x_at	ZNF367	zinc finger protein 367(ZNF367)

214744_s_at	SNORA21	small nucleolar RNA, H/ACA box 21(SNORA21)
203528_at	SEMA4D	semaphorin 4D(SEMA4D)
206618_at	IL18R1	interleukin 18 receptor 1(IL18R1)
217702_at	IL27RA	interleukin 27 receptor subunit alpha(IL27RA)
211080_s_at	NEK2	NIMA related kinase 2(NEK2)
213599_at	OIP5	Opa interacting protein 5(OIP5)
206499_s_at	RCC1	regulator of chromosome condensation 1(RCC1)
219262_at	SUV39H2	suppressor of variegation 3-9 homolog 2(SUV39H2)
201490_s_at	PPIF	peptidylprolyl isomerase F(PPIF)
211676_s_at	IFNGR1	interferon gamma receptor 1(IFNGR1)
223700_at	MND1	meiotic nuclear divisions 1(MND1)
204825_at	MELK	maternal embryonic leucine zipper kinase(MELK)
213515_x_at	HBG1	hemoglobin subunit gamma 1(HBG1)
219243_at	GIMAP4	GTPase, IMAP family member 4(GIMAP4)
209714_s_at	CDKN3	cyclin dependent kinase inhibitor 3(CDKN3)
49306_at	RASSF4	Ras association domain family member 4(RASSF4)
239680_at	WDR76	WD repeat domain 76(WDR76)
203695_s_at	DFNA5	DFNA5, deafness associated tumor suppressor(DFNA5)
212282_at	TMEM97	transmembrane protein 97(TMEM97)
1555270_a_at	WFS1	wolframin ER transmembrane glycoprotein(WFS1)
203761_at	SLA	Src-like-adaptor(SLA)
218585_s_at	DTL	denticleless E3 ubiquitin protein ligase homolog(DTL)
226980_at	DEPDC1B	DEP domain containing 1B(DEPDC1B)
218726_at	HJURP	Holliday junction recognition protein(HJURP)
202589_at	TYMS	thymidylate synthetase(TYMS)
218782_s_at	ATAD2	ATPase family, AAA domain containing 2(ATAD2)
231094_s_at	LOC100996643	monofunctional C1-tetrahydrofolate synthase, mitochondrial-like(LOC100996643)
233813_at	PPP1R16B	protein phosphatase 1 regulatory subunit 16B(PPP1R16B)
222039_at	KIF18B	kinesin family member 18B(KIF18B)
1569062_s_at	IQGAP3	IQ motif containing GTPase activating protein 3(IQGAP3)
203755_at	BUB1B	BUB1 mitotic checkpoint serine/threonine kinase B(BUB1B)

1558686_at	MPV17L	MPV17 mitochondrial inner membrane protein like(MPV17L)
244615_x_at	TARSL2	threonyl-tRNA synthetase like 2(TARSL2)
204146_at	RAD51AP1	RAD51 associated protein 1(RAD51AP1)
205983_at	DPEP1	dipeptidase 1 (renal)(DPEP1)
1559297_at	SNX29P2	sorting nexin 29 pseudogene 2(SNX29P2)
218113_at	TMEM2	transmembrane protein 2(TMEM2)
239219_at	AURKB	aurora kinase B(AURKB)
1568830_at	IRAK3	interleukin 1 receptor associated kinase 3(IRAK3)
226936_at	CENPW	centromere protein W(CENPW)
205786_s_at	ITGAM	integrin subunit alpha M(ITGAM)
221258_s_at	KIF18A	kinesin family member 18A(KIF18A)
200660_at	S100A11	S100 calcium binding protein A11(S100A11)
226530_at	BMF	Bcl2 modifying factor(BMF)
231772_x_at	CENPH	centromere protein H(CENPH)
213975_s_at	LYZ	lysozyme(LYZ)
206102_at	GINS1	GINS complex subunit 1(GINS1)
206637_at	P2RY14	purinergic receptor P2Y14(P2RY14)
206584_at	LY96	lymphocyte antigen 96(LY96)
223229_at	UBE2T	ubiquitin conjugating enzyme E2 T(UBE2T)
224325_at	MIR4683	microRNA 4683(MIR4683)
212242_at	TUBA4A	tubulin alpha 4a(TUBA4A)
209035_at	MDK	midkine (neurite growth-promoting factor 2)(MDK)
203119_at	CCDC86	coiled-coil domain containing 86(CCDC86)
216237_s_at	MCM5	minichromosome maintenance complex component 5(MCM5)
200920_s_at	BTG1	BTG anti-proliferation factor 1(BTG1)
213273_at	TENM4	teneurin transmembrane protein 4(TENM4)
203554_x_at	PTTG1	pituitary tumor-transforming 1(PTTG1)
242730_at	MYRIP	myosin VIIA and Rab interacting protein(MYRIP)
204709_s_at	KIF23	kinesin family member 23(KIF23)
207072_at	IL18RAP	interleukin 18 receptor accessory protein(IL18RAP)
218115_at	ASF1B	anti-silencing function 1B histone chaperone(ASF1B)
1569496_s_at	LOC100130872	uncharacterized LOC100130872(LOC100130872)

201202_at	PCNA	proliferating cell nuclear antigen(PCNA)
1552619_a_at	ANLN	anillin actin binding protein(ANLN)
220448_at	KCNK12	potassium two pore domain channel subfamily K member 12(KCNK12)
202420_s_at	DHX9	DExH-box helicase 9(DHX9)
204126_s_at	CDC45	cell division cycle 45(CDC45)
219990_at	E2F8	E2F transcription factor 8(E2F8)
202917_s_at	S100A8	S100 calcium binding protein A8(S100A8)
208438_s_at	FGR	FGR proto-oncogene, Src family tyrosine kinase(FGR)
226456_at	RMI2	RecQ mediated genome instability 2(RMI2)
219230_at	TMEM100	transmembrane protein 100(TMEM100)

Appendix B: Fundamental Boolean Networks of Glucocorticoid-induced Leukaemia

The fundamental Boolean networks for the common genes listed in Appendix A

Fundamental Boolean Network with 285 genes

Genes involved:

ABHD17B, AKAP12, ANLN, ANP32E, APITD1-CORT, ARPP21, ARRDC3, ASF1B, ASPM, ATAD2, AURKA, AURKB, B3GNT2, BCAT1, BCL10, BCL2L11, BIRC3, BIRC5, BMF, BRCA1, BRIP1, BTG1, BTNL9, BUB1, BUB1B, BYSL, C4orf46, C5orf24, CCDC34, CCDC86, CCNA2, CCNB1, CCNB2, CCNE2, CCNL1, CCR1, CD53, CDC20, CDC42EP3, CDC45, CDC6, CDCA2, CDCA3, CDCA5, CDK1, CDK6, CDKN3, CDT1, CELF2, CENPA, CENPE, CENPF, CENPH, CENPK, CENPN, CENPU, CENPV, CENPW, CEP55, CHEK1, CKAP2, CKAP2L, CKS1B, CLEC2B, CLN8, CPM, CRNDE, DDIT4, DEFA1, DENND3, DEPDC1, DEPDC1B, DFNA5, DHFR, DHX9, DLGAP5, DPEP1, DSCC1, DTL, E2F7, E2F8, ECT2, EGR1, ELL2, EMP1, EPPK1, F13A1, FABP5, FAM72C, FANCI, FCER1G, FCGR3B, FEN1, FGD2, FGL2, FGR, FH, FOXM1, FUS, GBP4, GGH, GIMAP4, GIMAP7, GINS1, GINS2, GSN, GVINP1, HBB, HBG1, HELLS, HIST4H4, HJURP, HMMR, HRK, HSP90AB1, ID2, ID3, IFNGR1, IGH, IGLC1, IGLL1, IL18R1, IL18RAP, IL1B, IL27RA, IL6ST, IQGAP3, IRAK3, ISG20, ITGAM, ITGB2-AS1, ITPKB, KCNK12, KIAA0101, KIF11, KIF14, KIF15, KIF18A, KIF18B, KIF20A, KIF23, KIF26A, KIF2C, KIF4A, KLF9, KNL1, LEF1, LGALS3, LILRA1, LILRB2, LOC100130872, LOC100505650, LOC100996643, LOC285097, LOC728175, LY96, LYZ, MAD2L1, MCM10, MCM4, MCM5, MCM7, MDK, MDM2, MELK, METTL7A, MIR4683, MIR6845, MIR8071-1, MKI67, MND1, MNDA, MPV17L, MS4A1, MS4A4A, MS4A7, MSH6, MTHFD2, MTSS1, MYRIP, NCAPG, NCAPH, NEAT1, NEDD9, NEK2, NME1, NPCDR1, NUF2, NUSAP1, OIP5, P2RX5, P2RY14, PAICS, PBK, PCNA, PDE4B, PFKFB2, PHF19, PIK3IP1, PLK4, POLE2, POLQ, POU4F1, PPBP, PPIF, PPP1R16B, PRDM1, PRPS2, PRR11, PSPH, PTP4A1, PTTG1, RAB31, RAD51, RAD51AP1, RAG1, RAG2, RASSF4, RBM14, RBMS3, RCC1, RFC3, RHOBTB3, RMI2, RNASET2, RPS6KA2, RRM2, S100A11, S100A8, SCML4, SELENOI, SEMA4D, SERPINA1, SERPINB9, SESN1, SHCBP1, SIK1, SKP2, SLA, SLC22A23, SMAP2, SMC2, SMIM3, SNORA21, SNORD3B-1, SNTB2, SNX10, SNX29P2, SOCS1, SOS1, SQLE, STAB1, STIL, SUV39H2, TARSL2, TBXA2R, TCF19, TENM4, TIMELESS, TIPIN, TK1, TMEM100, TMEM2, TMEM97, TNFRSF21, TNFSF8, TOP2A, TPX2, TRIB1, TRIP13, TTK, TUBA4A, TXNIP, TYMS, UBE2C, UBE2T, WASF1, WDHD1, WDR76, WFS1, ZBTB16, ZFP36L2, ZNF367, ZWINT, FKBP5

Networks:

Multiple Transition Functions for ABHD17B with decay value = 1:

ABHD17B_1_Activator: ABHD17B = B3GNT2 (Confidence: 1, TimeStep: 1)

ABHD17B_2_Activator: ABHD17B = BMF (Confidence: 1, TimeStep: 1)

ABHD17B_3_Activator: ABHD17B = ABHD17B&!SEMA4D (Confidence: 1, TimeStep: 1)

ABHD17B_4_Activator: ABHD17B = DHX9 (Confidence: 1, TimeStep: 2)

ABHD17B_5_Activator: ABHD17B = HSP90AB1 (Confidence: 1, TimeStep: 2)

ABHD17B_1_Inhibitor: ABHD17B = !B3GNT2 (Confidence: 1, TimeStep: 2)

ABHD17B_2_Inhibitor: ABHD17B = !ABHD17B (Confidence: 1, TimeStep: 2)

ABHD17B_3_Inhibitor: ABHD17B = !L18RAP (Confidence: 1, TimeStep: 2)

ABHD17B_4_Inhibitor: ABHD17B = SEMA4D (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for AKAP12 with decay value = 1:

AKAP12_1_Activator: AKAP12 = RPS6KA2 (Confidence: 1, TimeStep: 1)

AKAP12_2_Activator: AKAP12 = AKAP12&RAG1 (Confidence: 1, TimeStep: 1)

AKAP12_3_Activator: AKAP12 = AKAP12&TMEM2 (Confidence: 1, TimeStep: 1)

AKAP12_4_Activator: AKAP12 = IGLL1&IRAK3 (Confidence: 1, TimeStep: 1)

AKAP12_5_Activator: AKAP12 = !CENPV&DPEP1 (Confidence: 1, TimeStep: 1)

AKAP12_1_Inhibitor: AKAP12 = !AKAP12 (Confidence: 1, TimeStep: 1)

AKAP12_2_Inhibitor: AKAP12 = !TMEM2 (Confidence: 1, TimeStep: 1)

AKAP12_3_Inhibitor: AKAP12 = !IGLL1 (Confidence: 1, TimeStep: 1)

AKAP12_4_Inhibitor: AKAP12 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

AKAP12_5_Inhibitor: AKAP12 = !RAG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ANLN with decay value = 1:

ANLN_1_Activator: ANLN = ATAD2 (Confidence: 1, TimeStep: 1)

ANLN_2_Activator: ANLN = BIRC5 (Confidence: 1, TimeStep: 1)

ANLN_3_Activator: ANLN = BUB1 (Confidence: 1, TimeStep: 1)

ANLN_4_Activator: ANLN = CCNA2 (Confidence: 1, TimeStep: 1)

ANLN_5_Activator: ANLN = CDCA5 (Confidence: 1, TimeStep: 1)

ANLN_1_Inhibitor: ANLN = !ATAD2 (Confidence: 1, TimeStep: 1)

ANLN_2_Inhibitor: ANLN = !BIRC5 (Confidence: 1, TimeStep: 1)

ANLN_3_Inhibitor: ANLN = !BUB1 (Confidence: 1, TimeStep: 1)

ANLN_4_Inhibitor: ANLN = !CCNA2 (Confidence: 1, TimeStep: 1)

ANLN_5_Inhibitor: ANLN = !CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ANP32E with decay value = 1:

ANP32E_1_Activator: ANP32E = APITD1-CORT (Confidence: 1, TimeStep: 1)

ANP32E_2_Activator: ANP32E = ASF1B (Confidence: 1, TimeStep: 1)

ANP32E_3_Activator: ANP32E = AURKA (Confidence: 1, TimeStep: 1)

ANP32E_4_Activator: ANP32E = !BTG1 (Confidence: 1, TimeStep: 1)

ANP32E_5_Activator: ANP32E = CCDC34 (Confidence: 1, TimeStep: 1)

ANP32E_1_Inhibitor: ANP32E = !CENPF (Confidence: 1, TimeStep: 1)

ANP32E_2_Inhibitor: ANP32E = !DEPDC1B (Confidence: 1, TimeStep: 1)

ANP32E_3_Inhibitor: ANP32E = !NEK2 (Confidence: 1, TimeStep: 1)

ANP32E_4_Inhibitor: ANP32E = !ANP32E (Confidence: 1, TimeStep: 1)

ANP32E_5_Inhibitor: ANP32E = !FH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for APITD1-CORT with decay value = 1:

APITD1-CORT_1_Activator: APITD1-CORT = APITD1-CORT (Confidence: 1, TimeStep: 1)

APITD1-CORT_2_Activator: APITD1-CORT = ASF1B (Confidence: 1, TimeStep: 1)

APITD1-CORT_3_Activator: APITD1-CORT = AURKA (Confidence: 1, TimeStep: 1)

APITD1-CORT_4_Activator: APITD1-CORT = !BTG1 (Confidence: 1, TimeStep: 1)

APITD1-CORT_5_Activator: APITD1-CORT = CCDC34 (Confidence: 1, TimeStep: 1)

APITD1-CORT_1_Inhibitor: APITD1-CORT = !APITD1-CORT (Confidence: 1, TimeStep: 1)

APITD1-CORT_2_Inhibitor: APITD1-CORT = !ASF1B (Confidence: 1, TimeStep: 1)

APITD1-CORT_3_Inhibitor: APITD1-CORT = !AURKA (Confidence: 1, TimeStep: 1)

APITD1-CORT_4_Inhibitor: APITD1-CORT = BTG1 (Confidence: 1, TimeStep: 1)

APITD1-CORT_5_Inhibitor: APITD1-CORT = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ARPP21 with decay value = 1:

ARPP21_1_Activator: ARPP21 = AKAP12&P2RY14 (Confidence: 1, TimeStep: 1)

ARPP21_2_Activator: ARPP21 = ID2&!ITPKB (Confidence: 1, TimeStep: 1)

ARPP21_3_Activator: ARPP21 = !CENPH&KCNK12 (Confidence: 1, TimeStep: 1)

ARPP21_4_Activator: ARPP21 = !FH&KCNK12 (Confidence: 1, TimeStep: 1)

ARPP21_5_Activator: ARPP21 = KCNK12&P2RY14 (Confidence: 1, TimeStep: 1)

ARPP21_1_Inhibitor: ARPP21 = !RAG1 (Confidence: 1, TimeStep: 1)

ARPP21_2_Inhibitor: ARPP21 = !AKAP12&!ARPP21 (Confidence: 1, TimeStep: 1)

ARPP21_3_Inhibitor: ARPP21 = !BMF&!LOC285097 (Confidence: 1, TimeStep: 1)

ARPP21_4_Inhibitor: ARPP21 = !LOC285097&!NPCDR1 (Confidence: 1, TimeStep: 1)

ARPP21_5_Inhibitor: ARPP21 = !AKAP12&!RAG2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ARRDC3 with decay value = 1:

ARRDC3_1_Activator: ARRDC3 = !B3GNT2&PRDM1 (Confidence: 1, TimeStep: 1)

ARRDC3_2_Activator: ARRDC3 = !B3GNT2&SOCS1 (Confidence: 1, TimeStep: 1)

ARRDC3_3_Activator: ARRDC3 = ARRDC3&FGL2 (Confidence: 1, TimeStep: 1)

ARRDC3_4_Activator: ARRDC3 = ARRDC3&PPBP (Confidence: 1, TimeStep: 1)

ARRDC3_5_Activator: ARRDC3 = !EMP1&FGL2 (Confidence: 1, TimeStep: 1)

ARRDC3_1_Inhibitor: ARRDC3 = POU4F1 (Confidence: 1, TimeStep: 1)

ARRDC3_2_Inhibitor: ARRDC3 = !SNX10 (Confidence: 1, TimeStep: 1)

ARRDC3_3_Inhibitor: ARRDC3 = !IL18RAP&!TNFSF8 (Confidence: 1, TimeStep: 1)

ARRDC3_4_Inhibitor: ARRDC3 = ID3&PRPS2 (Confidence: 1, TimeStep: 1)

ARRDC3_5_Inhibitor: ARRDC3 = !IL18R1&!TNFSF8 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ASF1B with decay value = 1:

ASF1B_1_Activator: ASF1B = APITD1-CORT (Confidence: 1, TimeStep: 1)

ASF1B_2_Activator: ASF1B = ASF1B (Confidence: 1, TimeStep: 1)

ASF1B_3_Activator: ASF1B = AURKA (Confidence: 1, TimeStep: 1)

ASF1B_4_Activator: ASF1B = !BTG1 (Confidence: 1, TimeStep: 1)

ASF1B_5_Activator: ASF1B = CCDC34 (Confidence: 1, TimeStep: 1)

ASF1B_1_Inhibitor: ASF1B = !APITD1-CORT (Confidence: 1, TimeStep: 1)

ASF1B_2_Inhibitor: ASF1B = !ASF1B (Confidence: 1, TimeStep: 1)

ASF1B_3_Inhibitor: ASF1B = !AURKA (Confidence: 1, TimeStep: 1)

ASF1B_4_Inhibitor: ASF1B = BTG1 (Confidence: 1, TimeStep: 1)

ASF1B_5_Inhibitor: ASF1B = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ASPM with decay value = 1:

ASPM_1_Activator: ASPM = CENPH (Confidence: 1, TimeStep: 1)

ASPM_2_Activator: ASPM = APITD1-CORT (Confidence: 1, TimeStep: 1)

ASPM_3_Activator: ASPM = ASF1B (Confidence: 1, TimeStep: 1)

ASPM_4_Activator: ASPM = AURKA (Confidence: 1, TimeStep: 1)

ASPM_5_Activator: ASPM = !BTG1 (Confidence: 1, TimeStep: 1)

ASPM_1_Inhibitor: ASPM = !FH (Confidence: 1, TimeStep: 1)

ASPM_2_Inhibitor: ASPM = !NUSAP1 (Confidence: 1, TimeStep: 1)

ASPM_3_Inhibitor: ASPM = !RFC3 (Confidence: 1, TimeStep: 1)

ASPM_4_Inhibitor: ASPM = !NUF2 (Confidence: 1, TimeStep: 1)

ASPM_5_Inhibitor: ASPM = !KIF11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ATAD2 with decay value = 1:

ATAD2_1_Activator: ATAD2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

ATAD2_2_Activator: ATAD2 = ASF1B (Confidence: 1, TimeStep: 1)

ATAD2_3_Activator: ATAD2 = AURKA (Confidence: 1, TimeStep: 1)

ATAD2_4_Activator: ATAD2 = !BTG1 (Confidence: 1, TimeStep: 1)

ATAD2_5_Activator: ATAD2 = CCDC34 (Confidence: 1, TimeStep: 1)

ATAD2_1_Inhibitor: ATAD2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

ATAD2_2_Inhibitor: ATAD2 = !ASF1B (Confidence: 1, TimeStep: 1)

ATAD2_3_Inhibitor: ATAD2 = !AURKA (Confidence: 1, TimeStep: 1)

ATAD2_4_Inhibitor: ATAD2 = BTG1 (Confidence: 1, TimeStep: 1)

ATAD2_5_Inhibitor: ATAD2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for AURKA with decay value = 1:

AURKA_1_Activator: AURKA = APITD1-CORT (Confidence: 1, TimeStep: 1)

AURKA_2_Activator: AURKA = ASF1B (Confidence: 1, TimeStep: 1)

AURKA_3_Activator: AURKA = AURKA (Confidence: 1, TimeStep: 1)

AURKA_4_Activator: AURKA = !BTG1 (Confidence: 1, TimeStep: 1)

AURKA_5_Activator: AURKA = CCDC34 (Confidence: 1, TimeStep: 1)

AURKA_1_Inhibitor: AURKA = !APITD1-CORT (Confidence: 1, TimeStep: 1)

AURKA_2_Inhibitor: AURKA = !ASF1B (Confidence: 1, TimeStep: 1)

AURKA_3_Inhibitor: AURKA = !AURKA (Confidence: 1, TimeStep: 1)

AURKA_4_Inhibitor: AURKA = BTG1 (Confidence: 1, TimeStep: 1)

AURKA_5_Inhibitor: AURKA = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for AURKB with decay value = 1:

AURKB_1_Activator: AURKB = APITD1-CORT (Confidence: 1, TimeStep: 1)

AURKB_2_Activator: AURKB = ASF1B (Confidence: 1, TimeStep: 1)

AURKB_3_Activator: AURKB = AURKA (Confidence: 1, TimeStep: 1)

AURKB_4_Activator: AURKB = !BTG1 (Confidence: 1, TimeStep: 1)

AURKB_5_Activator: AURKB = CCDC34 (Confidence: 1, TimeStep: 1)

AURKB_1_Inhibitor: AURKB = !APITD1-CORT (Confidence: 1, TimeStep: 1)

AURKB_2_Inhibitor: AURKB = !ASF1B (Confidence: 1, TimeStep: 1)

AURKB_3_Inhibitor: AURKB = !AURKA (Confidence: 1, TimeStep: 1)

AURKB_4_Inhibitor: AURKB = BTG1 (Confidence: 1, TimeStep: 1)

AURKB_5_Inhibitor: AURKB = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for B3GNT2 with decay value = 1:

B3GNT2_1_Activator: B3GNT2 = EMP1 (Confidence: 1, TimeStep: 1)

B3GNT2_2_Activator: B3GNT2 = B3GNT2&!HBG1 (Confidence: 1, TimeStep: 1)

B3GNT2_3_Activator: B3GNT2 = CDK6&DENND3 (Confidence: 1, TimeStep: 1)

B3GNT2_4_Activator: B3GNT2 = DENND3&LEF1 (Confidence: 1, TimeStep: 1)

B3GNT2_5_Activator: B3GNT2 = B3GNT2&DENND3 (Confidence: 1, TimeStep: 1)

B3GNT2_1_Inhibitor: B3GNT2 = !ABHD17B (Confidence: 1, TimeStep: 1)

B3GNT2_2_Inhibitor: B3GNT2 = SEMA4D (Confidence: 1, TimeStep: 1)

B3GNT2_3_Inhibitor: B3GNT2 = !WASF1 (Confidence: 1, TimeStep: 1)

B3GNT2_4_Inhibitor: B3GNT2 = !B3GNT2&!HRK (Confidence: 1, TimeStep: 1)

B3GNT2_5_Inhibitor: B3GNT2 = !EMP1&!MSH6 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BCAT1 with decay value = 1:

BCAT1_1_Activator: BCAT1 = !TXNIP (Confidence: 1, TimeStep: 1)

BCAT1_2_Activator: BCAT1 = CKAP2 (Confidence: 1, TimeStep: 1)

BCAT1_3_Activator: BCAT1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

BCAT1_4_Activator: BCAT1 = ASF1B (Confidence: 1, TimeStep: 1)

BCAT1_5_Activator: BCAT1 = AURKA (Confidence: 1, TimeStep: 1)

BCAT1_1_Inhibitor: BCAT1 = !BCAT1 (Confidence: 1, TimeStep: 1)

BCAT1_2_Inhibitor: BCAT1 = !LOC100996643 (Confidence: 1, TimeStep: 1)

BCAT1_3_Inhibitor: BCAT1 = !PAICS (Confidence: 1, TimeStep: 1)

BCAT1_4_Inhibitor: BCAT1 = CCR1 (Confidence: 1, TimeStep: 1)

BCAT1_5_Inhibitor: BCAT1 = !FABP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BCL10 with decay value = 1:

BCL10_1_Inhibitor: BCL10 = GIMAP4 (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for BCL2L11 with decay value = 1:

BCL2L11_1_Activator: BCL2L11 = !CDK6&!IL1B (Confidence: 1, TimeStep: 1)

BCL2L11_2_Activator: BCL2L11 = !CDK6&!DENND3 (Confidence: 1, TimeStep: 1)

BCL2L11_3_Activator: BCL2L11 = !ATAD2&BCL2L11 (Confidence: 1, TimeStep: 1)

BCL2L11_4_Activator: BCL2L11 = !AURKB&BCL2L11 (Confidence: 1, TimeStep: 1)

BCL2L11_5_Activator: BCL2L11 = BIRC3&CENPU (Confidence: 1, TimeStep: 1)

BCL2L11_1_Inhibitor: BCL2L11 = !DEPDC1B&!SLA (Confidence: 1, TimeStep: 1)

BCL2L11_2_Inhibitor: BCL2L11 = !BIRC3&DENND3 (Confidence: 1, TimeStep: 1)

BCL2L11_3_Inhibitor: BCL2L11 = !BIRC3&IL1B (Confidence: 1, TimeStep: 1)

BCL2L11_4_Inhibitor: BCL2L11 = !CENPF&!SLA (Confidence: 1, TimeStep: 1)

BCL2L11_5_Inhibitor: BCL2L11 = !KNL1&!SLA (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BIRC3 with decay value = 1:

BIRC3_1_Activator: BIRC3 = !EMP1&!SELENOI (Confidence: 1, TimeStep: 1)

BIRC3_2_Activator: BIRC3 = ARDC3&SERPINA1 (Confidence: 1, TimeStep: 1)

BIRC3_3_Activator: BIRC3 = FGR&IGH (Confidence: 1, TimeStep: 1)

BIRC3_4_Activator: BIRC3 = HBB&!SELENOI (Confidence: 1, TimeStep: 1)

BIRC3_5_Activator: BIRC3 = !BIRC3&!TYMS (Confidence: 1, TimeStep: 1)

BIRC3_1_Inhibitor: BIRC3 = LOC728175 (Confidence: 1, TimeStep: 1)

BIRC3_2_Inhibitor: BIRC3 = !HBG1&MYRIP (Confidence: 1, TimeStep: 1)

BIRC3_3_Inhibitor: BIRC3 = LOC100996643&MYRIP (Confidence: 1, TimeStep: 1)

BIRC3_4_Inhibitor: BIRC3 = EMP1&MYRIP (Confidence: 1, TimeStep: 1)

BIRC3_5_Inhibitor: BIRC3 = !HBB&MYRIP (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BIRC5 with decay value = 1:

BIRC5_1_Activator: BIRC5 = APITD1-CORT (Confidence: 1, TimeStep: 1)

BIRC5_2_Activator: BIRC5 = ASF1B (Confidence: 1, TimeStep: 1)

BIRC5_3_Activator: BIRC5 = AURKA (Confidence: 1, TimeStep: 1)

BIRC5_4_Activator: BIRC5 = !BTG1 (Confidence: 1, TimeStep: 1)

BIRC5_5_Activator: BIRC5 = CCDC34 (Confidence: 1, TimeStep: 1)

BIRC5_1_Inhibitor: BIRC5 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

BIRC5_2_Inhibitor: BIRC5 = !ASF1B (Confidence: 1, TimeStep: 1)

BIRC5_3_Inhibitor: BIRC5 = !AURKA (Confidence: 1, TimeStep: 1)

BIRC5_4_Inhibitor: BIRC5 = BTG1 (Confidence: 1, TimeStep: 1)

BIRC5_5_Inhibitor: BIRC5 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BMF with decay value = 1:

BMF_1_Activator: BMF = BMF (Confidence: 1, TimeStep: 1)

BMF_2_Activator: BMF = HRK (Confidence: 1, TimeStep: 1)

BMF_3_Activator: BMF = MIR4683 (Confidence: 1, TimeStep: 1)

BMF_4_Activator: BMF = RBMS3 (Confidence: 1, TimeStep: 1)

BMF_5_Activator: BMF = RPS6KA2 (Confidence: 1, TimeStep: 1)

BMF_1_Inhibitor: BMF = FOXM1 (Confidence: 1, TimeStep: 1)

BMF_2_Inhibitor: BMF = STIL (Confidence: 1, TimeStep: 1)

BMF_3_Inhibitor: BMF = ATAD2 (Confidence: 1, TimeStep: 1)

BMF_4_Inhibitor: BMF = BIRC5 (Confidence: 1, TimeStep: 1)

BMF_5_Inhibitor: BMF = BUB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BRCA1 with decay value = 1:

BRCA1_1_Activator: BRCA1 = !APITD1-CORT&BRIP1 (Confidence: 1, TimeStep: 1)

BRCA1_2_Activator: BRCA1 = !APITD1-CORT&CHEK1 (Confidence: 1, TimeStep: 1)

BRCA1_3_Activator: BRCA1 = !APITD1-CORT&FANCI (Confidence: 1, TimeStep: 1)

BRCA1_4_Activator: BRCA1 = !APITD1-CORT&TTK (Confidence: 1, TimeStep: 1)

BRCA1_5_Activator: BRCA1 = !ASF1B&BRIP1 (Confidence: 1, TimeStep: 1)

BRCA1_1_Inhibitor: BRCA1 = !BRCA1 (Confidence: 1, TimeStep: 1)

BRCA1_2_Inhibitor: BRCA1 = !BYSL (Confidence: 1, TimeStep: 1)

BRCA1_3_Inhibitor: BRCA1 = !FABP5 (Confidence: 1, TimeStep: 1)

BRCA1_4_Inhibitor: BRCA1 = GVINP1 (Confidence: 1, TimeStep: 1)

BRCA1_5_Inhibitor: BRCA1 = PRDM1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BRIP1 with decay value = 1:

BRIP1_1_Activator: BRIP1 = CCNB2&!P2RX5 (Confidence: 1, TimeStep: 1)

BRIP1_2_Activator: BRIP1 = CDC45&!P2RX5 (Confidence: 1, TimeStep: 1)

BRIP1_3_Activator: BRIP1 = CENPA&!P2RX5 (Confidence: 1, TimeStep: 1)

BRIP1_4_Activator: BRIP1 = DLGAP5&!P2RX5 (Confidence: 1, TimeStep: 1)

BRIP1_5_Activator: BRIP1 = MAD2L1&!P2RX5 (Confidence: 1, TimeStep: 1)

BRIP1_1_Inhibitor: BRIP1 = !KIF2C (Confidence: 1, TimeStep: 1)

BRIP1_2_Inhibitor: BRIP1 = !CCNB2 (Confidence: 1, TimeStep: 1)

BRIP1_3_Inhibitor: BRIP1 = !CDC45 (Confidence: 1, TimeStep: 1)

BRIP1_4_Inhibitor: BRIP1 = !CENPA (Confidence: 1, TimeStep: 1)

BRIP1_5_Inhibitor: BRIP1 = !DLGAP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BTG1 with decay value = 1:

BTG1_1_Activator: BTG1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

BTG1_2_Activator: BTG1 = !ASF1B (Confidence: 1, TimeStep: 1)

BTG1_3_Activator: BTG1 = !AURKA (Confidence: 1, TimeStep: 1)

BTG1_4_Activator: BTG1 = BTG1 (Confidence: 1, TimeStep: 1)

BTG1_5_Activator: BTG1 = !CCDC34 (Confidence: 1, TimeStep: 1)

BTG1_1_Inhibitor: BTG1 = UBE2C (Confidence: 1, TimeStep: 1)

BTG1_2_Inhibitor: BTG1 = ANLN&!B3GNT2 (Confidence: 1, TimeStep: 1)

BTG1_3_Inhibitor: BTG1 = ANP32E&!B3GNT2 (Confidence: 1, TimeStep: 1)

BTG1_4_Inhibitor: BTG1 = ASPM&!B3GNT2 (Confidence: 1, TimeStep: 1)

BTG1_5_Inhibitor: BTG1 = !B3GNT2&BIRC5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BTNL9 with decay value = 1:

BTNL9_1_Activator: BTNL9 = !CEP55&LOC285097 (Confidence: 1, TimeStep: 1)

BTNL9_2_Activator: BTNL9 = !BCL2L11&BTNL9 (Confidence: 1, TimeStep: 1)

BTNL9_3_Activator: BTNL9 = BMF&BTNL9 (Confidence: 1, TimeStep: 1)

BTNL9_4_Activator: BTNL9 = !CEP55&RPS6KA2 (Confidence: 1, TimeStep: 1)

BTNL9_5_Activator: BTNL9 = !DEPDC1B&RPS6KA2 (Confidence: 1, TimeStep: 1)

BTNL9_1_Inhibitor: BTNL9 = BCL2L11&!KIF26A (Confidence: 1, TimeStep: 1)

BTNL9_2_Inhibitor: BTNL9 = BCL2L11&!SIK1 (Confidence: 1, TimeStep: 1)

BTNL9_3_Inhibitor: BTNL9 = !IL1B&S100A11 (Confidence: 1, TimeStep: 1)

BTNL9_4_Inhibitor: BTNL9 = !KIF26A&!RNASET2 (Confidence: 1, TimeStep: 1)

BTNL9_5_Inhibitor: BTNL9 = !RBMS3&S100A11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BUB1 with decay value = 1:

BUB1_1_Activator: BUB1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

BUB1_2_Activator: BUB1 = ASF1B (Confidence: 1, TimeStep: 1)

BUB1_3_Activator: BUB1 = AURKA (Confidence: 1, TimeStep: 1)

BUB1_4_Activator: BUB1 = !BTG1 (Confidence: 1, TimeStep: 1)

BUB1_5_Activator: BUB1 = CCDC34 (Confidence: 1, TimeStep: 1)

BUB1_1_Inhibitor: BUB1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

BUB1_2_Inhibitor: BUB1 = !ASF1B (Confidence: 1, TimeStep: 1)

BUB1_3_Inhibitor: BUB1 = !AURKA (Confidence: 1, TimeStep: 1)

BUB1_4_Inhibitor: BUB1 = BTG1 (Confidence: 1, TimeStep: 1)

BUB1_5_Inhibitor: BUB1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BUB1B with decay value = 1:

BUB1B_1_Activator: BUB1B = CENPH (Confidence: 1, TimeStep: 1)

BUB1B_2_Activator: BUB1B = APITD1-CORT (Confidence: 1, TimeStep: 1)

BUB1B_3_Activator: BUB1B = ASF1B (Confidence: 1, TimeStep: 1)

BUB1B_4_Activator: BUB1B = AURKA (Confidence: 1, TimeStep: 1)

BUB1B_5_Activator: BUB1B = !BTG1 (Confidence: 1, TimeStep: 1)

BUB1B_1_Inhibitor: BUB1B = !CENPH (Confidence: 1, TimeStep: 1)

BUB1B_2_Inhibitor: BUB1B = !CDK1 (Confidence: 1, TimeStep: 1)

BUB1B_3_Inhibitor: BUB1B = !HMMR (Confidence: 1, TimeStep: 1)

BUB1B_4_Inhibitor: BUB1B = !KIF14 (Confidence: 1, TimeStep: 1)

BUB1B_5_Inhibitor: BUB1B = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for BYSL with decay value = 1:

BYSL_1_Activator: BYSL = ANLN&!PFKFB2 (Confidence: 1, TimeStep: 1)

BYSL_2_Activator: BYSL = BRIP1&HIST4H4 (Confidence: 1, TimeStep: 1)

BYSL_3_Activator: BYSL = BUB1B&HIST4H4 (Confidence: 1, TimeStep: 1)

BYSL_4_Activator: BYSL = C4orf46&HIST4H4 (Confidence: 1, TimeStep: 1)

BYSL_5_Activator: BYSL = CDC20&HIST4H4 (Confidence: 1, TimeStep: 1)

BYSL_1_Inhibitor: BYSL = !BYSL (Confidence: 1, TimeStep: 1)

BYSL_2_Inhibitor: BYSL = !PAICS (Confidence: 1, TimeStep: 1)

BYSL_3_Inhibitor: BYSL = !SELENOI (Confidence: 1, TimeStep: 1)

BYSL_4_Inhibitor: BYSL = !FABP5 (Confidence: 1, TimeStep: 1)

BYSL_5_Inhibitor: BYSL = GVINP1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for C4orf46 with decay value = 1:

C4orf46_1_Activator: C4orf46 = TMEM97 (Confidence: 1, TimeStep: 1)

C4orf46_2_Activator: C4orf46 = WDHD1 (Confidence: 1, TimeStep: 1)

C4orf46_3_Activator: C4orf46 = CENPH (Confidence: 1, TimeStep: 1)

C4orf46_4_Activator: C4orf46 = APITD1-CORT (Confidence: 1, TimeStep: 1)

C4orf46_5_Activator: C4orf46 = ASF1B (Confidence: 1, TimeStep: 1)

C4orf46_1_Inhibitor: C4orf46 = !TMEM97 (Confidence: 1, TimeStep: 1)

C4orf46_2_Inhibitor: C4orf46 = !WDHD1 (Confidence: 1, TimeStep: 1)

C4orf46_3_Inhibitor: C4orf46 = !CCNB1 (Confidence: 1, TimeStep: 1)

C4orf46_4_Inhibitor: C4orf46 = !IQGAP3 (Confidence: 1, TimeStep: 1)

C4orf46_5_Inhibitor: C4orf46 = !BUB1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for C5orf24 with decay value = 1:

C5orf24_1_Activator: C5orf24 = KIF4A (Confidence: 1, TimeStep: 1)

C5orf24_2_Activator: C5orf24 = CCNB1 (Confidence: 1, TimeStep: 1)

C5orf24_3_Activator: C5orf24 = CENPF (Confidence: 1, TimeStep: 1)

C5orf24_4_Activator: C5orf24 = IQGAP3 (Confidence: 1, TimeStep: 1)

C5orf24_5_Activator: C5orf24 = NEK2 (Confidence: 1, TimeStep: 1)

C5orf24_1_Inhibitor: C5orf24 = LGALS3 (Confidence: 1, TimeStep: 1)

C5orf24_2_Inhibitor: C5orf24 = LOC100130872 (Confidence: 1, TimeStep: 1)

C5orf24_3_Inhibitor: C5orf24 = !CKS1B&!PSPH (Confidence: 1, TimeStep: 1)

C5orf24_4_Inhibitor: C5orf24 = !BCAT1&HBB (Confidence: 1, TimeStep: 1)

C5orf24_5_Inhibitor: C5orf24 = !BCAT1&HBG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCDC34 with decay value = 1:

CCDC34_1_Activator: CCDC34 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CCDC34_2_Activator: CCDC34 = ASF1B (Confidence: 1, TimeStep: 1)

CCDC34_3_Activator: CCDC34 = AURKA (Confidence: 1, TimeStep: 1)

CCDC34_4_Activator: CCDC34 = !BTG1 (Confidence: 1, TimeStep: 1)

CCDC34_5_Activator: CCDC34 = CCDC34 (Confidence: 1, TimeStep: 1)

CCDC34_1_Inhibitor: CCDC34 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CCDC34_2_Inhibitor: CCDC34 = !ASF1B (Confidence: 1, TimeStep: 1)

CCDC34_3_Inhibitor: CCDC34 = !AURKA (Confidence: 1, TimeStep: 1)

CCDC34_4_Inhibitor: CCDC34 = BTG1 (Confidence: 1, TimeStep: 1)

CCDC34_5_Inhibitor: CCDC34 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCDC86 with decay value = 1:

CCDC86_1_Activator: CCDC86 = KIF2C (Confidence: 1, TimeStep: 1)

CCDC86_2_Activator: CCDC86 = !TXNIP (Confidence: 1, TimeStep: 1)

CCDC86_3_Activator: CCDC86 = ATAD2 (Confidence: 1, TimeStep: 1)

CCDC86_4_Activator: CCDC86 = BIRC5 (Confidence: 1, TimeStep: 1)

CCDC86_5_Activator: CCDC86 = BUB1 (Confidence: 1, TimeStep: 1)

CCDC86_1_Inhibitor: CCDC86 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

CCDC86_2_Inhibitor: CCDC86 = !BYSL (Confidence: 1, TimeStep: 1)

CCDC86_3_Inhibitor: CCDC86 = !PAICS (Confidence: 1, TimeStep: 1)

CCDC86_4_Inhibitor: CCDC86 = !SELENOI (Confidence: 1, TimeStep: 1)

CCDC86_5_Inhibitor: CCDC86 = CDC42EP3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCNA2 with decay value = 1:

CCNA2_1_Activator: CCNA2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNA2_2_Activator: CCNA2 = ASF1B (Confidence: 1, TimeStep: 1)

CCNA2_3_Activator: CCNA2 = AURKA (Confidence: 1, TimeStep: 1)

CCNA2_4_Activator: CCNA2 = !BTG1 (Confidence: 1, TimeStep: 1)

CCNA2_5_Activator: CCNA2 = CCDC34 (Confidence: 1, TimeStep: 1)

CCNA2_1_Inhibitor: CCNA2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNA2_2_Inhibitor: CCNA2 = !ASF1B (Confidence: 1, TimeStep: 1)

CCNA2_3_Inhibitor: CCNA2 = !AURKA (Confidence: 1, TimeStep: 1)

CCNA2_4_Inhibitor: CCNA2 = BTG1 (Confidence: 1, TimeStep: 1)

CCNA2_5_Inhibitor: CCNA2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCNB1 with decay value = 1:

CCNB1_1_Activator: CCNB1 = CENPH (Confidence: 1, TimeStep: 1)

CCNB1_2_Activator: CCNB1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNB1_3_Activator: CCNB1 = ASF1B (Confidence: 1, TimeStep: 1)

CCNB1_4_Activator: CCNB1 = AURKA (Confidence: 1, TimeStep: 1)

CCNB1_5_Activator: CCNB1 = !BTG1 (Confidence: 1, TimeStep: 1)

CCNB1_1_Inhibitor: CCNB1 = !CENPH (Confidence: 1, TimeStep: 1)

CCNB1_2_Inhibitor: CCNB1 = !CDK1 (Confidence: 1, TimeStep: 1)

CCNB1_3_Inhibitor: CCNB1 = !HMMR (Confidence: 1, TimeStep: 1)

CCNB1_4_Inhibitor: CCNB1 = !KIF14 (Confidence: 1, TimeStep: 1)

CCNB1_5_Inhibitor: CCNB1 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCNB2 with decay value = 1:

CCNB2_1_Activator: CCNB2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNB2_2_Activator: CCNB2 = ASF1B (Confidence: 1, TimeStep: 1)

CCNB2_3_Activator: CCNB2 = AURKA (Confidence: 1, TimeStep: 1)

CCNB2_4_Activator: CCNB2 = !BTG1 (Confidence: 1, TimeStep: 1)

CCNB2_5_Activator: CCNB2 = CCDC34 (Confidence: 1, TimeStep: 1)

CCNB2_1_Inhibitor: CCNB2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNB2_2_Inhibitor: CCNB2 = !ASF1B (Confidence: 1, TimeStep: 1)

CCNB2_3_Inhibitor: CCNB2 = !AURKA (Confidence: 1, TimeStep: 1)

CCNB2_4_Inhibitor: CCNB2 = BTG1 (Confidence: 1, TimeStep: 1)

CCNB2_5_Inhibitor: CCNB2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCNE2 with decay value = 1:

CCNE2_1_Activator: CCNE2 = !BMF&MDK (Confidence: 1, TimeStep: 1)

CCNE2_2_Activator: CCNE2 = CENPV&MDK (Confidence: 1, TimeStep: 1)

CCNE2_3_Activator: CCNE2 = E2F7&MDK (Confidence: 1, TimeStep: 1)

CCNE2_4_Activator: CCNE2 = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

CCNE2_5_Activator: CCNE2 = !IL1B&MDK (Confidence: 1, TimeStep: 1)

CCNE2_1_Inhibitor: CCNE2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CCNE2_2_Inhibitor: CCNE2 = !ASF1B (Confidence: 1, TimeStep: 1)

CCNE2_3_Inhibitor: CCNE2 = !AURKA (Confidence: 1, TimeStep: 1)

CCNE2_4_Inhibitor: CCNE2 = BTG1 (Confidence: 1, TimeStep: 1)

CCNE2_5_Inhibitor: CCNE2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCNL1 with decay value = 1:

CCNL1_1_Activator: CCNL1 = !BYSL&ELL2 (Confidence: 1, TimeStep: 1)

CCNL1_2_Activator: CCNL1 = !BYSL&CENPV (Confidence: 1, TimeStep: 1)

CCNL1_3_Activator: CCNL1 = !BYSL&!IRAK3 (Confidence: 1, TimeStep: 1)

CCNL1_4_Activator: CCNL1 = GINS2&!PCNA (Confidence: 1, TimeStep: 1)

CCNL1_5_Activator: CCNL1 = !ITGAM&!PCNA&SOCS1 (Confidence: 1, TimeStep: 1)

CCNL1_1_Inhibitor: CCNL1 = BRCA1 (Confidence: 1, TimeStep: 1)

CCNL1_2_Inhibitor: CCNL1 = PCNA (Confidence: 1, TimeStep: 1)

CCNL1_3_Inhibitor: CCNL1 = ITGAM (Confidence: 1, TimeStep: 1)

CCNL1_4_Inhibitor: CCNL1 = LOC285097 (Confidence: 1, TimeStep: 1)

CCNL1_5_Inhibitor: CCNL1 = TK1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CCR1 with decay value = 1:

CCR1_1_Activator: CCR1 = RAB31 (Confidence: 1, TimeStep: 1)

CCR1_2_Activator: CCR1 = !ASPM&CCR1 (Confidence: 1, TimeStep: 1)

CCR1_3_Activator: CCR1 = !ANP32E&MNDA (Confidence: 1, TimeStep: 1)

CCR1_4_Activator: CCR1 = !ASPM&MNDA (Confidence: 1, TimeStep: 1)

CCR1_5_Activator: CCR1 = !BYSL&PPBP (Confidence: 1, TimeStep: 1)

CCR1_1_Inhibitor: CCR1 = !MTSS1 (Confidence: 1, TimeStep: 1)

CCR1_2_Inhibitor: CCR1 = !SERPINB9 (Confidence: 1, TimeStep: 1)

CCR1_3_Inhibitor: CCR1 = !IRAK3 (Confidence: 1, TimeStep: 1)

CCR1_4_Inhibitor: CCR1 = !LILRB2 (Confidence: 1, TimeStep: 1)

CCR1_5_Inhibitor: CCR1 = E2F7 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CD53 with decay value = 1:

CD53_1_Activator: CD53 = SMAP2 (Confidence: 1, TimeStep: 1)

CD53_2_Activator: CD53 = CD53 (Confidence: 1, TimeStep: 1)

CD53_3_Activator: CD53 = ISG20 (Confidence: 1, TimeStep: 1)

CD53_4_Activator: CD53 = LILRB2 (Confidence: 1, TimeStep: 1)

CD53_5_Activator: CD53 = RNASET2 (Confidence: 1, TimeStep: 1)

CD53_1_Inhibitor: CD53 = UBE2C (Confidence: 1, TimeStep: 1)

CD53_2_Inhibitor: CD53 = ANLN&!GSN (Confidence: 1, TimeStep: 1)

CD53_3_Inhibitor: CD53 = ANLN&!KIF26A (Confidence: 1, TimeStep: 1)

CD53_4_Inhibitor: CD53 = ANLN&!IL6ST (Confidence: 1, TimeStep: 1)

CD53_5_Inhibitor: CD53 = ANP32E&!SIK1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDC20 with decay value = 1:

CDC20_1_Activator: CDC20 = CENPH (Confidence: 1, TimeStep: 1)

CDC20_2_Activator: CDC20 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDC20_3_Activator: CDC20 = ASF1B (Confidence: 1, TimeStep: 1)

CDC20_4_Activator: CDC20 = AURKA (Confidence: 1, TimeStep: 1)

CDC20_5_Activator: CDC20 = !BTG1 (Confidence: 1, TimeStep: 1)

CDC20_1_Inhibitor: CDC20 = !CENPH (Confidence: 1, TimeStep: 1)

CDC20_2_Inhibitor: CDC20 = !CDK1 (Confidence: 1, TimeStep: 1)

CDC20_3_Inhibitor: CDC20 = !HMMR (Confidence: 1, TimeStep: 1)

CDC20_4_Inhibitor: CDC20 = !KIF14 (Confidence: 1, TimeStep: 1)

CDC20_5_Inhibitor: CDC20 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDC42EP3 with decay value = 1:

CDC42EP3_1_Activator: CDC42EP3 = CDC42EP3 (Confidence: 1, TimeStep: 1)

CDC42EP3_2_Activator: CDC42EP3 = PPBP (Confidence: 1, TimeStep: 1)

CDC42EP3_3_Activator: CDC42EP3 = FGL2 (Confidence: 1, TimeStep: 1)

CDC42EP3_4_Activator: CDC42EP3 = LGALS3 (Confidence: 1, TimeStep: 1)

CDC42EP3_5_Activator: CDC42EP3 = RAB31 (Confidence: 1, TimeStep: 1)

CDC42EP3_1_Inhibitor: CDC42EP3 = CKS1B (Confidence: 1, TimeStep: 1)

CDC42EP3_2_Inhibitor: CDC42EP3 = !GBP4 (Confidence: 1, TimeStep: 1)

CDC42EP3_3_Inhibitor: CDC42EP3 = KNL1 (Confidence: 1, TimeStep: 1)

CDC42EP3_4_Inhibitor: CDC42EP3 = CDT1 (Confidence: 1, TimeStep: 1)

CDC42EP3_5_Inhibitor: CDC42EP3 = !NEAT1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDC45 with decay value = 1:

CDC45_1_Activator: CDC45 = !BMF&MDK (Confidence: 1, TimeStep: 1)

CDC45_2_Activator: CDC45 = CENPV&MDK (Confidence: 1, TimeStep: 1)

CDC45_3_Activator: CDC45 = E2F7&MDK (Confidence: 1, TimeStep: 1)

CDC45_4_Activator: CDC45 = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

CDC45_5_Activator: CDC45 = !IL1B&MDK (Confidence: 1, TimeStep: 1)

CDC45_1_Inhibitor: CDC45 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDC45_2_Inhibitor: CDC45 = !ASF1B (Confidence: 1, TimeStep: 1)

CDC45_3_Inhibitor: CDC45 = !AURKA (Confidence: 1, TimeStep: 1)

CDC45_4_Inhibitor: CDC45 = BTG1 (Confidence: 1, TimeStep: 1)

CDC45_5_Inhibitor: CDC45 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDC6 with decay value = 1:

CDC6_1_Activator: CDC6 = !BMF&MDK (Confidence: 1, TimeStep: 1)

CDC6_2_Activator: CDC6 = CENPV&MDK (Confidence: 1, TimeStep: 1)

CDC6_3_Activator: CDC6 = E2F7&MDK (Confidence: 1, TimeStep: 1)

CDC6_4_Activator: CDC6 = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

CDC6_5_Activator: CDC6 = !IL1B&MDK (Confidence: 1, TimeStep: 1)

CDC6_1_Inhibitor: CDC6 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDC6_2_Inhibitor: CDC6 = !ASF1B (Confidence: 1, TimeStep: 1)

CDC6_3_Inhibitor: CDC6 = !AURKA (Confidence: 1, TimeStep: 1)

CDC6_4_Inhibitor: CDC6 = BTG1 (Confidence: 1, TimeStep: 1)

CDC6_5_Inhibitor: CDC6 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDCA2 with decay value = 1:

CDCA2_1_Activator: CDCA2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA2_2_Activator: CDCA2 = ASF1B (Confidence: 1, TimeStep: 1)

CDCA2_3_Activator: CDCA2 = AURKA (Confidence: 1, TimeStep: 1)

CDCA2_4_Activator: CDCA2 = !BTG1 (Confidence: 1, TimeStep: 1)

CDCA2_5_Activator: CDCA2 = CCDC34 (Confidence: 1, TimeStep: 1)

CDCA2_1_Inhibitor: CDCA2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA2_2_Inhibitor: CDCA2 = !ASF1B (Confidence: 1, TimeStep: 1)

CDCA2_3_Inhibitor: CDCA2 = !AURKA (Confidence: 1, TimeStep: 1)

CDCA2_4_Inhibitor: CDCA2 = BTG1 (Confidence: 1, TimeStep: 1)

CDCA2_5_Inhibitor: CDCA2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDCA3 with decay value = 1:

CDCA3_1_Activator: CDCA3 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA3_2_Activator: CDCA3 = ASF1B (Confidence: 1, TimeStep: 1)

CDCA3_3_Activator: CDCA3 = AURKA (Confidence: 1, TimeStep: 1)

CDCA3_4_Activator: CDCA3 = !BTG1 (Confidence: 1, TimeStep: 1)

CDCA3_5_Activator: CDCA3 = CCDC34 (Confidence: 1, TimeStep: 1)

CDCA3_1_Inhibitor: CDCA3 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA3_2_Inhibitor: CDCA3 = !ASF1B (Confidence: 1, TimeStep: 1)

CDCA3_3_Inhibitor: CDCA3 = !AURKA (Confidence: 1, TimeStep: 1)

CDCA3_4_Inhibitor: CDCA3 = BTG1 (Confidence: 1, TimeStep: 1)

CDCA3_5_Inhibitor: CDCA3 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDCA5 with decay value = 1:

CDCA5_1_Activator: CDCA5 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA5_2_Activator: CDCA5 = ASF1B (Confidence: 1, TimeStep: 1)

CDCA5_3_Activator: CDCA5 = AURKA (Confidence: 1, TimeStep: 1)

CDCA5_4_Activator: CDCA5 = !BTG1 (Confidence: 1, TimeStep: 1)

CDCA5_5_Activator: CDCA5 = CCDC34 (Confidence: 1, TimeStep: 1)

CDCA5_1_Inhibitor: CDCA5 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDCA5_2_Inhibitor: CDCA5 = !ASF1B (Confidence: 1, TimeStep: 1)

CDCA5_3_Inhibitor: CDCA5 = !AURKA (Confidence: 1, TimeStep: 1)

CDCA5_4_Inhibitor: CDCA5 = BTG1 (Confidence: 1, TimeStep: 1)

CDCA5_5_Inhibitor: CDCA5 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDK1 with decay value = 1:

CDK1_1_Activator: CDK1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDK1_2_Activator: CDK1 = ASF1B (Confidence: 1, TimeStep: 1)

CDK1_3_Activator: CDK1 = AURKA (Confidence: 1, TimeStep: 1)

CDK1_4_Activator: CDK1 = !BTG1 (Confidence: 1, TimeStep: 1)

CDK1_5_Activator: CDK1 = CCDC34 (Confidence: 1, TimeStep: 1)

CDK1_1_Inhibitor: CDK1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDK1_2_Inhibitor: CDK1 = !ASF1B (Confidence: 1, TimeStep: 1)

CDK1_3_Inhibitor: CDK1 = !AURKA (Confidence: 1, TimeStep: 1)

CDK1_4_Inhibitor: CDK1 = BTG1 (Confidence: 1, TimeStep: 1)

CDK1_5_Inhibitor: CDK1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDK6 with decay value = 1:

CDK6_1_Activator: CDK6 = LEF1&!SNTB2 (Confidence: 1, TimeStep: 1)

CDK6_2_Activator: CDK6 = GIMAP4&!KLF9 (Confidence: 1, TimeStep: 1)

CDK6_3_Activator: CDK6 = !KLF9&!RHOBTB3 (Confidence: 1, TimeStep: 1)

CDK6_4_Activator: CDK6 = B3GNT2&!KLF9 (Confidence: 1, TimeStep: 1)

CDK6_5_Activator: CDK6 = !EMP1&!KLF9&!TRIB1 (Confidence: 1, TimeStep: 1)

CDK6_1_Inhibitor: CDK6 = !LEF1 (Confidence: 1, TimeStep: 1)

CDK6_2_Inhibitor: CDK6 = !ZFP36L2 (Confidence: 1, TimeStep: 1)

CDK6_3_Inhibitor: CDK6 = BCL2L11&KLF9 (Confidence: 1, TimeStep: 1)

CDK6_4_Inhibitor: CDK6 = BCL2L11&!RAG1 (Confidence: 1, TimeStep: 1)

CDK6_5_Inhibitor: CDK6 = !BCL2L11&!CDK6 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDKN3 with decay value = 1:

CDKN3_1_Activator: CDKN3 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CDKN3_2_Activator: CDKN3 = ASF1B (Confidence: 1, TimeStep: 1)

CDKN3_3_Activator: CDKN3 = AURKA (Confidence: 1, TimeStep: 1)

CDKN3_4_Activator: CDKN3 = !BTG1 (Confidence: 1, TimeStep: 1)

CDKN3_5_Activator: CDKN3 = CCDC34 (Confidence: 1, TimeStep: 1)

CDKN3_1_Inhibitor: CDKN3 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CDKN3_2_Inhibitor: CDKN3 = !ASF1B (Confidence: 1, TimeStep: 1)

CDKN3_3_Inhibitor: CDKN3 = !AURKA (Confidence: 1, TimeStep: 1)

CDKN3_4_Inhibitor: CDKN3 = BTG1 (Confidence: 1, TimeStep: 1)

CDKN3_5_Inhibitor: CDKN3 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CDT1 with decay value = 1:

CDT1_1_Activator: CDT1 = MDK&MKI67 (Confidence: 1, TimeStep: 1)

CDT1_2_Activator: CDT1 = MDK&NCAPG (Confidence: 1, TimeStep: 1)

CDT1_3_Activator: CDT1 = MDK&NEK2 (Confidence: 1, TimeStep: 1)

CDT1_4_Activator: CDT1 = MDK&NUSAP1 (Confidence: 1, TimeStep: 1)

CDT1_5_Activator: CDT1 = MDK&POLQ (Confidence: 1, TimeStep: 1)

CDT1_1_Inhibitor: CDT1 = !CCNB2 (Confidence: 1, TimeStep: 1)

CDT1_2_Inhibitor: CDT1 = !CDC45 (Confidence: 1, TimeStep: 1)

CDT1_3_Inhibitor: CDT1 = !CENPA (Confidence: 1, TimeStep: 1)

CDT1_4_Inhibitor: CDT1 = !DLGAP5 (Confidence: 1, TimeStep: 1)

CDT1_5_Inhibitor: CDT1 = !MAD2L1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CELF2 with decay value = 1:

CELF2_1_Activator: CELF2 = !CRNDE (Confidence: 1, TimeStep: 1)

CELF2_2_Activator: CELF2 = !SELENOI (Confidence: 1, TimeStep: 1)

CELF2_3_Activator: CELF2 = !DHX9 (Confidence: 1, TimeStep: 1)

CELF2_4_Activator: CELF2 = PPP1R16B (Confidence: 1, TimeStep: 1)

CELF2_5_Activator: CELF2 = !FABP5 (Confidence: 1, TimeStep: 1)

CELF2_1_Inhibitor: CELF2 = !CELF2&!DDIT4 (Confidence: 1, TimeStep: 1)

CELF2_2_Inhibitor: CELF2 = !DDIT4&IQGAP3 (Confidence: 1, TimeStep: 1)

CELF2_3_Inhibitor: CELF2 = !DDIT4&SHCBP1 (Confidence: 1, TimeStep: 1)

CELF2_4_Inhibitor: CELF2 = CDK1&!DDIT4 (Confidence: 1, TimeStep: 1)

CELF2_5_Inhibitor: CELF2 = CKAP2L&!DDIT4 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPA with decay value = 1:

CENPA_1_Activator: CENPA = APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPA_2_Activator: CENPA = ASF1B (Confidence: 1, TimeStep: 1)

CENPA_3_Activator: CENPA = AURKA (Confidence: 1, TimeStep: 1)

CENPA_4_Activator: CENPA = !BTG1 (Confidence: 1, TimeStep: 1)

CENPA_5_Activator: CENPA = CCDC34 (Confidence: 1, TimeStep: 1)

CENPA_1_Inhibitor: CENPA = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPA_2_Inhibitor: CENPA = !ASF1B (Confidence: 1, TimeStep: 1)

CENPA_3_Inhibitor: CENPA = !AURKA (Confidence: 1, TimeStep: 1)

CENPA_4_Inhibitor: CENPA = BTG1 (Confidence: 1, TimeStep: 1)

CENPA_5_Inhibitor: CENPA = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPE with decay value = 1:

CENPE_1_Activator: CENPE = APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPE_2_Activator: CENPE = ASF1B (Confidence: 1, TimeStep: 1)

CENPE_3_Activator: CENPE = AURKA (Confidence: 1, TimeStep: 1)

CENPE_4_Activator: CENPE = !BTG1 (Confidence: 1, TimeStep: 1)

CENPE_5_Activator: CENPE = CCDC34 (Confidence: 1, TimeStep: 1)

CENPE_1_Inhibitor: CENPE = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPE_2_Inhibitor: CENPE = !ASF1B (Confidence: 1, TimeStep: 1)

CENPE_3_Inhibitor: CENPE = !AURKA (Confidence: 1, TimeStep: 1)

CENPE_4_Inhibitor: CENPE = BTG1 (Confidence: 1, TimeStep: 1)

CENPE_5_Inhibitor: CENPE = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPF with decay value = 1:

CENPF_1_Activator: CENPF = APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPF_2_Activator: CENPF = ASF1B (Confidence: 1, TimeStep: 1)

CENPF_3_Activator: CENPF = AURKA (Confidence: 1, TimeStep: 1)

CENPF_4_Activator: CENPF = !BTG1 (Confidence: 1, TimeStep: 1)

CENPF_5_Activator: CENPF = CCDC34 (Confidence: 1, TimeStep: 1)

CENPF_1_Inhibitor: CENPF = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPF_2_Inhibitor: CENPF = !ASF1B (Confidence: 1, TimeStep: 1)

CENPF_3_Inhibitor: CENPF = !AURKA (Confidence: 1, TimeStep: 1)

CENPF_4_Inhibitor: CENPF = BTG1 (Confidence: 1, TimeStep: 1)

CENPF_5_Inhibitor: CENPF = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPH with decay value = 1:

CENPH_1_Activator: CENPH = !BMF&MDK (Confidence: 1, TimeStep: 1)

CENPH_2_Activator: CENPH = CENPV&MDK (Confidence: 1, TimeStep: 1)

CENPH_3_Activator: CENPH = E2F7&MDK (Confidence: 1, TimeStep: 1)

CENPH_4_Activator: CENPH = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

CENPH_5_Activator: CENPH = !IL1B&MDK (Confidence: 1, TimeStep: 1)

CENPH_1_Inhibitor: CENPH = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPH_2_Inhibitor: CENPH = !ASF1B (Confidence: 1, TimeStep: 1)

CENPH_3_Inhibitor: CENPH = !AURKA (Confidence: 1, TimeStep: 1)

CENPH_4_Inhibitor: CENPH = BTG1 (Confidence: 1, TimeStep: 1)

CENPH_5_Inhibitor: CENPH = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPK with decay value = 1:

CENPK_1_Activator: CENPK = !CDC42EP3&LOC100505650 (Confidence: 1, TimeStep: 1)

CENPK_2_Activator: CENPK = !CPM&LOC100505650 (Confidence: 1, TimeStep: 1)

CENPK_3_Activator: CENPK = CRNDE&LOC100505650 (Confidence: 1, TimeStep: 1)

CENPK_4_Activator: CENPK = !APITD1-CORT&ECT2 (Confidence: 1, TimeStep: 1)

CENPK_5_Activator: CENPK = !APITD1-CORT&!METTL7A (Confidence: 1, TimeStep: 1)

CENPK_1_Inhibitor: CENPK = !ECT2 (Confidence: 1, TimeStep: 1)

CENPK_2_Inhibitor: CENPK = !CENPK (Confidence: 1, TimeStep: 1)

CENPK_3_Inhibitor: CENPK = ISG20 (Confidence: 1, TimeStep: 1)

CENPK_4_Inhibitor: CENPK = GBP4 (Confidence: 1, TimeStep: 1)

CENPK_5_Inhibitor: CENPK = LILRB2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPN with decay value = 1:

CENPN_1_Activator: CENPN = CENPH (Confidence: 1, TimeStep: 1)

CENPN_2_Activator: CENPN = APITD1-CORT (Confidence: 1, TimeStep: 1)

CENPN_3_Activator: CENPN = ASF1B (Confidence: 1, TimeStep: 1)

CENPN_4_Activator: CENPN = AURKA (Confidence: 1, TimeStep: 1)

CENPN_5_Activator: CENPN = !BTG1 (Confidence: 1, TimeStep: 1)

CENPN_1_Inhibitor: CENPN = !CENPH (Confidence: 1, TimeStep: 1)

CENPN_2_Inhibitor: CENPN = !CDK1 (Confidence: 1, TimeStep: 1)

CENPN_3_Inhibitor: CENPN = !HMMR (Confidence: 1, TimeStep: 1)

CENPN_4_Inhibitor: CENPN = !KIF14 (Confidence: 1, TimeStep: 1)

CENPN_5_Inhibitor: CENPN = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPU with decay value = 1:

CENPU_1_Activator: CENPU = FOXM1 (Confidence: 1, TimeStep: 1)

CENPU_2_Activator: CENPU = KIF18B (Confidence: 1, TimeStep: 1)

CENPU_3_Activator: CENPU = STIL (Confidence: 1, TimeStep: 1)

CENPU_4_Activator: CENPU = UBE2T (Confidence: 1, TimeStep: 1)

CENPU_5_Activator: CENPU = ATAD2 (Confidence: 1, TimeStep: 1)

CENPU_1_Inhibitor: CENPU = CLEC2B (Confidence: 1, TimeStep: 1)

CENPU_2_Inhibitor: CENPU = TARSL2 (Confidence: 1, TimeStep: 1)

CENPU_3_Inhibitor: CENPU = !BYSL (Confidence: 1, TimeStep: 1)

CENPU_4_Inhibitor: CENPU = P2RY14 (Confidence: 1, TimeStep: 1)

CENPU_5_Inhibitor: CENPU = !PAICS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPV with decay value = 1:

CENPV_1_Activator: CENPV = ECT2 (Confidence: 1, TimeStep: 1)

CENPV_2_Activator: CENPV = FOXM1 (Confidence: 1, TimeStep: 1)

CENPV_3_Activator: CENPV = STIL (Confidence: 1, TimeStep: 1)

CENPV_4_Activator: CENPV = ATAD2 (Confidence: 1, TimeStep: 1)

CENPV_5_Activator: CENPV = BIRC5 (Confidence: 1, TimeStep: 1)

CENPV_1_Inhibitor: CENPV = !CENPV (Confidence: 1, TimeStep: 1)

CENPV_2_Inhibitor: CENPV = ITGAM (Confidence: 1, TimeStep: 1)

CENPV_3_Inhibitor: CENPV = MIR4683 (Confidence: 1, TimeStep: 1)

CENPV_4_Inhibitor: CENPV = RBMS3 (Confidence: 1, TimeStep: 1)

CENPV_5_Inhibitor: CENPV = IL18R1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CENPW with decay value = 1:

CENPW_1_Activator: CENPW = CDK1 (Confidence: 1, TimeStep: 1)
CENPW_2_Activator: CENPW = CKAP2L (Confidence: 1, TimeStep: 1)
CENPW_3_Activator: CENPW = HMMR (Confidence: 1, TimeStep: 1)
CENPW_4_Activator: CENPW = KIF14 (Confidence: 1, TimeStep: 1)
CENPW_5_Activator: CENPW = KIF20A (Confidence: 1, TimeStep: 1)
CENPW_1_Inhibitor: CENPW = !ANLN (Confidence: 1, TimeStep: 1)
CENPW_2_Inhibitor: CENPW = !PRR11 (Confidence: 1, TimeStep: 1)
CENPW_3_Inhibitor: CENPW = !CDC20 (Confidence: 1, TimeStep: 1)
CENPW_4_Inhibitor: CENPW = !KIF4A (Confidence: 1, TimeStep: 1)
CENPW_5_Inhibitor: CENPW = !MKI67 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CEP55 with decay value = 1:

CEP55_1_Activator: CEP55 = ATAD2 (Confidence: 1, TimeStep: 1)
CEP55_2_Activator: CEP55 = BIRC5 (Confidence: 1, TimeStep: 1)
CEP55_3_Activator: CEP55 = BUB1 (Confidence: 1, TimeStep: 1)
CEP55_4_Activator: CEP55 = CCNA2 (Confidence: 1, TimeStep: 1)
CEP55_5_Activator: CEP55 = CDCA5 (Confidence: 1, TimeStep: 1)
CEP55_1_Inhibitor: CEP55 = !ATAD2 (Confidence: 1, TimeStep: 1)
CEP55_2_Inhibitor: CEP55 = !BIRC5 (Confidence: 1, TimeStep: 1)
CEP55_3_Inhibitor: CEP55 = !BUB1 (Confidence: 1, TimeStep: 1)
CEP55_4_Inhibitor: CEP55 = !CCNA2 (Confidence: 1, TimeStep: 1)
CEP55_5_Inhibitor: CEP55 = !CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CHEK1 with decay value = 1:

CHEK1_1_Activator: CHEK1 = KIF2C (Confidence: 1, TimeStep: 1)
CHEK1_2_Activator: CHEK1 = ATAD2 (Confidence: 1, TimeStep: 1)
CHEK1_3_Activator: CHEK1 = BIRC5 (Confidence: 1, TimeStep: 1)
CHEK1_4_Activator: CHEK1 = BUB1 (Confidence: 1, TimeStep: 1)
CHEK1_5_Activator: CHEK1 = CCNA2 (Confidence: 1, TimeStep: 1)
CHEK1_1_Inhibitor: CHEK1 = !KIF2C (Confidence: 1, TimeStep: 1)
CHEK1_2_Inhibitor: CHEK1 = !CCNB2 (Confidence: 1, TimeStep: 1)
CHEK1_3_Inhibitor: CHEK1 = !CDC45 (Confidence: 1, TimeStep: 1)

CHEK1_4_Inhibitor: CHEK1 = !CENPA (Confidence: 1, TimeStep: 1)

CHEK1_5_Inhibitor: CHEK1 = !DLGAP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CKAP2 with decay value = 1:

CKAP2_1_Activator: CKAP2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

CKAP2_2_Activator: CKAP2 = ASF1B (Confidence: 1, TimeStep: 1)

CKAP2_3_Activator: CKAP2 = AURKA (Confidence: 1, TimeStep: 1)

CKAP2_4_Activator: CKAP2 = !BTG1 (Confidence: 1, TimeStep: 1)

CKAP2_5_Activator: CKAP2 = CCDC34 (Confidence: 1, TimeStep: 1)

CKAP2_1_Inhibitor: CKAP2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CKAP2_2_Inhibitor: CKAP2 = !ASF1B (Confidence: 1, TimeStep: 1)

CKAP2_3_Inhibitor: CKAP2 = !AURKA (Confidence: 1, TimeStep: 1)

CKAP2_4_Inhibitor: CKAP2 = BTG1 (Confidence: 1, TimeStep: 1)

CKAP2_5_Inhibitor: CKAP2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CKAP2L with decay value = 1:

CKAP2L_1_Activator: CKAP2L = APITD1-CORT (Confidence: 1, TimeStep: 1)

CKAP2L_2_Activator: CKAP2L = ASF1B (Confidence: 1, TimeStep: 1)

CKAP2L_3_Activator: CKAP2L = AURKA (Confidence: 1, TimeStep: 1)

CKAP2L_4_Activator: CKAP2L = !BTG1 (Confidence: 1, TimeStep: 1)

CKAP2L_5_Activator: CKAP2L = CCDC34 (Confidence: 1, TimeStep: 1)

CKAP2L_1_Inhibitor: CKAP2L = !APITD1-CORT (Confidence: 1, TimeStep: 1)

CKAP2L_2_Inhibitor: CKAP2L = !ASF1B (Confidence: 1, TimeStep: 1)

CKAP2L_3_Inhibitor: CKAP2L = !AURKA (Confidence: 1, TimeStep: 1)

CKAP2L_4_Inhibitor: CKAP2L = BTG1 (Confidence: 1, TimeStep: 1)

CKAP2L_5_Inhibitor: CKAP2L = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CKS1B with decay value = 1:

CKS1B_1_Activator: CKS1B = CCNB1 (Confidence: 1, TimeStep: 1)

CKS1B_2_Activator: CKS1B = CDK1 (Confidence: 1, TimeStep: 1)

CKS1B_3_Activator: CKS1B = HMMR (Confidence: 1, TimeStep: 1)

CKS1B_4_Activator: CKS1B = KIF14 (Confidence: 1, TimeStep: 1)

CKS1B_5_Activator: CKS1B = KIF20A (Confidence: 1, TimeStep: 1)

CKS1B_1_Inhibitor: CKS1B = !CCNB1 (Confidence: 1, TimeStep: 1)

CKS1B_2_Inhibitor: CKS1B = !BUB1B (Confidence: 1, TimeStep: 1)

CKS1B_3_Inhibitor: CKS1B = !CENPN (Confidence: 1, TimeStep: 1)

CKS1B_4_Inhibitor: CKS1B = !KIF15 (Confidence: 1, TimeStep: 1)

CKS1B_5_Inhibitor: CKS1B = !MCM10 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CLEC2B with decay value = 1:

CLEC2B_1_Activator: CLEC2B = LILRA1 (Confidence: 1, TimeStep: 1)

CLEC2B_2_Activator: CLEC2B = !APITD1-CORT&TUBA4A (Confidence: 1, TimeStep: 1)

CLEC2B_3_Activator: CLEC2B = !ANLN&TUBA4A (Confidence: 1, TimeStep: 1)

CLEC2B_4_Activator: CLEC2B = !APITD1-CORT&STAB1 (Confidence: 1, TimeStep: 1)

CLEC2B_5_Activator: CLEC2B = !ANLN&MPV17L (Confidence: 1, TimeStep: 1)

CLEC2B_1_Inhibitor: CLEC2B = CDT1 (Confidence: 1, TimeStep: 1)

CLEC2B_2_Inhibitor: CLEC2B = CENPU (Confidence: 1, TimeStep: 1)

CLEC2B_3_Inhibitor: CLEC2B = BRIP1 (Confidence: 1, TimeStep: 1)

CLEC2B_4_Inhibitor: CLEC2B = CHEK1 (Confidence: 1, TimeStep: 1)

CLEC2B_5_Inhibitor: CLEC2B = FANCI (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CLN8 with decay value = 1:

CLN8_1_Activator: CLN8 = !BMF&CLN8 (Confidence: 1, TimeStep: 1)

CLN8_1_Inhibitor: CLN8 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

CLN8_2_Inhibitor: CLN8 = !CLN8&!HRK (Confidence: 1, TimeStep: 1)

CLN8_3_Inhibitor: CLN8 = !BMF&!CLN8 (Confidence: 1, TimeStep: 1)

CLN8_4_Inhibitor: CLN8 = !BMF&!SMIM3 (Confidence: 1, TimeStep: 1)

CLN8_5_Inhibitor: CLN8 = !POU4F1&!SMIM3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CPM with decay value = 1:

CPM_1_Activator: CPM = CPM (Confidence: 1, TimeStep: 1)

CPM_2_Activator: CPM = !ARRDC3 (Confidence: 1, TimeStep: 2)

CPM_3_Activator: CPM = !LY96 (Confidence: 1, TimeStep: 2)

CPM_1_Inhibitor: CPM = !CD53 (Confidence: 1, TimeStep: 1)

CPM_2_Inhibitor: CPM = ECT2 (Confidence: 1, TimeStep: 1)

CPM_3_Inhibitor: CPM = GGH (Confidence: 1, TimeStep: 1)

CPM_4_Inhibitor: CPM = FOXM1 (Confidence: 1, TimeStep: 1)

CPM_5_Inhibitor: CPM = KIF2C (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for CRNDE with decay value = 1:

CRNDE_1_Activator: CRNDE = HELLS (Confidence: 1, TimeStep: 1)

CRNDE_2_Activator: CRNDE = !TMEM2 (Confidence: 1, TimeStep: 1)

CRNDE_3_Activator: CRNDE = ANP32E (Confidence: 1, TimeStep: 1)

CRNDE_4_Activator: CRNDE = ECT2 (Confidence: 1, TimeStep: 1)

CRNDE_5_Activator: CRNDE = FH (Confidence: 1, TimeStep: 1)

CRNDE_1_Inhibitor: CRNDE = !CRNDE (Confidence: 1, TimeStep: 1)

CRNDE_2_Inhibitor: CRNDE = LGALS3 (Confidence: 1, TimeStep: 1)

CRNDE_3_Inhibitor: CRNDE = RAB31 (Confidence: 1, TimeStep: 1)

CRNDE_4_Inhibitor: CRNDE = LILRA1 (Confidence: 1, TimeStep: 1)

CRNDE_5_Inhibitor: CRNDE = CDC42EP3&!LOC100996643 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DDIT4 with decay value = 1:

DDIT4_1_Activator: DDIT4 = RBMS3 (Confidence: 1, TimeStep: 1)

DDIT4_2_Activator: DDIT4 = PFKFB2 (Confidence: 1, TimeStep: 1)

DDIT4_3_Activator: DDIT4 = RPS6KA2 (Confidence: 1, TimeStep: 1)

DDIT4_4_Activator: DDIT4 = !CEP55&SMIM3 (Confidence: 1, TimeStep: 1)

DDIT4_5_Activator: DDIT4 = !CKAP2L&SMIM3 (Confidence: 1, TimeStep: 1)

DDIT4_1_Inhibitor: DDIT4 = !ZFP36L2 (Confidence: 1, TimeStep: 1)

DDIT4_2_Inhibitor: DDIT4 = HIST4H4&!SMIM3 (Confidence: 1, TimeStep: 1)

DDIT4_3_Inhibitor: DDIT4 = C4orf46&!SMIM3 (Confidence: 1, TimeStep: 1)

DDIT4_4_Inhibitor: DDIT4 = MKI67&!SMIM3 (Confidence: 1, TimeStep: 1)

DDIT4_5_Inhibitor: DDIT4 = PRR11&!SMIM3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DEFA1 with decay value = 1:

DEFA1_1_Activator: DEFA1 = !FH&LOC100505650 (Confidence: 1, TimeStep: 1)

DEFA1_2_Activator: DEFA1 = !CKS1B&LOC100505650 (Confidence: 1, TimeStep: 1)

DEFA1_3_Activator: DEFA1 = !LGALS3&LOC100130872 (Confidence: 1, TimeStep: 1)

DEFA1_4_Activator: DEFA1 = !IGH&LOC100130872 (Confidence: 1, TimeStep: 1)

DEFA1_5_Activator: DEFA1 = CKS1B&!IGLL1 (Confidence: 1, TimeStep: 1)

DEFA1_1_Inhibitor: DEFA1 = !HBB (Confidence: 1, TimeStep: 1)

DEFA1_2_Inhibitor: DEFA1 = !S100A8 (Confidence: 1, TimeStep: 1)

DEFA1_3_Inhibitor: DEFA1 = RFC3 (Confidence: 1, TimeStep: 1)

DEFA1_4_Inhibitor: DEFA1 = SHCBP1 (Confidence: 1, TimeStep: 1)

DEFA1_5_Inhibitor: DEFA1 = FH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DENND3 with decay value = 1:

DENND3_1_Activator: DENND3 = DENND3&!IL1B (Confidence: 1, TimeStep: 1)

DENND3_2_Activator: DENND3 = DENND3&!ITGB2-AS1 (Confidence: 1, TimeStep: 1)

DENND3_3_Activator: DENND3 = DENND3&!LOC100130872 (Confidence: 1, TimeStep: 1)

DENND3_4_Activator: DENND3 = DENND3&RAG1 (Confidence: 1, TimeStep: 1)

DENND3_5_Activator: DENND3 = DENND3&!SEMA4D (Confidence: 1, TimeStep: 1)

DENND3_1_Inhibitor: DENND3 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

DENND3_2_Inhibitor: DENND3 = LOC100130872 (Confidence: 1, TimeStep: 1)

DENND3_3_Inhibitor: DENND3 = !METTL7A (Confidence: 1, TimeStep: 1)

DENND3_4_Inhibitor: DENND3 = SEMA4D (Confidence: 1, TimeStep: 1)

DENND3_5_Inhibitor: DENND3 = !WASF1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DEPDC1 with decay value = 1:

DEPDC1_1_Activator: DEPDC1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

DEPDC1_2_Activator: DEPDC1 = ASF1B (Confidence: 1, TimeStep: 1)

DEPDC1_3_Activator: DEPDC1 = AURKA (Confidence: 1, TimeStep: 1)

DEPDC1_4_Activator: DEPDC1 = !BTG1 (Confidence: 1, TimeStep: 1)

DEPDC1_5_Activator: DEPDC1 = CCDC34 (Confidence: 1, TimeStep: 1)

DEPDC1_1_Inhibitor: DEPDC1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

DEPDC1_2_Inhibitor: DEPDC1 = !ASF1B (Confidence: 1, TimeStep: 1)

DEPDC1_3_Inhibitor: DEPDC1 = !AURKA (Confidence: 1, TimeStep: 1)

DEPDC1_4_Inhibitor: DEPDC1 = BTG1 (Confidence: 1, TimeStep: 1)

DEPDC1_5_Inhibitor: DEPDC1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DEPDC1B with decay value = 1:

DEPDC1B_1_Activator: DEPDC1B = APITD1-CORT (Confidence: 1, TimeStep: 1)

DEPDC1B_2_Activator: DEPDC1B = ASF1B (Confidence: 1, TimeStep: 1)

DEPDC1B_3_Activator: DEPDC1B = AURKA (Confidence: 1, TimeStep: 1)

DEPDC1B_4_Activator: DEPDC1B = !BTG1 (Confidence: 1, TimeStep: 1)

DEPDC1B_5_Activator: DEPDC1B = CCDC34 (Confidence: 1, TimeStep: 1)

DEPDC1B_1_Inhibitor: DEPDC1B = !CENPF (Confidence: 1, TimeStep: 1)

DEPDC1B_2_Inhibitor: DEPDC1B = !DEPDC1B (Confidence: 1, TimeStep: 1)

DEPDC1B_3_Inhibitor: DEPDC1B = !NEK2 (Confidence: 1, TimeStep: 1)

DEPDC1B_4_Inhibitor: DEPDC1B = !ANP32E (Confidence: 1, TimeStep: 1)

DEPDC1B_5_Inhibitor: DEPDC1B = !FH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DFNA5 with decay value = 1:

DFNA5_1_Activator: DFNA5 = DFNA5 (Confidence: 1, TimeStep: 1)

DFNA5_2_Activator: DFNA5 = DEPDC1B (Confidence: 1, TimeStep: 1)

DFNA5_3_Activator: DFNA5 = PTTG1 (Confidence: 1, TimeStep: 1)

DFNA5_4_Activator: DFNA5 = CKAP2L (Confidence: 1, TimeStep: 1)

DFNA5_5_Activator: DFNA5 = AURKB (Confidence: 1, TimeStep: 1)

DFNA5_1_Inhibitor: DFNA5 = !FABP5 (Confidence: 1, TimeStep: 1)

DFNA5_2_Inhibitor: DFNA5 = !HSP90AB1 (Confidence: 1, TimeStep: 1)

DFNA5_3_Inhibitor: DFNA5 = MPV17L (Confidence: 1, TimeStep: 1)

DFNA5_4_Inhibitor: DFNA5 = !IGLL1 (Confidence: 1, TimeStep: 1)

DFNA5_5_Inhibitor: DFNA5 = LGALS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DHFR with decay value = 1:

DHFR_1_Activator: DHFR = CDK1 (Confidence: 1, TimeStep: 1)

DHFR_2_Activator: DHFR = HMMR (Confidence: 1, TimeStep: 1)

DHFR_3_Activator: DHFR = KIF14 (Confidence: 1, TimeStep: 1)

DHFR_4_Activator: DHFR = KIF20A (Confidence: 1, TimeStep: 1)

DHFR_5_Activator: DHFR = POLQ (Confidence: 1, TimeStep: 1)

DHFR_1_Inhibitor: DHFR = !CDK1 (Confidence: 1, TimeStep: 1)

DHFR_2_Inhibitor: DHFR = !HMMR (Confidence: 1, TimeStep: 1)

DHFR_3_Inhibitor: DHFR = !KIF14 (Confidence: 1, TimeStep: 1)

DHFR_4_Inhibitor: DHFR = !KIF20A (Confidence: 1, TimeStep: 1)

DHFR_5_Inhibitor: DHFR = !POLQ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DHX9 with decay value = 1:

DHX9_1_Activator: DHX9 = KIF11 (Confidence: 1, TimeStep: 1)

DHX9_2_Activator: DHX9 = CDC20 (Confidence: 1, TimeStep: 1)

DHX9_3_Activator: DHX9 = KIF4A (Confidence: 1, TimeStep: 1)

DHX9_4_Activator: DHX9 = NUSAP1 (Confidence: 1, TimeStep: 1)

DHX9_5_Activator: DHX9 = ZWINT (Confidence: 1, TimeStep: 1)

DHX9_1_Inhibitor: DHX9 = LGALS3 (Confidence: 1, TimeStep: 1)

DHX9_2_Inhibitor: DHX9 = LILRA1 (Confidence: 1, TimeStep: 1)

DHX9_3_Inhibitor: DHX9 = HBB&!ZNF367 (Confidence: 1, TimeStep: 1)

DHX9_4_Inhibitor: DHX9 = !DTL&HBB (Confidence: 1, TimeStep: 1)

DHX9_5_Inhibitor: DHX9 = !GINS2&HBB (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DLGAP5 with decay value = 1:

DLGAP5_1_Activator: DLGAP5 = APITD1-CORT (Confidence: 1, TimeStep: 1)

DLGAP5_2_Activator: DLGAP5 = ASF1B (Confidence: 1, TimeStep: 1)

DLGAP5_3_Activator: DLGAP5 = AURKA (Confidence: 1, TimeStep: 1)

DLGAP5_4_Activator: DLGAP5 = !BTG1 (Confidence: 1, TimeStep: 1)

DLGAP5_5_Activator: DLGAP5 = CCDC34 (Confidence: 1, TimeStep: 1)

DLGAP5_1_Inhibitor: DLGAP5 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

DLGAP5_2_Inhibitor: DLGAP5 = !ASF1B (Confidence: 1, TimeStep: 1)

DLGAP5_3_Inhibitor: DLGAP5 = !AURKA (Confidence: 1, TimeStep: 1)

DLGAP5_4_Inhibitor: DLGAP5 = BTG1 (Confidence: 1, TimeStep: 1)

DLGAP5_5_Inhibitor: DLGAP5 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DPEP1 with decay value = 1:

DPEP1_1_Activator: DPEP1 = DPEP1 (Confidence: 1, TimeStep: 1)

DPEP1_2_Activator: DPEP1 = HRK (Confidence: 1, TimeStep: 1)

DPEP1_3_Activator: DPEP1 = POU4F1 (Confidence: 1, TimeStep: 1)
DPEP1_4_Activator: DPEP1 = RPS6KA2 (Confidence: 1, TimeStep: 1)
DPEP1_5_Activator: DPEP1 = ISG20&!LILRA1 (Confidence: 1, TimeStep: 1)
DPEP1_1_Inhibitor: DPEP1 = LOC100130872 (Confidence: 1, TimeStep: 1)
DPEP1_2_Inhibitor: DPEP1 = !METTL7A (Confidence: 1, TimeStep: 1)
DPEP1_3_Inhibitor: DPEP1 = LILRA1 (Confidence: 1, TimeStep: 1)
DPEP1_4_Inhibitor: DPEP1 = !ARPP21&!MSH6 (Confidence: 1, TimeStep: 1)
DPEP1_5_Inhibitor: DPEP1 = !ARPP21&!SIK1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DSCC1 with decay value = 1:

DSCC1_1_Activator: DSCC1 = APITD1-CORT (Confidence: 1, TimeStep: 1)
DSCC1_2_Activator: DSCC1 = ASF1B (Confidence: 1, TimeStep: 1)
DSCC1_3_Activator: DSCC1 = AURKA (Confidence: 1, TimeStep: 1)
DSCC1_4_Activator: DSCC1 = !BTG1 (Confidence: 1, TimeStep: 1)
DSCC1_5_Activator: DSCC1 = CCDC34 (Confidence: 1, TimeStep: 1)
DSCC1_1_Inhibitor: DSCC1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)
DSCC1_2_Inhibitor: DSCC1 = !ASF1B (Confidence: 1, TimeStep: 1)
DSCC1_3_Inhibitor: DSCC1 = !AURKA (Confidence: 1, TimeStep: 1)
DSCC1_4_Inhibitor: DSCC1 = BTG1 (Confidence: 1, TimeStep: 1)
DSCC1_5_Inhibitor: DSCC1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for DTL with decay value = 1:

DTL_1_Activator: DTL = ANLN (Confidence: 1, TimeStep: 1)
DTL_2_Activator: DTL = CEP55 (Confidence: 1, TimeStep: 1)
DTL_3_Activator: DTL = CCNB2 (Confidence: 1, TimeStep: 1)
DTL_4_Activator: DTL = CDC45 (Confidence: 1, TimeStep: 1)
DTL_5_Activator: DTL = CENPA (Confidence: 1, TimeStep: 1)
DTL_1_Inhibitor: DTL = !ZNF367 (Confidence: 1, TimeStep: 1)
DTL_2_Inhibitor: DTL = !E2F8 (Confidence: 1, TimeStep: 1)
DTL_3_Inhibitor: DTL = !GINS2 (Confidence: 1, TimeStep: 1)
DTL_4_Inhibitor: DTL = !DTL (Confidence: 1, TimeStep: 1)
DTL_5_Inhibitor: DTL = !MCM7 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for E2F7 with decay value = 1:

E2F7_1_Activator: E2F7 = ATAD2 (Confidence: 1, TimeStep: 1)

E2F7_2_Activator: E2F7 = BIRC5 (Confidence: 1, TimeStep: 1)

E2F7_3_Activator: E2F7 = BUB1 (Confidence: 1, TimeStep: 1)

E2F7_4_Activator: E2F7 = CCNA2 (Confidence: 1, TimeStep: 1)

E2F7_5_Activator: E2F7 = CDCA5 (Confidence: 1, TimeStep: 1)

E2F7_1_Inhibitor: E2F7 = !CKS1B (Confidence: 1, TimeStep: 1)

E2F7_2_Inhibitor: E2F7 = GIMAP4 (Confidence: 1, TimeStep: 1)

E2F7_3_Inhibitor: E2F7 = LILRB2 (Confidence: 1, TimeStep: 1)

E2F7_4_Inhibitor: E2F7 = RNASET2 (Confidence: 1, TimeStep: 1)

E2F7_5_Inhibitor: E2F7 = !TIMELESS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for E2F8 with decay value = 1:

E2F8_1_Activator: E2F8 = CKAP2L (Confidence: 1, TimeStep: 1)

E2F8_2_Activator: E2F8 = KIF18B (Confidence: 1, TimeStep: 1)

E2F8_3_Activator: E2F8 = UBE2T (Confidence: 1, TimeStep: 1)

E2F8_4_Activator: E2F8 = ATAD2 (Confidence: 1, TimeStep: 1)

E2F8_5_Activator: E2F8 = AURKB (Confidence: 1, TimeStep: 1)

E2F8_1_Inhibitor: E2F8 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

E2F8_2_Inhibitor: E2F8 = !BYSL (Confidence: 1, TimeStep: 1)

E2F8_3_Inhibitor: E2F8 = !SELENOI (Confidence: 1, TimeStep: 1)

E2F8_4_Inhibitor: E2F8 = CCR1 (Confidence: 1, TimeStep: 1)

E2F8_5_Inhibitor: E2F8 = !PTP4A1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ECT2 with decay value = 1:

ECT2_1_Activator: ECT2 = ECT2 (Confidence: 1, TimeStep: 1)

ECT2_2_Activator: ECT2 = FOXM1 (Confidence: 1, TimeStep: 1)

ECT2_3_Activator: ECT2 = STIL (Confidence: 1, TimeStep: 1)

ECT2_4_Activator: ECT2 = ATAD2 (Confidence: 1, TimeStep: 1)

ECT2_5_Activator: ECT2 = BIRC5 (Confidence: 1, TimeStep: 1)

ECT2_1_Inhibitor: ECT2 = !ECT2 (Confidence: 1, TimeStep: 1)

ECT2_2_Inhibitor: ECT2 = !CENPK (Confidence: 1, TimeStep: 1)

ECT2_3_Inhibitor: ECT2 = ISG20 (Confidence: 1, TimeStep: 1)

ECT2_4_Inhibitor: ECT2 = GBP4 (Confidence: 1, TimeStep: 1)

ECT2_5_Inhibitor: ECT2 = LILRB2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for EGR1 with decay value = 1:

EGR1_1_Activator: EGR1 = !DEPDC1B&EGR1&PAICS (Confidence: 1, TimeStep: 1)

EGR1_2_Activator: EGR1 = !LYZ (Confidence: 1, TimeStep: 2)

EGR1_3_Activator: EGR1 = GGH (Confidence: 1, TimeStep: 2)

EGR1_4_Activator: EGR1 = !S100A8 (Confidence: 1, TimeStep: 2)

EGR1_1_Inhibitor: EGR1 = DEPDC1B (Confidence: 1, TimeStep: 1)

EGR1_2_Inhibitor: EGR1 = !GGH&LYZ (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for ELL2 with decay value = 1:

ELL2_1_Activator: ELL2 = ELL2 (Confidence: 1, TimeStep: 1)

ELL2_2_Activator: ELL2 = KLF9&!NEDD9 (Confidence: 1, TimeStep: 1)

ELL2_3_Activator: ELL2 = !GIMAP7&KLF9 (Confidence: 1, TimeStep: 1)

ELL2_4_Activator: ELL2 = !CELF2 (Confidence: 1, TimeStep: 2)

ELL2_1_Inhibitor: ELL2 = LOC100130872 (Confidence: 1, TimeStep: 1)

ELL2_2_Inhibitor: ELL2 = CELF2&!KLF9 (Confidence: 1, TimeStep: 1)

ELL2_3_Inhibitor: ELL2 = CELF2&!ELL2&GIMAP7 (Confidence: 1, TimeStep: 1)

ELL2_4_Inhibitor: ELL2 = !PAICS (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for EMP1 with decay value = 1:

EMP1_1_Activator: EMP1 = !BCL2L11&EMP1 (Confidence: 1, TimeStep: 1)

EMP1_2_Activator: EMP1 = !CENPU&EMP1 (Confidence: 1, TimeStep: 1)

EMP1_3_Activator: EMP1 = DENND3&EMP1 (Confidence: 1, TimeStep: 1)

EMP1_4_Activator: EMP1 = !E2F7&EMP1 (Confidence: 1, TimeStep: 1)

EMP1_5_Activator: EMP1 = !ECT2&EMP1 (Confidence: 1, TimeStep: 1)

EMP1_1_Inhibitor: EMP1 = !EMP1 (Confidence: 1, TimeStep: 1)

EMP1_2_Inhibitor: EMP1 = !IL1B (Confidence: 1, TimeStep: 1)

EMP1_3_Inhibitor: EMP1 = !DENND3 (Confidence: 1, TimeStep: 1)

EMP1_4_Inhibitor: EMP1 = !B3GNT2 (Confidence: 1, TimeStep: 1)

EMP1_5_Inhibitor: EMP1 = BCL2L11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for EPPK1 with decay value = 1:

EPPK1_1_Activator: EPPK1 = BTNL9&!CRNDE (Confidence: 1, TimeStep: 1)

EPPK1_2_Activator: EPPK1 = BTNL9&CDC42EP3 (Confidence: 1, TimeStep: 1)

EPPK1_3_Activator: EPPK1 = CLN8&!SNTB2 (Confidence: 1, TimeStep: 1)

EPPK1_4_Activator: EPPK1 = !CPM&EPPK1 (Confidence: 1, TimeStep: 1)

EPPK1_5_Activator: EPPK1 = !CPM&!CRNDE&P2RX5 (Confidence: 1, TimeStep: 1)

EPPK1_1_Inhibitor: EPPK1 = BCL2L11 (Confidence: 1, TimeStep: 1)

EPPK1_2_Inhibitor: EPPK1 = !IRAK3 (Confidence: 1, TimeStep: 1)

EPPK1_3_Inhibitor: EPPK1 = !LILRB2 (Confidence: 1, TimeStep: 1)

EPPK1_4_Inhibitor: EPPK1 = S100A11 (Confidence: 1, TimeStep: 1)

EPPK1_5_Inhibitor: EPPK1 = CKS1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for F13A1 with decay value = 1:

F13A1_1_Activator: F13A1 = F13A1 (Confidence: 1, TimeStep: 1)

F13A1_2_Activator: F13A1 = !BYSL&FGL2 (Confidence: 1, TimeStep: 1)

F13A1_3_Activator: F13A1 = CD53&FGL2 (Confidence: 1, TimeStep: 1)

F13A1_4_Activator: F13A1 = CDC42EP3&FGD2 (Confidence: 1, TimeStep: 1)

F13A1_5_Activator: F13A1 = CDC42EP3&!RHOBTB3 (Confidence: 1, TimeStep: 1)

F13A1_1_Inhibitor: F13A1 = !MIR8071-1 (Confidence: 1, TimeStep: 1)

F13A1_2_Inhibitor: F13A1 = !MTSS1 (Confidence: 1, TimeStep: 1)

F13A1_3_Inhibitor: F13A1 = !PDE4B (Confidence: 1, TimeStep: 1)

F13A1_4_Inhibitor: F13A1 = !SERPINB9 (Confidence: 1, TimeStep: 1)

F13A1_5_Inhibitor: F13A1 = CCDC86 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FABP5 with decay value = 1:

FABP5_1_Activator: FABP5 = NME1 (Confidence: 1, TimeStep: 1)

FABP5_2_Activator: FABP5 = KIF11 (Confidence: 1, TimeStep: 1)

FABP5_3_Activator: FABP5 = MTHFD2 (Confidence: 1, TimeStep: 1)

FABP5_4_Activator: FABP5 = CDC20 (Confidence: 1, TimeStep: 1)

FABP5_5_Activator: FABP5 = KIF4A (Confidence: 1, TimeStep: 1)

FABP5_1_Inhibitor: FABP5 = LGALS3 (Confidence: 1, TimeStep: 1)

FABP5_2_Inhibitor: FABP5 = RAB31 (Confidence: 1, TimeStep: 1)

FABP5_3_Inhibitor: FABP5 = !CCDC86&HBG1 (Confidence: 1, TimeStep: 1)

FABP5_4_Inhibitor: FABP5 = HBG1&!RBM14 (Confidence: 1, TimeStep: 1)

FABP5_5_Inhibitor: FABP5 = !BYSL&HBG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FAM72C with decay value = 1:

FAM72C_1_Activator: FAM72C = APITD1-CORT (Confidence: 1, TimeStep: 1)

FAM72C_2_Activator: FAM72C = ASF1B (Confidence: 1, TimeStep: 1)

FAM72C_3_Activator: FAM72C = AURKA (Confidence: 1, TimeStep: 1)

FAM72C_4_Activator: FAM72C = !BTG1 (Confidence: 1, TimeStep: 1)

FAM72C_5_Activator: FAM72C = CCDC34 (Confidence: 1, TimeStep: 1)

FAM72C_1_Inhibitor: FAM72C = !APITD1-CORT (Confidence: 1, TimeStep: 1)

FAM72C_2_Inhibitor: FAM72C = !ASF1B (Confidence: 1, TimeStep: 1)

FAM72C_3_Inhibitor: FAM72C = !AURKA (Confidence: 1, TimeStep: 1)

FAM72C_4_Inhibitor: FAM72C = BTG1 (Confidence: 1, TimeStep: 1)

FAM72C_5_Inhibitor: FAM72C = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FANCI with decay value = 1:

FANCI_1_Activator: FANCI = FOXM1 (Confidence: 1, TimeStep: 1)

FANCI_2_Activator: FANCI = KIF2C (Confidence: 1, TimeStep: 1)

FANCI_3_Activator: FANCI = STIL (Confidence: 1, TimeStep: 1)

FANCI_4_Activator: FANCI = ATAD2 (Confidence: 1, TimeStep: 1)

FANCI_5_Activator: FANCI = BIRC5 (Confidence: 1, TimeStep: 1)

FANCI_1_Inhibitor: FANCI = !ANLN (Confidence: 1, TimeStep: 1)

FANCI_2_Inhibitor: FANCI = !BRIP1 (Confidence: 1, TimeStep: 1)

FANCI_3_Inhibitor: FANCI = !CHEK1 (Confidence: 1, TimeStep: 1)

FANCI_4_Inhibitor: FANCI = !FANCI (Confidence: 1, TimeStep: 1)

FANCI_5_Inhibitor: FANCI = !HELLS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FCER1G with decay value = 1:

FCER1G_1_Activator: FCER1G = LILRA1 (Confidence: 1, TimeStep: 1)
FCER1G_2_Activator: FCER1G = GVINP1&IGH (Confidence: 1, TimeStep: 1)
FCER1G_3_Activator: FCER1G = GVINP1&IL18R1 (Confidence: 1, TimeStep: 1)
FCER1G_4_Activator: FCER1G = GVINP1&!PRPS2 (Confidence: 1, TimeStep: 1)
FCER1G_5_Activator: FCER1G = BIRC3&FGL2 (Confidence: 1, TimeStep: 1)
FCER1G_1_Inhibitor: FCER1G = IQGAP3 (Confidence: 1, TimeStep: 1)
FCER1G_2_Inhibitor: FCER1G = TMEM97 (Confidence: 1, TimeStep: 1)
FCER1G_3_Inhibitor: FCER1G = WDHD1 (Confidence: 1, TimeStep: 1)
FCER1G_4_Inhibitor: FCER1G = CENPH (Confidence: 1, TimeStep: 1)
FCER1G_5_Inhibitor: FCER1G = APITD1-CORT (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FCGR3B with decay value = 1:

FCGR3B_1_Activator: FCGR3B = FGL2 (Confidence: 1, TimeStep: 1)
FCGR3B_2_Activator: FCGR3B = LILRA1 (Confidence: 1, TimeStep: 1)
FCGR3B_3_Activator: FCGR3B = !E2F7&FCGR3B (Confidence: 1, TimeStep: 1)
FCGR3B_4_Activator: FCGR3B = FCGR3B&!MTHFD2 (Confidence: 1, TimeStep: 1)
FCGR3B_5_Activator: FCGR3B = !CCDC86&FCGR3B (Confidence: 1, TimeStep: 1)
FCGR3B_1_Inhibitor: FCGR3B = IQGAP3 (Confidence: 1, TimeStep: 1)
FCGR3B_2_Inhibitor: FCGR3B = TMEM97 (Confidence: 1, TimeStep: 1)
FCGR3B_3_Inhibitor: FCGR3B = WDHD1 (Confidence: 1, TimeStep: 1)
FCGR3B_4_Inhibitor: FCGR3B = CENPH (Confidence: 1, TimeStep: 1)
FCGR3B_5_Inhibitor: FCGR3B = APITD1-CORT (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FEN1 with decay value = 1:

FEN1_1_Activator: FEN1 = !BMF&MDK (Confidence: 1, TimeStep: 1)
FEN1_2_Activator: FEN1 = CENPV&MDK (Confidence: 1, TimeStep: 1)
FEN1_3_Activator: FEN1 = E2F7&MDK (Confidence: 1, TimeStep: 1)
FEN1_4_Activator: FEN1 = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)
FEN1_5_Activator: FEN1 = !IL1B&MDK (Confidence: 1, TimeStep: 1)
FEN1_1_Inhibitor: FEN1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)
FEN1_2_Inhibitor: FEN1 = !ASF1B (Confidence: 1, TimeStep: 1)
FEN1_3_Inhibitor: FEN1 = !AURKA (Confidence: 1, TimeStep: 1)

FEN1_4_Inhibitor: FEN1 = BTG1 (Confidence: 1, TimeStep: 1)

FEN1_5_Inhibitor: FEN1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FGD2 with decay value = 1:

FGD2_1_Activator: FGD2 = FGD2 (Confidence: 1, TimeStep: 1)

FGD2_2_Activator: FGD2 = LILRA1 (Confidence: 1, TimeStep: 1)

FGD2_3_Activator: FGD2 = FCGR3B&IL6ST (Confidence: 1, TimeStep: 1)

FGD2_4_Activator: FGD2 = !ANLN&F13A1 (Confidence: 1, TimeStep: 1)

FGD2_5_Activator: FGD2 = !CEP55&F13A1 (Confidence: 1, TimeStep: 1)

FGD2_1_Inhibitor: FGD2 = !SMAP2 (Confidence: 1, TimeStep: 1)

FGD2_2_Inhibitor: FGD2 = ECT2 (Confidence: 1, TimeStep: 1)

FGD2_3_Inhibitor: FGD2 = !MS4A1 (Confidence: 1, TimeStep: 1)

FGD2_4_Inhibitor: FGD2 = FOXM1 (Confidence: 1, TimeStep: 1)

FGD2_5_Inhibitor: FGD2 = STIL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FGL2 with decay value = 1:

FGL2_1_Activator: FGL2 = BIRC3&FGL2 (Confidence: 1, TimeStep: 1)

FGL2_2_Activator: FGL2 = BIRC3&PPBP (Confidence: 1, TimeStep: 1)

FGL2_3_Activator: FGL2 = F13A1&!MYRIP (Confidence: 1, TimeStep: 1)

FGL2_4_Activator: FGL2 = BIRC3&F13A1 (Confidence: 1, TimeStep: 1)

FGL2_5_Activator: FGL2 = CDC42EP3&!ID3 (Confidence: 1, TimeStep: 1)

FGL2_1_Inhibitor: FGL2 = !PIK3IP1 (Confidence: 1, TimeStep: 1)

FGL2_2_Inhibitor: FGL2 = !LYZ (Confidence: 1, TimeStep: 1)

FGL2_3_Inhibitor: FGL2 = KIF11 (Confidence: 1, TimeStep: 1)

FGL2_4_Inhibitor: FGL2 = MTHFD2 (Confidence: 1, TimeStep: 1)

FGL2_5_Inhibitor: FGL2 = NUF2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FGR with decay value = 1:

FGR_1_Activator: FGR = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

FGR_2_Activator: FGR = LOC100130872 (Confidence: 1, TimeStep: 1)

FGR_3_Activator: FGR = BCL10 (Confidence: 1, TimeStep: 1)

FGR_4_Activator: FGR = !ANP32E&TNFSF8 (Confidence: 1, TimeStep: 1)

FGR_5_Activator: FGR = !ASPM&TNFSF8 (Confidence: 1, TimeStep: 1)

FGR_1_Inhibitor: FGR = FH (Confidence: 1, TimeStep: 1)

FGR_2_Inhibitor: FGR = IQGAP3 (Confidence: 1, TimeStep: 1)

FGR_3_Inhibitor: FGR = TMEM97 (Confidence: 1, TimeStep: 1)

FGR_4_Inhibitor: FGR = WDHD1 (Confidence: 1, TimeStep: 1)

FGR_5_Inhibitor: FGR = CENPH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FH with decay value = 1:

FH_1_Activator: FH = APITD1-CORT (Confidence: 1, TimeStep: 1)

FH_2_Activator: FH = ASF1B (Confidence: 1, TimeStep: 1)

FH_3_Activator: FH = AURKA (Confidence: 1, TimeStep: 1)

FH_4_Activator: FH = !BTG1 (Confidence: 1, TimeStep: 1)

FH_5_Activator: FH = CCDC34 (Confidence: 1, TimeStep: 1)

FH_1_Inhibitor: FH = !DEPDC1B (Confidence: 1, TimeStep: 1)

FH_2_Inhibitor: FH = !ANP32E (Confidence: 1, TimeStep: 1)

FH_3_Inhibitor: FH = !FH (Confidence: 1, TimeStep: 1)

FH_4_Inhibitor: FH = !E2F7 (Confidence: 1, TimeStep: 1)

FH_5_Inhibitor: FH = NEAT1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FKBP5 with decay value = 1:

Multiple Transition Functions for FOXM1 with decay value = 1:

FOXM1_1_Activator: FOXM1 = ATAD2 (Confidence: 1, TimeStep: 1)

FOXM1_2_Activator: FOXM1 = BIRC5 (Confidence: 1, TimeStep: 1)

FOXM1_3_Activator: FOXM1 = BUB1 (Confidence: 1, TimeStep: 1)

FOXM1_4_Activator: FOXM1 = CCNA2 (Confidence: 1, TimeStep: 1)

FOXM1_5_Activator: FOXM1 = CDCA5 (Confidence: 1, TimeStep: 1)

FOXM1_1_Inhibitor: FOXM1 = !ATAD2 (Confidence: 1, TimeStep: 1)

FOXM1_2_Inhibitor: FOXM1 = !BIRC5 (Confidence: 1, TimeStep: 1)

FOXM1_3_Inhibitor: FOXM1 = !BUB1 (Confidence: 1, TimeStep: 1)

FOXM1_4_Inhibitor: FOXM1 = !CCNA2 (Confidence: 1, TimeStep: 1)

FOXM1_5_Inhibitor: FOXM1 = !CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for FUS with decay value = 1:

FUS_1_Activator: FUS = C5orf24&MPV17L (Confidence: 1, TimeStep: 1)

FUS_2_Activator: FUS = DTL&HBG1 (Confidence: 1, TimeStep: 1)

FUS_3_Activator: FUS = BRCA1&HBG1 (Confidence: 1, TimeStep: 1)

FUS_4_Activator: FUS = BYSL&IGLL1 (Confidence: 1, TimeStep: 1)

FUS_5_Activator: FUS = !CEL2F&GVINP1 (Confidence: 1, TimeStep: 1)

FUS_1_Inhibitor: FUS = CCNB1 (Confidence: 1, TimeStep: 1)

FUS_2_Inhibitor: FUS = IQGAP3 (Confidence: 1, TimeStep: 1)

FUS_3_Inhibitor: FUS = PTTG1 (Confidence: 1, TimeStep: 1)

FUS_4_Inhibitor: FUS = CDK1 (Confidence: 1, TimeStep: 1)

FUS_5_Inhibitor: FUS = CKAP2L (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GBP4 with decay value = 1:

GBP4_1_Activator: GBP4 = !CDT1&DENND3 (Confidence: 1, TimeStep: 1)

GBP4_2_Activator: GBP4 = DENND3&!KNL1 (Confidence: 1, TimeStep: 1)

GBP4_3_Activator: GBP4 = !CDT1&IL1B (Confidence: 1, TimeStep: 1)

GBP4_4_Activator: GBP4 = DENND3&!MCM5 (Confidence: 1, TimeStep: 1)

GBP4_5_Activator: GBP4 = DENND3&!RAD51 (Confidence: 1, TimeStep: 1)

GBP4_1_Inhibitor: GBP4 = !GBP4 (Confidence: 1, TimeStep: 1)

GBP4_2_Inhibitor: GBP4 = KNL1 (Confidence: 1, TimeStep: 1)

GBP4_3_Inhibitor: GBP4 = CDT1 (Confidence: 1, TimeStep: 1)

GBP4_4_Inhibitor: GBP4 = BRIP1 (Confidence: 1, TimeStep: 1)

GBP4_5_Inhibitor: GBP4 = C4orf46 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GGH with decay value = 1:

GGH_1_Activator: GGH = CENPH (Confidence: 1, TimeStep: 1)

GGH_2_Activator: GGH = APITD1-CORT (Confidence: 1, TimeStep: 1)

GGH_3_Activator: GGH = ASF1B (Confidence: 1, TimeStep: 1)

GGH_4_Activator: GGH = AURKA (Confidence: 1, TimeStep: 1)

GGH_5_Activator: GGH = !BTG1 (Confidence: 1, TimeStep: 1)

GGH_1_Inhibitor: GGH = !CENPH (Confidence: 1, TimeStep: 1)

GGH_2_Inhibitor: GGH = !CDK1 (Confidence: 1, TimeStep: 1)

GGH_3_Inhibitor: GGH = !HMMR (Confidence: 1, TimeStep: 1)

GGH_4_Inhibitor: GGH = !KIF14 (Confidence: 1, TimeStep: 1)

GGH_5_Inhibitor: GGH = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GIMAP4 with decay value = 1:

GIMAP4_1_Activator: GIMAP4 = CCR1 (Confidence: 1, TimeStep: 1)

GIMAP4_2_Activator: GIMAP4 = GVINP1 (Confidence: 1, TimeStep: 1)

GIMAP4_3_Activator: GIMAP4 = F13A1 (Confidence: 1, TimeStep: 1)

GIMAP4_4_Activator: GIMAP4 = FGL2 (Confidence: 1, TimeStep: 1)

GIMAP4_5_Activator: GIMAP4 = !ANP32E&PIK3IP1 (Confidence: 1, TimeStep: 1)

GIMAP4_1_Inhibitor: GIMAP4 = E2F7 (Confidence: 1, TimeStep: 1)

GIMAP4_2_Inhibitor: GIMAP4 = DEPDC1B (Confidence: 1, TimeStep: 1)

GIMAP4_3_Inhibitor: GIMAP4 = FOXM1 (Confidence: 1, TimeStep: 1)

GIMAP4_4_Inhibitor: GIMAP4 = KIF18B (Confidence: 1, TimeStep: 1)

GIMAP4_5_Inhibitor: GIMAP4 = STIL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GIMAP7 with decay value = 1:

GIMAP7_1_Activator: GIMAP7 = GVINP1 (Confidence: 1, TimeStep: 1)

GIMAP7_2_Activator: GIMAP7 = MPV17L (Confidence: 1, TimeStep: 1)

GIMAP7_3_Activator: GIMAP7 = F13A1 (Confidence: 1, TimeStep: 1)

GIMAP7_4_Activator: GIMAP7 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

GIMAP7_5_Activator: GIMAP7 = FGL2 (Confidence: 1, TimeStep: 1)

GIMAP7_1_Inhibitor: GIMAP7 = DEPDC1B (Confidence: 1, TimeStep: 1)

GIMAP7_2_Inhibitor: GIMAP7 = PTTG1 (Confidence: 1, TimeStep: 1)

GIMAP7_3_Inhibitor: GIMAP7 = AURKB (Confidence: 1, TimeStep: 1)

GIMAP7_4_Inhibitor: GIMAP7 = KIF18A (Confidence: 1, TimeStep: 1)

GIMAP7_5_Inhibitor: GIMAP7 = OIP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GINS1 with decay value = 1:

GINS1_1_Activator: GINS1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

GINS1_2_Activator: GINS1 = ASF1B (Confidence: 1, TimeStep: 1)

GINS1_3_Activator: GINS1 = AURKA (Confidence: 1, TimeStep: 1)

GINS1_4_Activator: GINS1 = !BTG1 (Confidence: 1, TimeStep: 1)

GINS1_5_Activator: GINS1 = CCDC34 (Confidence: 1, TimeStep: 1)

GINS1_1_Inhibitor: GINS1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

GINS1_2_Inhibitor: GINS1 = !ASF1B (Confidence: 1, TimeStep: 1)

GINS1_3_Inhibitor: GINS1 = !AURKA (Confidence: 1, TimeStep: 1)

GINS1_4_Inhibitor: GINS1 = BTG1 (Confidence: 1, TimeStep: 1)

GINS1_5_Inhibitor: GINS1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GINS2 with decay value = 1:

GINS2_1_Activator: GINS2 = ANLN (Confidence: 1, TimeStep: 1)

GINS2_2_Activator: GINS2 = BRIP1 (Confidence: 1, TimeStep: 1)

GINS2_3_Activator: GINS2 = CHEK1 (Confidence: 1, TimeStep: 1)

GINS2_4_Activator: GINS2 = FANCI (Confidence: 1, TimeStep: 1)

GINS2_5_Activator: GINS2 = TTK (Confidence: 1, TimeStep: 1)

GINS2_1_Inhibitor: GINS2 = !RAD51 (Confidence: 1, TimeStep: 1)

GINS2_2_Inhibitor: GINS2 = !PCNA (Confidence: 1, TimeStep: 1)

GINS2_3_Inhibitor: GINS2 = !ZNF367 (Confidence: 1, TimeStep: 1)

GINS2_4_Inhibitor: GINS2 = !BRCA1 (Confidence: 1, TimeStep: 1)

GINS2_5_Inhibitor: GINS2 = !GINS2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GSN with decay value = 1:

GSN_1_Activator: GSN = SERPINB9 (Confidence: 1, TimeStep: 1)

GSN_2_Activator: GSN = ITGAM (Confidence: 1, TimeStep: 1)

GSN_3_Activator: GSN = TBXA2R (Confidence: 1, TimeStep: 1)

GSN_4_Activator: GSN = CCR1 (Confidence: 1, TimeStep: 1)

GSN_5_Activator: GSN = RBMS3 (Confidence: 1, TimeStep: 1)

GSN_1_Inhibitor: GSN = !METTL7A (Confidence: 1, TimeStep: 1)

GSN_2_Inhibitor: GSN = UBE2C (Confidence: 1, TimeStep: 1)

GSN_3_Inhibitor: GSN = !AKAP12&!LILRB2 (Confidence: 1, TimeStep: 1)

GSN_4_Inhibitor: GSN = !AKAP12&!IL6ST (Confidence: 1, TimeStep: 1)

GSN_5_Inhibitor: GSN = !AKAP12&!KLF9 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for GVINP1 with decay value = 1:

GVINP1_1_Activator: GVINP1 = PPBP (Confidence: 1, TimeStep: 1)

GVINP1_2_Activator: GVINP1 = FGL2 (Confidence: 1, TimeStep: 1)

GVINP1_3_Activator: GVINP1 = LILRA1 (Confidence: 1, TimeStep: 1)

GVINP1_4_Activator: GVINP1 = !BRCA1&SERPINA1 (Confidence: 1, TimeStep: 1)

GVINP1_5_Activator: GVINP1 = !BYSL&MS4A7 (Confidence: 1, TimeStep: 1)

GVINP1_1_Inhibitor: GVINP1 = MCM7 (Confidence: 1, TimeStep: 1)

GVINP1_2_Inhibitor: GVINP1 = KIF11 (Confidence: 1, TimeStep: 1)

GVINP1_3_Inhibitor: GVINP1 = KNL1 (Confidence: 1, TimeStep: 1)

GVINP1_4_Inhibitor: GVINP1 = DHFR (Confidence: 1, TimeStep: 1)

GVINP1_5_Inhibitor: GVINP1 = MTHFD2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HBB with decay value = 1:

HBB_1_Activator: HBB = LGALS3 (Confidence: 1, TimeStep: 1)

HBB_2_Activator: HBB = LOC100130872 (Confidence: 1, TimeStep: 1)

HBB_3_Activator: HBB = !CENPF&DEFA1 (Confidence: 1, TimeStep: 1)

HBB_4_Activator: HBB = !CKS1B&IPSPH (Confidence: 1, TimeStep: 1)

HBB_5_Activator: HBB = HBB&IGH (Confidence: 1, TimeStep: 1)

HBB_1_Inhibitor: HBB = SHCBP1 (Confidence: 1, TimeStep: 1)

HBB_2_Inhibitor: HBB = CCNB1 (Confidence: 1, TimeStep: 1)

HBB_3_Inhibitor: HBB = CENPF (Confidence: 1, TimeStep: 1)

HBB_4_Inhibitor: HBB = NEK2 (Confidence: 1, TimeStep: 1)

HBB_5_Inhibitor: HBB = CDK1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HBG1 with decay value = 1:

HBG1_1_Activator: HBG1 = LGALS3 (Confidence: 1, TimeStep: 1)

HBG1_2_Activator: HBG1 = LOC100130872 (Confidence: 1, TimeStep: 1)

HBG1_3_Activator: HBG1 = LILRA1 (Confidence: 1, TimeStep: 1)

HBG1_4_Activator: HBG1 = !CENPF&DEFA1 (Confidence: 1, TimeStep: 1)

HBG1_5_Activator: HBG1 = HBB&IGH (Confidence: 1, TimeStep: 1)

HBG1_1_Inhibitor: HBG1 = !S100A8 (Confidence: 1, TimeStep: 1)

HBG1_2_Inhibitor: HBG1 = RFC3 (Confidence: 1, TimeStep: 1)

HBG1_3_Inhibitor: HBG1 = SHCBP1 (Confidence: 1, TimeStep: 1)

HBG1_4_Inhibitor: HBG1 = FH (Confidence: 1, TimeStep: 1)

HBG1_5_Inhibitor: HBG1 = CCNB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HELLS with decay value = 1:

HELLS_1_Activator: HELLS = KIF2C (Confidence: 1, TimeStep: 1)

HELLS_2_Activator: HELLS = ATAD2 (Confidence: 1, TimeStep: 1)

HELLS_3_Activator: HELLS = BIRC5 (Confidence: 1, TimeStep: 1)

HELLS_4_Activator: HELLS = BUB1 (Confidence: 1, TimeStep: 1)

HELLS_5_Activator: HELLS = CCNA2 (Confidence: 1, TimeStep: 1)

HELLS_1_Inhibitor: HELLS = !KIF2C (Confidence: 1, TimeStep: 1)

HELLS_2_Inhibitor: HELLS = !CCNB2 (Confidence: 1, TimeStep: 1)

HELLS_3_Inhibitor: HELLS = !CDC45 (Confidence: 1, TimeStep: 1)

HELLS_4_Inhibitor: HELLS = !CENPA (Confidence: 1, TimeStep: 1)

HELLS_5_Inhibitor: HELLS = !DLGAP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HIST4H4 with decay value = 1:

HIST4H4_1_Activator: HIST4H4 = !TXNIP (Confidence: 1, TimeStep: 1)

HIST4H4_2_Activator: HIST4H4 = AURKB (Confidence: 1, TimeStep: 1)

HIST4H4_3_Activator: HIST4H4 = CKAP2 (Confidence: 1, TimeStep: 1)

HIST4H4_4_Activator: HIST4H4 = KIF18A (Confidence: 1, TimeStep: 1)

HIST4H4_5_Activator: HIST4H4 = OIP5 (Confidence: 1, TimeStep: 1)

HIST4H4_1_Inhibitor: HIST4H4 = EMP1 (Confidence: 1, TimeStep: 1)

HIST4H4_2_Inhibitor: HIST4H4 = !TNFRSF21 (Confidence: 1, TimeStep: 1)

HIST4H4_3_Inhibitor: HIST4H4 = !C5orf24 (Confidence: 1, TimeStep: 1)

HIST4H4_4_Inhibitor: HIST4H4 = !BCL2L11&CDK6 (Confidence: 1, TimeStep: 1)

HIST4H4_5_Inhibitor: HIST4H4 = B3GNT2&!CENPU (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HJURP with decay value = 1:

HJURP_1_Activator: HJURP = APITD1-CORT (Confidence: 1, TimeStep: 1)

HJURP_2_Activator: HJURP = ASF1B (Confidence: 1, TimeStep: 1)

HJURP_3_Activator: HJURP = AURKA (Confidence: 1, TimeStep: 1)

HJURP_4_Activator: HJURP = !BTG1 (Confidence: 1, TimeStep: 1)

HJURP_5_Activator: HJURP = CCDC34 (Confidence: 1, TimeStep: 1)

HJURP_1_Inhibitor: HJURP = !APITD1-CORT (Confidence: 1, TimeStep: 1)

HJURP_2_Inhibitor: HJURP = !ASF1B (Confidence: 1, TimeStep: 1)

HJURP_3_Inhibitor: HJURP = !AURKA (Confidence: 1, TimeStep: 1)

HJURP_4_Inhibitor: HJURP = BTG1 (Confidence: 1, TimeStep: 1)

HJURP_5_Inhibitor: HJURP = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HMMR with decay value = 1:

HMMR_1_Activator: HMMR = APITD1-CORT (Confidence: 1, TimeStep: 1)

HMMR_2_Activator: HMMR = ASF1B (Confidence: 1, TimeStep: 1)

HMMR_3_Activator: HMMR = AURKA (Confidence: 1, TimeStep: 1)

HMMR_4_Activator: HMMR = !BTG1 (Confidence: 1, TimeStep: 1)

HMMR_5_Activator: HMMR = CCDC34 (Confidence: 1, TimeStep: 1)

HMMR_1_Inhibitor: HMMR = !APITD1-CORT (Confidence: 1, TimeStep: 1)

HMMR_2_Inhibitor: HMMR = !ASF1B (Confidence: 1, TimeStep: 1)

HMMR_3_Inhibitor: HMMR = !AURKA (Confidence: 1, TimeStep: 1)

HMMR_4_Inhibitor: HMMR = BTG1 (Confidence: 1, TimeStep: 1)

HMMR_5_Inhibitor: HMMR = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HRK with decay value = 1:

HRK_1_Activator: HRK = DPEP1&HRK (Confidence: 1, TimeStep: 1)

HRK_2_Activator: HRK = DENND3&HRK (Confidence: 1, TimeStep: 1)

HRK_3_Activator: HRK = !ARRDC3&HRK (Confidence: 1, TimeStep: 1)

HRK_4_Activator: HRK = DPEP1&!RHOBTB3 (Confidence: 1, TimeStep: 1)

HRK_5_Activator: HRK = IL1B&!RHOBTB3 (Confidence: 1, TimeStep: 1)

HRK_1_Inhibitor: HRK = !DPEP1 (Confidence: 1, TimeStep: 1)

HRK_2_Inhibitor: HRK = !MSH6 (Confidence: 1, TimeStep: 1)

HRK_3_Inhibitor: HRK = !ISG20 (Confidence: 1, TimeStep: 1)

HRK_4_Inhibitor: HRK = !SMAP2 (Confidence: 1, TimeStep: 1)

HRK_5_Inhibitor: HRK = ECT2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for HSP90AB1 with decay value = 1:

HSP90AB1_1_Activator: HSP90AB1 = KIF11 (Confidence: 1, TimeStep: 1)

HSP90AB1_2_Activator: HSP90AB1 = KNL1 (Confidence: 1, TimeStep: 1)

HSP90AB1_3_Activator: HSP90AB1 = DHFR (Confidence: 1, TimeStep: 1)

HSP90AB1_4_Activator: HSP90AB1 = RAD51 (Confidence: 1, TimeStep: 1)

HSP90AB1_5_Activator: HSP90AB1 = !CD53 (Confidence: 1, TimeStep: 1)

HSP90AB1_1_Inhibitor: HSP90AB1 = ASPM&!CRNDE (Confidence: 1, TimeStep: 1)

HSP90AB1_2_Inhibitor: HSP90AB1 = ASPM&LGALS3 (Confidence: 1, TimeStep: 1)

HSP90AB1_3_Inhibitor: HSP90AB1 = ASPM&!TYMS (Confidence: 1, TimeStep: 1)

HSP90AB1_4_Inhibitor: HSP90AB1 = !ANLN&FABP5&!HSP90AB1 (Confidence: 1, TimeStep: 1)

HSP90AB1_5_Inhibitor: HSP90AB1 = !ANLN&HSP90AB1&LGALS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ID2 with decay value = 1:

ID2_1_Activator: ID2 = PRDM1 (Confidence: 1, TimeStep: 1)

ID2_2_Activator: ID2 = CENPV&ID2 (Confidence: 1, TimeStep: 1)

ID2_1_Inhibitor: ID2 = !CCNB2&ECT2 (Confidence: 1, TimeStep: 1)

ID2_2_Inhibitor: ID2 = !CCNB2&!METTL7A (Confidence: 1, TimeStep: 1)

ID2_3_Inhibitor: ID2 = !CDC45&ECT2 (Confidence: 1, TimeStep: 1)

ID2_4_Inhibitor: ID2 = !CDC45&!METTL7A (Confidence: 1, TimeStep: 1)

ID2_5_Inhibitor: ID2 = !CENPA&ECT2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ID3 with decay value = 1:

ID3_1_Activator: ID3 = HRK (Confidence: 1, TimeStep: 1)

ID3_2_Activator: ID3 = EMP1 (Confidence: 1, TimeStep: 1)

ID3_3_Activator: ID3 = MDK (Confidence: 1, TimeStep: 1)

ID3_4_Activator: ID3 = MDM2 (Confidence: 1, TimeStep: 1)

ID3_5_Activator: ID3 = ABHD17B&PTP4A1 (Confidence: 1, TimeStep: 1)

ID3_1_Inhibitor: ID3 = SEMA4D (Confidence: 1, TimeStep: 1)

ID3_2_Inhibitor: ID3 = !B3GNT2&FGR (Confidence: 1, TimeStep: 1)

ID3_3_Inhibitor: ID3 = !DTL&!IL1B (Confidence: 1, TimeStep: 1)

ID3_4_Inhibitor: ID3 = !AKAP12&!TYMS (Confidence: 1, TimeStep: 1)

ID3_5_Inhibitor: ID3 = !DHX9&!IL1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IFNGR1 with decay value = 1:

IFNGR1_1_Activator: IFNGR1 = LILRB2 (Confidence: 1, TimeStep: 1)

IFNGR1_2_Activator: IFNGR1 = !CENPV (Confidence: 1, TimeStep: 1)

IFNGR1_3_Activator: IFNGR1 = IRAK3 (Confidence: 1, TimeStep: 1)

IFNGR1_4_Activator: IFNGR1 = DENND3 (Confidence: 1, TimeStep: 1)

IFNGR1_5_Activator: IFNGR1 = IFNGR1 (Confidence: 1, TimeStep: 1)

IFNGR1_1_Inhibitor: IFNGR1 = ECT2 (Confidence: 1, TimeStep: 1)

IFNGR1_2_Inhibitor: IFNGR1 = FOXM1 (Confidence: 1, TimeStep: 1)

IFNGR1_3_Inhibitor: IFNGR1 = STIL (Confidence: 1, TimeStep: 1)

IFNGR1_4_Inhibitor: IFNGR1 = ATAD2 (Confidence: 1, TimeStep: 1)

IFNGR1_5_Inhibitor: IFNGR1 = BIRC5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IGH with decay value = 1:

IGH_1_Activator: IGH = LGALS3 (Confidence: 1, TimeStep: 1)

IGH_2_Activator: IGH = !CCDC86&IGH (Confidence: 1, TimeStep: 1)

IGH_3_Activator: IGH = IGH&PDE4B (Confidence: 1, TimeStep: 1)

IGH_4_Activator: IGH = !CCDC86&!PRPS2 (Confidence: 1, TimeStep: 1)

IGH_5_Activator: IGH = GBP4&IGLC1 (Confidence: 1, TimeStep: 1)

IGH_1_Inhibitor: IGH = MTHFD2 (Confidence: 1, TimeStep: 1)

IGH_2_Inhibitor: IGH = !NEAT1 (Confidence: 1, TimeStep: 1)

IGH_3_Inhibitor: IGH = !MS4A1 (Confidence: 1, TimeStep: 1)

IGH_4_Inhibitor: IGH = !TXNIP (Confidence: 1, TimeStep: 1)

IGH_5_Inhibitor: IGH = CKAP2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IGLC1 with decay value = 1:

IGLC1_1_Activator: IGLC1 = LGALS3 (Confidence: 1, TimeStep: 1)

IGLC1_2_Activator: IGLC1 = !CCDC86&IGH (Confidence: 1, TimeStep: 1)

IGLC1_3_Activator: IGLC1 = !CCDC86&IGLC1 (Confidence: 1, TimeStep: 1)

IGLC1_4_Activator: IGLC1 = IGH&PDE4B (Confidence: 1, TimeStep: 1)

IGLC1_5_Activator: IGLC1 = !CCDC86&!PRPS2 (Confidence: 1, TimeStep: 1)

IGLC1_1_Inhibitor: IGLC1 = !MS4A1 (Confidence: 1, TimeStep: 1)

IGLC1_2_Inhibitor: IGLC1 = !TXNIP (Confidence: 1, TimeStep: 1)

IGLC1_3_Inhibitor: IGLC1 = CKAP2 (Confidence: 1, TimeStep: 1)

IGLC1_4_Inhibitor: IGLC1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

IGLC1_5_Inhibitor: IGLC1 = ASF1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IGLL1 with decay value = 1:

IGLL1_1_Activator: IGLL1 = IGLL1 (Confidence: 1, TimeStep: 1)

IGLL1_2_Activator: IGLL1 = AKAP12 (Confidence: 1, TimeStep: 1)

IGLL1_3_Activator: IGLL1 = !IL27RA (Confidence: 1, TimeStep: 1)

IGLL1_4_Activator: IGLL1 = BCAT1 (Confidence: 1, TimeStep: 1)

IGLL1_5_Activator: IGLL1 = !SLA (Confidence: 1, TimeStep: 1)

IGLL1_1_Inhibitor: IGLL1 = !IGLL1 (Confidence: 1, TimeStep: 1)

IGLL1_2_Inhibitor: IGLL1 = LOC100130872 (Confidence: 1, TimeStep: 1)

IGLL1_3_Inhibitor: IGLL1 = !BCAT1&!BMF (Confidence: 1, TimeStep: 1)

IGLL1_4_Inhibitor: IGLL1 = !AKAP12&!C5orf24 (Confidence: 1, TimeStep: 1)

IGLL1_5_Inhibitor: IGLL1 = !AKAP12&PPP1R16B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IL18R1 with decay value = 1:

IL18R1_1_Activator: IL18R1 = IL18R1 (Confidence: 1, TimeStep: 1)

IL18R1_2_Activator: IL18R1 = IL18RAP (Confidence: 1, TimeStep: 1)

IL18R1_3_Activator: IL18R1 = RAB31 (Confidence: 1, TimeStep: 1)

IL18R1_4_Activator: IL18R1 = ID2&PPP1R16B (Confidence: 1, TimeStep: 1)

IL18R1_5_Activator: IL18R1 = ID2&!PRPS2 (Confidence: 1, TimeStep: 1)

IL18R1_1_Inhibitor: IL18R1 = CENPV (Confidence: 1, TimeStep: 1)

IL18R1_2_Inhibitor: IL18R1 = !IRAK3 (Confidence: 1, TimeStep: 1)

IL18R1_3_Inhibitor: IL18R1 = !TMEM2 (Confidence: 1, TimeStep: 1)

IL18R1_4_Inhibitor: IL18R1 = ANP32E (Confidence: 1, TimeStep: 1)

IL18R1_5_Inhibitor: IL18R1 = CEP55 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IL18RAP with decay value = 1:

IL18RAP_1_Activator: IL18RAP = IL18RAP (Confidence: 1, TimeStep: 1)

IL18RAP_2_Activator: IL18RAP = ID2&PPP1R16B (Confidence: 1, TimeStep: 1)

IL18RAP_3_Activator: IL18RAP = ID2&!PRPS2 (Confidence: 1, TimeStep: 1)

IL18RAP_4_Activator: IL18RAP = !DHX9&IL18R1 (Confidence: 1, TimeStep: 1)

IL18RAP_5_Activator: IL18RAP = !DHX9&!LEF1 (Confidence: 1, TimeStep: 1)

IL18RAP_1_Inhibitor: IL18RAP = CENPV (Confidence: 1, TimeStep: 1)

IL18RAP_2_Inhibitor: IL18RAP = !IRAK3 (Confidence: 1, TimeStep: 1)

IL18RAP_3_Inhibitor: IL18RAP = !TMEM2 (Confidence: 1, TimeStep: 1)

IL18RAP_4_Inhibitor: IL18RAP = ANP32E (Confidence: 1, TimeStep: 1)

IL18RAP_5_Inhibitor: IL18RAP = CEP55 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IL1B with decay value = 1:

IL1B_1_Activator: IL1B = GBP4&!HBG1 (Confidence: 1, TimeStep: 1)

IL1B_2_Activator: IL1B = DENND3&!SLA (Confidence: 1, TimeStep: 1)

IL1B_3_Activator: IL1B = EMP1&GBP4 (Confidence: 1, TimeStep: 1)

IL1B_4_Activator: IL1B = EMP1&!SQLE (Confidence: 1, TimeStep: 1)

IL1B_5_Activator: IL1B = !HBG1&!KNL1 (Confidence: 1, TimeStep: 1)

IL1B_1_Inhibitor: IL1B = !IL1B (Confidence: 1, TimeStep: 1)

IL1B_2_Inhibitor: IL1B = !DENND3 (Confidence: 1, TimeStep: 1)

IL1B_3_Inhibitor: IL1B = !GBP4 (Confidence: 1, TimeStep: 1)

IL1B_4_Inhibitor: IL1B = TNFSF8 (Confidence: 1, TimeStep: 1)

IL1B_5_Inhibitor: IL1B = ECT2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IL27RA with decay value = 1:

IL27RA_1_Activator: IL27RA = IL27RA (Confidence: 1, TimeStep: 1)

IL27RA_2_Activator: IL27RA = !IGLL1 (Confidence: 1, TimeStep: 1)

IL27RA_3_Activator: IL27RA = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

IL27RA_4_Activator: IL27RA = GIMAP7&SLA (Confidence: 1, TimeStep: 1)

IL27RA_5_Activator: IL27RA = NEAT1&!SIK1 (Confidence: 1, TimeStep: 1)

IL27RA_1_Inhibitor: IL27RA = !ZFP36L2 (Confidence: 1, TimeStep: 1)

IL27RA_2_Inhibitor: IL27RA = !IL27RA&RHOBTB3 (Confidence: 1, TimeStep: 1)

IL27RA_3_Inhibitor: IL27RA = CCDC86&!IL27RA (Confidence: 1, TimeStep: 1)

IL27RA_4_Inhibitor: IL27RA = !CDK6&!IL27RA (Confidence: 1, TimeStep: 1)

IL27RA_5_Inhibitor: IL27RA = !GIMAP4&!IL27RA (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IL6ST with decay value = 1:

IL6ST_1_Activator: IL6ST = MTSS1 (Confidence: 1, TimeStep: 1)

IL6ST_2_Activator: IL6ST = ITGAM (Confidence: 1, TimeStep: 1)

IL6ST_3_Activator: IL6ST = TBXA2R (Confidence: 1, TimeStep: 1)

IL6ST_4_Activator: IL6ST = FGD2 (Confidence: 1, TimeStep: 1)

IL6ST_5_Activator: IL6ST = CCR1 (Confidence: 1, TimeStep: 1)

IL6ST_1_Inhibitor: IL6ST = BCL10 (Confidence: 1, TimeStep: 1)

IL6ST_2_Inhibitor: IL6ST = !ATAD2&!MS4A1 (Confidence: 1, TimeStep: 1)

IL6ST_3_Inhibitor: IL6ST = !ATAD2&!SMAP2 (Confidence: 1, TimeStep: 1)

IL6ST_4_Inhibitor: IL6ST = !ATAD2&ECT2 (Confidence: 1, TimeStep: 1)

IL6ST_5_Inhibitor: IL6ST = !ATAD2&!METTL7A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IQGAP3 with decay value = 1:

IQGAP3_1_Activator: IQGAP3 = CENPH (Confidence: 1, TimeStep: 1)

IQGAP3_2_Activator: IQGAP3 = APITD1-CORT (Confidence: 1, TimeStep: 1)

IQGAP3_3_Activator: IQGAP3 = ASF1B (Confidence: 1, TimeStep: 1)

IQGAP3_4_Activator: IQGAP3 = AURKA (Confidence: 1, TimeStep: 1)

IQGAP3_5_Activator: IQGAP3 = !BTG1 (Confidence: 1, TimeStep: 1)

IQGAP3_1_Inhibitor: IQGAP3 = !CENPH (Confidence: 1, TimeStep: 1)

IQGAP3_2_Inhibitor: IQGAP3 = !CDK1 (Confidence: 1, TimeStep: 1)

IQGAP3_3_Inhibitor: IQGAP3 = !HMMR (Confidence: 1, TimeStep: 1)

IQGAP3_4_Inhibitor: IQGAP3 = !KIF14 (Confidence: 1, TimeStep: 1)

IQGAP3_5_Inhibitor: IQGAP3 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for IRAK3 with decay value = 1:

IRAK3_1_Activator: IRAK3 = IRAK3 (Confidence: 1, TimeStep: 1)

IRAK3_2_Activator: IRAK3 = MTSS1 (Confidence: 1, TimeStep: 1)

IRAK3_3_Activator: IRAK3 = PDE4B (Confidence: 1, TimeStep: 1)

IRAK3_4_Activator: IRAK3 = SERPINB9 (Confidence: 1, TimeStep: 1)

IRAK3_5_Activator: IRAK3 = !CRNDE (Confidence: 1, TimeStep: 1)

IRAK3_1_Inhibitor: IRAK3 = !TMEM2 (Confidence: 1, TimeStep: 1)

IRAK3_2_Inhibitor: IRAK3 = ECT2 (Confidence: 1, TimeStep: 1)

IRAK3_3_Inhibitor: IRAK3 = FOXM1 (Confidence: 1, TimeStep: 1)

IRAK3_4_Inhibitor: IRAK3 = STIL (Confidence: 1, TimeStep: 1)

IRAK3_5_Inhibitor: IRAK3 = ATAD2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ISG20 with decay value = 1:

ISG20_1_Activator: ISG20 = !ECT2 (Confidence: 1, TimeStep: 1)

ISG20_2_Activator: ISG20 = SMAP2 (Confidence: 1, TimeStep: 1)

ISG20_3_Activator: ISG20 = !CENPK (Confidence: 1, TimeStep: 1)

ISG20_4_Activator: ISG20 = ISG20 (Confidence: 1, TimeStep: 1)

ISG20_5_Activator: ISG20 = GBP4 (Confidence: 1, TimeStep: 1)

ISG20_1_Inhibitor: ISG20 = !APITD1-CORT&ATAD2 (Confidence: 1, TimeStep: 1)

ISG20_2_Inhibitor: ISG20 = !APITD1-CORT&BIRC5 (Confidence: 1, TimeStep: 1)

ISG20_3_Inhibitor: ISG20 = !APITD1-CORT&BUB1 (Confidence: 1, TimeStep: 1)

ISG20_4_Inhibitor: ISG20 = !APITD1-CORT&CCNA2 (Confidence: 1, TimeStep: 1)

ISG20_5_Inhibitor: ISG20 = !APITD1-CORT&CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ITGAM with decay value = 1:

ITGAM_1_Activator: ITGAM = ITGAM (Confidence: 1, TimeStep: 1)

ITGAM_2_Activator: ITGAM = F13A1 (Confidence: 1, TimeStep: 1)

ITGAM_3_Activator: ITGAM = RAB31 (Confidence: 1, TimeStep: 1)

ITGAM_4_Activator: ITGAM = BMF&LOC285097 (Confidence: 1, TimeStep: 1)

ITGAM_5_Activator: ITGAM = !CCNL1&!PAICS (Confidence: 1, TimeStep: 1)

ITGAM_1_Inhibitor: ITGAM = !SERPINB9 (Confidence: 1, TimeStep: 1)

ITGAM_2_Inhibitor: ITGAM = CENPV (Confidence: 1, TimeStep: 1)

ITGAM_3_Inhibitor: ITGAM = !IRAK3 (Confidence: 1, TimeStep: 1)

ITGAM_4_Inhibitor: ITGAM = !LILRB2 (Confidence: 1, TimeStep: 1)

ITGAM_5_Inhibitor: ITGAM = !RNASET2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ITGB2-AS1 with decay value = 1:

ITGB2-AS1_1_Activator: ITGB2-AS1 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_2_Activator: ITGB2-AS1 = LOC100130872 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_3_Activator: ITGB2-AS1 = LILRA1 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_4_Activator: ITGB2-AS1 = !AKAP12&HBB (Confidence: 1, TimeStep: 1)

ITGB2-AS1_5_Activator: ITGB2-AS1 = !AKAP12&FGR (Confidence: 1, TimeStep: 1)

ITGB2-AS1_1_Inhibitor: ITGB2-AS1 = !HBB (Confidence: 1, TimeStep: 1)

ITGB2-AS1_2_Inhibitor: ITGB2-AS1 = MSH6 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_3_Inhibitor: ITGB2-AS1 = AKAP12 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_4_Inhibitor: ITGB2-AS1 = WFS1 (Confidence: 1, TimeStep: 1)

ITGB2-AS1_5_Inhibitor: ITGB2-AS1 = !SLA (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ITPKB with decay value = 1:

ITPKB_1_Activator: ITPKB = LILRA1 (Confidence: 1, TimeStep: 1)

ITPKB_2_Activator: ITPKB = CELF2&IGLC1 (Confidence: 1, TimeStep: 1)

ITPKB_3_Activator: ITPKB = CELF2&IGH (Confidence: 1, TimeStep: 1)

ITPKB_4_Activator: ITPKB = IGLC1&SLA (Confidence: 1, TimeStep: 1)

ITPKB_5_Activator: ITPKB = ITPKB&MS4A7 (Confidence: 1, TimeStep: 1)

ITPKB_1_Inhibitor: ITPKB = CDK1 (Confidence: 1, TimeStep: 1)

ITPKB_2_Inhibitor: ITPKB = CKAP2L (Confidence: 1, TimeStep: 1)

ITPKB_3_Inhibitor: ITPKB = HMMR (Confidence: 1, TimeStep: 1)

ITPKB_4_Inhibitor: ITPKB = KIF14 (Confidence: 1, TimeStep: 1)

ITPKB_5_Inhibitor: ITPKB = KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KCNK12 with decay value = 1:

KCNK12_1_Activator: KCNK12 = !EGR1&MYRIP (Confidence: 1, TimeStep: 1)

KCNK12_2_Activator: KCNK12 = !EGR1&MDM2 (Confidence: 1, TimeStep: 1)

KCNK12_3_Activator: KCNK12 = FKBP5&!SLA (Confidence: 1, TimeStep: 1)

KCNK12_4_Activator: KCNK12 = AKAP12&LOC728175 (Confidence: 1, TimeStep: 1)

KCNK12_5_Activator: KCNK12 = !EGR1&LOC728175 (Confidence: 1, TimeStep: 1)

KCNK12_1_Inhibitor: KCNK12 = !FKBP5&!KCNK12 (Confidence: 1, TimeStep: 1)

KCNK12_2_Inhibitor: KCNK12 = !FKBP5&!MDM2 (Confidence: 1, TimeStep: 1)

KCNK12_3_Inhibitor: KCNK12 = !KCNK12 (Confidence: 1, TimeStep: 2)

KCNK12_4_Inhibitor: KCNK12 = !STAB1 (Confidence: 1, TimeStep: 2)

KCNK12_5_Inhibitor: KCNK12 = !TUBA4A (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for KIAA0101 with decay value = 1:

KIAA0101_1_Activator: KIAA0101 = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIAA0101_2_Activator: KIAA0101 = ASF1B (Confidence: 1, TimeStep: 1)

KIAA0101_3_Activator: KIAA0101 = AURKA (Confidence: 1, TimeStep: 1)

KIAA0101_4_Activator: KIAA0101 = !BTG1 (Confidence: 1, TimeStep: 1)

KIAA0101_5_Activator: KIAA0101 = CCDC34 (Confidence: 1, TimeStep: 1)

KIAA0101_1_Inhibitor: KIAA0101 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIAA0101_2_Inhibitor: KIAA0101 = !ASF1B (Confidence: 1, TimeStep: 1)

KIAA0101_3_Inhibitor: KIAA0101 = !AURKA (Confidence: 1, TimeStep: 1)

KIAA0101_4_Inhibitor: KIAA0101 = BTG1 (Confidence: 1, TimeStep: 1)

KIAA0101_5_Inhibitor: KIAA0101 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF11 with decay value = 1:

KIF11_1_Activator: KIF11 = CENPH (Confidence: 1, TimeStep: 1)

KIF11_2_Activator: KIF11 = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF11_3_Activator: KIF11 = ASF1B (Confidence: 1, TimeStep: 1)

KIF11_4_Activator: KIF11 = AURKA (Confidence: 1, TimeStep: 1)

KIF11_5_Activator: KIF11 = !BTG1 (Confidence: 1, TimeStep: 1)

KIF11_1_Inhibitor: KIF11 = !CENPF (Confidence: 1, TimeStep: 1)

KIF11_2_Inhibitor: KIF11 = !NEK2 (Confidence: 1, TimeStep: 1)

KIF11_3_Inhibitor: KIF11 = !FH (Confidence: 1, TimeStep: 1)

KIF11_4_Inhibitor: KIF11 = !C4orf46 (Confidence: 1, TimeStep: 1)

KIF11_5_Inhibitor: KIF11 = !HELLS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF14 with decay value = 1:

KIF14_1_Activator: KIF14 = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF14_2_Activator: KIF14 = ASF1B (Confidence: 1, TimeStep: 1)

KIF14_3_Activator: KIF14 = AURKA (Confidence: 1, TimeStep: 1)

KIF14_4_Activator: KIF14 = !BTG1 (Confidence: 1, TimeStep: 1)

KIF14_5_Activator: KIF14 = CCDC34 (Confidence: 1, TimeStep: 1)

KIF14_1_Inhibitor: KIF14 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF14_2_Inhibitor: KIF14 = !ASF1B (Confidence: 1, TimeStep: 1)

KIF14_3_Inhibitor: KIF14 = !AURKA (Confidence: 1, TimeStep: 1)

KIF14_4_Inhibitor: KIF14 = BTG1 (Confidence: 1, TimeStep: 1)

KIF14_5_Inhibitor: KIF14 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF15 with decay value = 1:

KIF15_1_Activator: KIF15 = CENPH (Confidence: 1, TimeStep: 1)

KIF15_2_Activator: KIF15 = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF15_3_Activator: KIF15 = ASF1B (Confidence: 1, TimeStep: 1)

KIF15_4_Activator: KIF15 = AURKA (Confidence: 1, TimeStep: 1)

KIF15_5_Activator: KIF15 = !BTG1 (Confidence: 1, TimeStep: 1)

KIF15_1_Inhibitor: KIF15 = !CENPH (Confidence: 1, TimeStep: 1)

KIF15_2_Inhibitor: KIF15 = !CDK1 (Confidence: 1, TimeStep: 1)

KIF15_3_Inhibitor: KIF15 = !HMMR (Confidence: 1, TimeStep: 1)

KIF15_4_Inhibitor: KIF15 = !KIF14 (Confidence: 1, TimeStep: 1)

KIF15_5_Inhibitor: KIF15 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF18A with decay value = 1:

KIF18A_1_Activator: KIF18A = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF18A_2_Activator: KIF18A = ASF1B (Confidence: 1, TimeStep: 1)

KIF18A_3_Activator: KIF18A = AURKA (Confidence: 1, TimeStep: 1)

KIF18A_4_Activator: KIF18A = !BTG1 (Confidence: 1, TimeStep: 1)

KIF18A_5_Activator: KIF18A = CCDC34 (Confidence: 1, TimeStep: 1)

KIF18A_1_Inhibitor: KIF18A = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF18A_2_Inhibitor: KIF18A = !ASF1B (Confidence: 1, TimeStep: 1)

KIF18A_3_Inhibitor: KIF18A = !AURKA (Confidence: 1, TimeStep: 1)

KIF18A_4_Inhibitor: KIF18A = BTG1 (Confidence: 1, TimeStep: 1)

KIF18A_5_Inhibitor: KIF18A = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF18B with decay value = 1:

KIF18B_1_Activator: KIF18B = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF18B_2_Activator: KIF18B = ASF1B (Confidence: 1, TimeStep: 1)

KIF18B_3_Activator: KIF18B = AURKA (Confidence: 1, TimeStep: 1)

KIF18B_4_Activator: KIF18B = !BTG1 (Confidence: 1, TimeStep: 1)

KIF18B_5_Activator: KIF18B = CCDC34 (Confidence: 1, TimeStep: 1)

KIF18B_1_Inhibitor: KIF18B = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF18B_2_Inhibitor: KIF18B = !ASF1B (Confidence: 1, TimeStep: 1)

KIF18B_3_Inhibitor: KIF18B = !AURKA (Confidence: 1, TimeStep: 1)

KIF18B_4_Inhibitor: KIF18B = BTG1 (Confidence: 1, TimeStep: 1)

KIF18B_5_Inhibitor: KIF18B = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF20A with decay value = 1:

KIF20A_1_Activator: KIF20A = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF20A_2_Activator: KIF20A = ASF1B (Confidence: 1, TimeStep: 1)

KIF20A_3_Activator: KIF20A = AURKA (Confidence: 1, TimeStep: 1)

KIF20A_4_Activator: KIF20A = !BTG1 (Confidence: 1, TimeStep: 1)

KIF20A_5_Activator: KIF20A = CCDC34 (Confidence: 1, TimeStep: 1)

KIF20A_1_Inhibitor: KIF20A = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF20A_2_Inhibitor: KIF20A = !ASF1B (Confidence: 1, TimeStep: 1)

KIF20A_3_Inhibitor: KIF20A = !AURKA (Confidence: 1, TimeStep: 1)

KIF20A_4_Inhibitor: KIF20A = BTG1 (Confidence: 1, TimeStep: 1)

KIF20A_5_Inhibitor: KIF20A = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF23 with decay value = 1:

KIF23_1_Activator: KIF23 = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF23_2_Activator: KIF23 = ASF1B (Confidence: 1, TimeStep: 1)

KIF23_3_Activator: KIF23 = AURKA (Confidence: 1, TimeStep: 1)

KIF23_4_Activator: KIF23 = !BTG1 (Confidence: 1, TimeStep: 1)

KIF23_5_Activator: KIF23 = CCDC34 (Confidence: 1, TimeStep: 1)

KIF23_1_Inhibitor: KIF23 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF23_2_Inhibitor: KIF23 = !ASF1B (Confidence: 1, TimeStep: 1)

KIF23_3_Inhibitor: KIF23 = !AURKA (Confidence: 1, TimeStep: 1)

KIF23_4_Inhibitor: KIF23 = BTG1 (Confidence: 1, TimeStep: 1)

KIF23_5_Inhibitor: KIF23 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF26A with decay value = 1:

KIF26A_1_Activator: KIF26A = TENM4 (Confidence: 1, TimeStep: 1)

KIF26A_2_Activator: KIF26A = !ZFP36L2 (Confidence: 1, TimeStep: 1)

KIF26A_3_Activator: KIF26A = AKAP12&TRIB1 (Confidence: 1, TimeStep: 1)

KIF26A_4_Activator: KIF26A = FABP5&IRAK3 (Confidence: 1, TimeStep: 1)

KIF26A_5_Activator: KIF26A = DPEP1&TRIB1 (Confidence: 1, TimeStep: 1)

KIF26A_1_Inhibitor: KIF26A = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

KIF26A_2_Inhibitor: KIF26A = !RAG1 (Confidence: 1, TimeStep: 1)

KIF26A_3_Inhibitor: KIF26A = LOC100130872 (Confidence: 1, TimeStep: 1)

KIF26A_4_Inhibitor: KIF26A = !METTL7A (Confidence: 1, TimeStep: 1)

KIF26A_5_Inhibitor: KIF26A = SEMA4D (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF2C with decay value = 1:

KIF2C_1_Activator: KIF2C = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF2C_2_Activator: KIF2C = ASF1B (Confidence: 1, TimeStep: 1)

KIF2C_3_Activator: KIF2C = AURKA (Confidence: 1, TimeStep: 1)

KIF2C_4_Activator: KIF2C = !BTG1 (Confidence: 1, TimeStep: 1)

KIF2C_5_Activator: KIF2C = CCDC34 (Confidence: 1, TimeStep: 1)

KIF2C_1_Inhibitor: KIF2C = !APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF2C_2_Inhibitor: KIF2C = !ASF1B (Confidence: 1, TimeStep: 1)

KIF2C_3_Inhibitor: KIF2C = !AURKA (Confidence: 1, TimeStep: 1)

KIF2C_4_Inhibitor: KIF2C = BTG1 (Confidence: 1, TimeStep: 1)

KIF2C_5_Inhibitor: KIF2C = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KIF4A with decay value = 1:

KIF4A_1_Activator: KIF4A = CENPH (Confidence: 1, TimeStep: 1)

KIF4A_2_Activator: KIF4A = APITD1-CORT (Confidence: 1, TimeStep: 1)

KIF4A_3_Activator: KIF4A = ASF1B (Confidence: 1, TimeStep: 1)

KIF4A_4_Activator: KIF4A = AURKA (Confidence: 1, TimeStep: 1)

KIF4A_5_Activator: KIF4A = !BTG1 (Confidence: 1, TimeStep: 1)

KIF4A_1_Inhibitor: KIF4A = !CENPH (Confidence: 1, TimeStep: 1)

KIF4A_2_Inhibitor: KIF4A = !CDK1 (Confidence: 1, TimeStep: 1)

KIF4A_3_Inhibitor: KIF4A = !HMMR (Confidence: 1, TimeStep: 1)

KIF4A_4_Inhibitor: KIF4A = !KIF14 (Confidence: 1, TimeStep: 1)

KIF4A_5_Inhibitor: KIF4A = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KLF9 with decay value = 1:

KLF9_1_Activator: KLF9 = KLF9 (Confidence: 1, TimeStep: 1)

KLF9_2_Activator: KLF9 = LGALS3 (Confidence: 1, TimeStep: 1)

KLF9_3_Activator: KLF9 = RAB31 (Confidence: 1, TimeStep: 1)

KLF9_4_Activator: KLF9 = !GGH&RBMS3 (Confidence: 1, TimeStep: 1)

KLF9_5_Activator: KLF9 = !HELLS&RBMS3 (Confidence: 1, TimeStep: 1)

KLF9_1_Inhibitor: KLF9 = !METTL7A (Confidence: 1, TimeStep: 1)

KLF9_2_Inhibitor: KLF9 = BCL10 (Confidence: 1, TimeStep: 1)

KLF9_3_Inhibitor: KLF9 = UBE2C (Confidence: 1, TimeStep: 1)

KLF9_4_Inhibitor: KLF9 = !KLF9&!RBMS3 (Confidence: 1, TimeStep: 1)

KLF9_5_Inhibitor: KLF9 = !DDIT4&!KLF9 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for KNL1 with decay value = 1:

KNL1_1_Activator: KNL1 = CCNB2 (Confidence: 1, TimeStep: 1)

KNL1_2_Activator: KNL1 = CDC45 (Confidence: 1, TimeStep: 1)

KNL1_3_Activator: KNL1 = CENPA (Confidence: 1, TimeStep: 1)

KNL1_4_Activator: KNL1 = CENPF (Confidence: 1, TimeStep: 1)

KNL1_5_Activator: KNL1 = DLGAP5 (Confidence: 1, TimeStep: 1)

KNL1_1_Inhibitor: KNL1 = !KNL1 (Confidence: 1, TimeStep: 1)

KNL1_2_Inhibitor: KNL1 = !ZNF367 (Confidence: 1, TimeStep: 1)

KNL1_3_Inhibitor: KNL1 = !E2F8 (Confidence: 1, TimeStep: 1)

KNL1_4_Inhibitor: KNL1 = !GINS2 (Confidence: 1, TimeStep: 1)

KNL1_5_Inhibitor: KNL1 = !DTL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LEF1 with decay value = 1:

LEF1_1_Activator: LEF1 = !CD53 (Confidence: 1, TimeStep: 1)

LEF1_2_Activator: LEF1 = CDC20 (Confidence: 1, TimeStep: 1)

LEF1_3_Activator: LEF1 = CENPK (Confidence: 1, TimeStep: 1)

LEF1_4_Activator: LEF1 = ZWINT (Confidence: 1, TimeStep: 1)

LEF1_5_Activator: LEF1 = ANLN (Confidence: 1, TimeStep: 1)

LEF1_1_Inhibitor: LEF1 = !ANLN&!CDK6 (Confidence: 1, TimeStep: 1)

LEF1_2_Inhibitor: LEF1 = !ANLN&!LEF1 (Confidence: 1, TimeStep: 1)

LEF1_3_Inhibitor: LEF1 = !BRIP1&!CDK6 (Confidence: 1, TimeStep: 1)

LEF1_4_Inhibitor: LEF1 = !BRIP1&!LEF1 (Confidence: 1, TimeStep: 1)

LEF1_5_Inhibitor: LEF1 = !BUB1B&!CDK6 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LGALS3 with decay value = 1:

LGALS3_1_Activator: LGALS3 = LGALS3 (Confidence: 1, TimeStep: 1)

LGALS3_2_Activator: LGALS3 = CDC42EP3&PPP1R16B (Confidence: 1, TimeStep: 1)

LGALS3_3_Activator: LGALS3 = CDC42EP3&!PRPS2 (Confidence: 1, TimeStep: 1)

LGALS3_4_Activator: LGALS3 = !CRNDE&HGB1 (Confidence: 1, TimeStep: 1)

LGALS3_5_Activator: LGALS3 = HBB&!PRPS2 (Confidence: 1, TimeStep: 1)

LGALS3_1_Inhibitor: LGALS3 = !LGALS3 (Confidence: 1, TimeStep: 1)

LGALS3_2_Inhibitor: LGALS3 = !PPP1R16B (Confidence: 1, TimeStep: 1)

LGALS3_3_Inhibitor: LGALS3 = PRPS2 (Confidence: 1, TimeStep: 1)

LGALS3_4_Inhibitor: LGALS3 = !CDC42EP3 (Confidence: 1, TimeStep: 1)

LGALS3_5_Inhibitor: LGALS3 = !HGB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LILRA1 with decay value = 1:

LILRA1_1_Activator: LILRA1 = LILRA1 (Confidence: 1, TimeStep: 1)

LILRA1_2_Activator: LILRA1 = FCER1G&KLF9 (Confidence: 1, TimeStep: 1)

LILRA1_3_Activator: LILRA1 = !ARPP21&FCER1G (Confidence: 1, TimeStep: 1)

LILRA1_4_Activator: LILRA1 = FCER1G&ZBTB16 (Confidence: 1, TimeStep: 1)

LILRA1_5_Activator: LILRA1 = FCGR3B&KLF9 (Confidence: 1, TimeStep: 1)

LILRA1_1_Inhibitor: LILRA1 = !KLF9 (Confidence: 1, TimeStep: 1)

LILRA1_2_Inhibitor: LILRA1 = ASPM (Confidence: 1, TimeStep: 1)

LILRA1_3_Inhibitor: LILRA1 = !LILRB2 (Confidence: 1, TimeStep: 1)

LILRA1_4_Inhibitor: LILRA1 = !IRNASET2 (Confidence: 1, TimeStep: 1)

LILRA1_5_Inhibitor: LILRA1 = KIF11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LILRB2 with decay value = 1:

LILRB2_1_Activator: LILRB2 = ITGAM (Confidence: 1, TimeStep: 1)

LILRB2_2_Activator: LILRB2 = TBXA2R (Confidence: 1, TimeStep: 1)

LILRB2_3_Activator: LILRB2 = MIR4683 (Confidence: 1, TimeStep: 1)

LILRB2_4_Activator: LILRB2 = CCR1 (Confidence: 1, TimeStep: 1)

LILRB2_5_Activator: LILRB2 = RPS6KA2 (Confidence: 1, TimeStep: 1)

LILRB2_1_Inhibitor: LILRB2 = ECT2 (Confidence: 1, TimeStep: 1)

LILRB2_2_Inhibitor: LILRB2 = FOXM1 (Confidence: 1, TimeStep: 1)

LILRB2_3_Inhibitor: LILRB2 = STIL (Confidence: 1, TimeStep: 1)

LILRB2_4_Inhibitor: LILRB2 = ATAD2 (Confidence: 1, TimeStep: 1)

LILRB2_5_Inhibitor: LILRB2 = BIRC5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LOC100130872 with decay value = 1:

LOC100130872_1_Activator: LOC100130872 = LOC100130872 (Confidence: 1, TimeStep: 1)

LOC100130872_2_Activator: LOC100130872 = !BCAT1&!MSH6 (Confidence: 1, TimeStep: 1)

LOC100130872_3_Activator: LOC100130872 = !C5orf24&FGR (Confidence: 1, TimeStep: 1)

LOC100130872_4_Activator: LOC100130872 = !C5orf24&!HRK (Confidence: 1, TimeStep: 1)

LOC100130872_5_Activator: LOC100130872 = !C5orf24&PPP1R16B (Confidence: 1, TimeStep: 1)

LOC100130872_1_Inhibitor: LOC100130872 = !HBG1 (Confidence: 1, TimeStep: 1)

LOC100130872_2_Inhibitor: LOC100130872 = !HBB (Confidence: 1, TimeStep: 1)

LOC100130872_3_Inhibitor: LOC100130872 = !SLA (Confidence: 1, TimeStep: 1)

LOC100130872_4_Inhibitor: LOC100130872 = HRK (Confidence: 1, TimeStep: 1)

LOC100130872_5_Inhibitor: LOC100130872 = !S100A8 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LOC100505650 with decay value = 1:

LOC100505650_1_Activator: LOC100505650 = LOC100505650 (Confidence: 1, TimeStep: 1)

LOC100505650_2_Activator: LOC100505650 = DEFA1&!ID2 (Confidence: 1, TimeStep: 1)

LOC100505650_3_Activator: LOC100505650 = !C5orf24&ECT2 (Confidence: 1, TimeStep: 1)

LOC100505650_4_Activator: LOC100505650 = !C5orf24&!ID2 (Confidence: 1, TimeStep: 1)

LOC100505650_5_Activator: LOC100505650 = !C5orf24&!METTL7A (Confidence: 1, TimeStep: 1)

LOC100505650_1_Inhibitor: LOC100505650 = DENND3 (Confidence: 1, TimeStep: 1)

LOC100505650_2_Inhibitor: LOC100505650 = TARSL2 (Confidence: 1, TimeStep: 1)

LOC100505650_3_Inhibitor: LOC100505650 = P2RY14 (Confidence: 1, TimeStep: 1)

LOC100505650_4_Inhibitor: LOC100505650 = SCML4 (Confidence: 1, TimeStep: 1)

LOC100505650_5_Inhibitor: LOC100505650 = MS4A4A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LOC100996643 with decay value = 1:

LOC100996643_1_Activator: LOC100996643 = !TXNIP (Confidence: 1, TimeStep: 1)

LOC100996643_2_Activator: LOC100996643 = CENPH (Confidence: 1, TimeStep: 1)

LOC100996643_3_Activator: LOC100996643 = CKAP2 (Confidence: 1, TimeStep: 1)

LOC100996643_4_Activator: LOC100996643 = APITD1-CORT (Confidence: 1, TimeStep: 1)

LOC100996643_5_Activator: LOC100996643 = ASF1B (Confidence: 1, TimeStep: 1)

LOC100996643_1_Inhibitor: LOC100996643 = !LOC100996643 (Confidence: 1, TimeStep: 1)

LOC100996643_2_Inhibitor: LOC100996643 = !BYSL (Confidence: 1, TimeStep: 1)

LOC100996643_3_Inhibitor: LOC100996643 = FGD2 (Confidence: 1, TimeStep: 1)

LOC100996643_4_Inhibitor: LOC100996643 = !FABP5 (Confidence: 1, TimeStep: 1)

LOC100996643_5_Inhibitor: LOC100996643 = SNX29P2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LOC285097 with decay value = 1:

LOC285097_1_Activator: LOC285097 = LOC285097&!PPIF (Confidence: 1, TimeStep: 1)

LOC285097_2_Activator: LOC285097 = LOC285097&!SEMA4D (Confidence: 1, TimeStep: 1)

LOC285097_3_Activator: LOC285097 = AKAP12&LOC285097 (Confidence: 1, TimeStep: 1)

LOC285097_4_Activator: LOC285097 = ISG20&LOC285097 (Confidence: 1, TimeStep: 1)

LOC285097_5_Activator: LOC285097 = B3GNT2&LOC285097 (Confidence: 1, TimeStep: 1)

LOC285097_1_Inhibitor: LOC285097 = SEMA4D (Confidence: 1, TimeStep: 1)

LOC285097_2_Inhibitor: LOC285097 = AKAP12&!LOC285097 (Confidence: 1, TimeStep: 1)

LOC285097_3_Inhibitor: LOC285097 = KIF26A&!LOC285097 (Confidence: 1, TimeStep: 1)

LOC285097_4_Inhibitor: LOC285097 = MSH6&PPIF (Confidence: 1, TimeStep: 1)

LOC285097_5_Inhibitor: LOC285097 = AKAP12&PPIF (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LOC728175 with decay value = 1:

LOC728175_1_Activator: LOC728175 = BCAT1&LOC728175 (Confidence: 1, TimeStep: 1)

LOC728175_2_Activator: LOC728175 = BCAT1&MYRIP (Confidence: 1, TimeStep: 1)
LOC728175_3_Activator: LOC728175 = !CELF2&LOC728175 (Confidence: 1, TimeStep: 1)
LOC728175_4_Activator: LOC728175 = ELL2&LOC728175 (Confidence: 1, TimeStep: 1)
LOC728175_5_Activator: LOC728175 = MYRIP&PTTG1 (Confidence: 1, TimeStep: 1)
LOC728175_1_Inhibitor: LOC728175 = !LOC728175 (Confidence: 1, TimeStep: 1)
LOC728175_2_Inhibitor: LOC728175 = !MYRIP (Confidence: 1, TimeStep: 1)
LOC728175_3_Inhibitor: LOC728175 = !BCAT1 (Confidence: 1, TimeStep: 1)
LOC728175_4_Inhibitor: LOC728175 = BIRC3 (Confidence: 1, TimeStep: 1)
LOC728175_5_Inhibitor: LOC728175 = IL27RA (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LY96 with decay value = 1:

LY96_1_Activator: LY96 = LY96 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for LYZ with decay value = 1:

LYZ_1_Activator: LYZ = F13A1 (Confidence: 1, TimeStep: 1)
LYZ_2_Activator: LYZ = LGALS3 (Confidence: 1, TimeStep: 1)
LYZ_3_Activator: LYZ = !CCNB1&IGH (Confidence: 1, TimeStep: 1)
LYZ_4_Activator: LYZ = !CENPV&MIR8071-1 (Confidence: 1, TimeStep: 1)
LYZ_5_Activator: LYZ = !BUB1B&IGH (Confidence: 1, TimeStep: 1)
LYZ_1_Inhibitor: LYZ = TMEM97 (Confidence: 1, TimeStep: 1)
LYZ_2_Inhibitor: LYZ = WDHD1 (Confidence: 1, TimeStep: 1)
LYZ_3_Inhibitor: LYZ = CENPH (Confidence: 1, TimeStep: 1)
LYZ_4_Inhibitor: LYZ = APITD1-CORT (Confidence: 1, TimeStep: 1)
LYZ_5_Inhibitor: LYZ = ASF1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MAD2L1 with decay value = 1:

MAD2L1_1_Activator: MAD2L1 = APITD1-CORT (Confidence: 1, TimeStep: 1)
MAD2L1_2_Activator: MAD2L1 = ASF1B (Confidence: 1, TimeStep: 1)
MAD2L1_3_Activator: MAD2L1 = AURKA (Confidence: 1, TimeStep: 1)
MAD2L1_4_Activator: MAD2L1 = !BTG1 (Confidence: 1, TimeStep: 1)
MAD2L1_5_Activator: MAD2L1 = CCDC34 (Confidence: 1, TimeStep: 1)
MAD2L1_1_Inhibitor: MAD2L1 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

MAD2L1_2_Inhibitor: MAD2L1 = !ASF1B (Confidence: 1, TimeStep: 1)

MAD2L1_3_Inhibitor: MAD2L1 = !AURKA (Confidence: 1, TimeStep: 1)

MAD2L1_4_Inhibitor: MAD2L1 = BTG1 (Confidence: 1, TimeStep: 1)

MAD2L1_5_Inhibitor: MAD2L1 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MCM10 with decay value = 1:

MCM10_1_Activator: MCM10 = !CD53&MDK (Confidence: 1, TimeStep: 1)

MCM10_2_Activator: MCM10 = CENPK&MDK (Confidence: 1, TimeStep: 1)

MCM10_3_Activator: MCM10 = !IGH&MDK (Confidence: 1, TimeStep: 1)

MCM10_4_Activator: MCM10 = !IGLC1&MDK (Confidence: 1, TimeStep: 1)

MCM10_5_Activator: MCM10 = !BMF&MDK (Confidence: 1, TimeStep: 1)

MCM10_1_Inhibitor: MCM10 = !CENPH (Confidence: 1, TimeStep: 1)

MCM10_2_Inhibitor: MCM10 = !CDK1 (Confidence: 1, TimeStep: 1)

MCM10_3_Inhibitor: MCM10 = !HMMR (Confidence: 1, TimeStep: 1)

MCM10_4_Inhibitor: MCM10 = !KIF14 (Confidence: 1, TimeStep: 1)

MCM10_5_Inhibitor: MCM10 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MCM4 with decay value = 1:

MCM4_1_Activator: MCM4 = APITD1-CORT (Confidence: 1, TimeStep: 1)

MCM4_2_Activator: MCM4 = ASF1B (Confidence: 1, TimeStep: 1)

MCM4_3_Activator: MCM4 = AURKA (Confidence: 1, TimeStep: 1)

MCM4_4_Activator: MCM4 = !BTG1 (Confidence: 1, TimeStep: 1)

MCM4_5_Activator: MCM4 = CCDC34 (Confidence: 1, TimeStep: 1)

MCM4_1_Inhibitor: MCM4 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

MCM4_2_Inhibitor: MCM4 = !ASF1B (Confidence: 1, TimeStep: 1)

MCM4_3_Inhibitor: MCM4 = !AURKA (Confidence: 1, TimeStep: 1)

MCM4_4_Inhibitor: MCM4 = BTG1 (Confidence: 1, TimeStep: 1)

MCM4_5_Inhibitor: MCM4 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MCM5 with decay value = 1:

MCM5_1_Activator: MCM5 = !ANP32E&BUB1B (Confidence: 1, TimeStep: 1)

MCM5_2_Activator: MCM5 = !ANP32E&C4orf46 (Confidence: 1, TimeStep: 1)

MCM5_3_Activator: MCM5 = !ANP32E&CCNB1 (Confidence: 1, TimeStep: 1)

MCM5_4_Activator: MCM5 = !ANP32E&CENPN (Confidence: 1, TimeStep: 1)

MCM5_5_Activator: MCM5 = !ANP32E&KIF15 (Confidence: 1, TimeStep: 1)

MCM5_1_Inhibitor: MCM5 = !CCNB1 (Confidence: 1, TimeStep: 1)

MCM5_2_Inhibitor: MCM5 = !BUB1B (Confidence: 1, TimeStep: 1)

MCM5_3_Inhibitor: MCM5 = !CENPN (Confidence: 1, TimeStep: 1)

MCM5_4_Inhibitor: MCM5 = !KIF15 (Confidence: 1, TimeStep: 1)

MCM5_5_Inhibitor: MCM5 = !MCM10 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MCM7 with decay value = 1:

MCM7_1_Activator: MCM7 = !APITD1-CORT&BRIP1 (Confidence: 1, TimeStep: 1)

MCM7_2_Activator: MCM7 = !APITD1-CORT&CHEK1 (Confidence: 1, TimeStep: 1)

MCM7_3_Activator: MCM7 = !APITD1-CORT&FANCI (Confidence: 1, TimeStep: 1)

MCM7_4_Activator: MCM7 = !APITD1-CORT&TTK (Confidence: 1, TimeStep: 1)

MCM7_5_Activator: MCM7 = !ASF1B&BRIP1 (Confidence: 1, TimeStep: 1)

MCM7_1_Inhibitor: MCM7 = !BRCA1 (Confidence: 1, TimeStep: 1)

MCM7_2_Inhibitor: MCM7 = !MCM7 (Confidence: 1, TimeStep: 1)

MCM7_3_Inhibitor: MCM7 = !BYSL (Confidence: 1, TimeStep: 1)

MCM7_4_Inhibitor: MCM7 = !PAICS (Confidence: 1, TimeStep: 1)

MCM7_5_Inhibitor: MCM7 = TBXA2R (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MDK with decay value = 1:

MDK_1_Activator: MDK = !CCNL1&MDK (Confidence: 1, TimeStep: 1)

MDK_2_Activator: MDK = BRCA1&MDK (Confidence: 1, TimeStep: 1)

MDK_3_Activator: MDK = BRIP1&MDK (Confidence: 1, TimeStep: 1)

MDK_4_Activator: MDK = BUB1B&MDK (Confidence: 1, TimeStep: 1)

MDK_5_Activator: MDK = C4orf46&MDK (Confidence: 1, TimeStep: 1)

MDK_1_Inhibitor: MDK = !MDK (Confidence: 1, TimeStep: 1)

MDK_2_Inhibitor: MDK = CLEC2B (Confidence: 1, TimeStep: 1)

MDK_3_Inhibitor: MDK = HBB (Confidence: 1, TimeStep: 1)

MDK_4_Inhibitor: MDK = !RMI2 (Confidence: 1, TimeStep: 1)

MDK_5_Inhibitor: MDK = CDC42EP3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MDM2 with decay value = 1:

MDM2_1_Activator: MDM2 = !IL6ST&MDM2 (Confidence: 1, TimeStep: 1)

MDM2_2_Activator: MDM2 = !IL6ST&TENM4 (Confidence: 1, TimeStep: 1)

MDM2_1_Inhibitor: MDM2 = !TMEM2 (Confidence: 1, TimeStep: 1)

MDM2_2_Inhibitor: MDM2 = !MDM2&!MYRIP (Confidence: 1, TimeStep: 1)

MDM2_3_Inhibitor: MDM2 = !KCNK12&!P2RY14 (Confidence: 1, TimeStep: 1)

MDM2_4_Inhibitor: MDM2 = !MYRIP&!P2RY14 (Confidence: 1, TimeStep: 1)

MDM2_5_Inhibitor: MDM2 = !P2RY14&!TENM4 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MELK with decay value = 1:

MELK_1_Activator: MELK = APITD1-CORT (Confidence: 1, TimeStep: 1)

MELK_2_Activator: MELK = ASF1B (Confidence: 1, TimeStep: 1)

MELK_3_Activator: MELK = AURKA (Confidence: 1, TimeStep: 1)

MELK_4_Activator: MELK = !BTG1 (Confidence: 1, TimeStep: 1)

MELK_5_Activator: MELK = CCDC34 (Confidence: 1, TimeStep: 1)

MELK_1_Inhibitor: MELK = !APITD1-CORT (Confidence: 1, TimeStep: 1)

MELK_2_Inhibitor: MELK = !ASF1B (Confidence: 1, TimeStep: 1)

MELK_3_Inhibitor: MELK = !AURKA (Confidence: 1, TimeStep: 1)

MELK_4_Inhibitor: MELK = BTG1 (Confidence: 1, TimeStep: 1)

MELK_5_Inhibitor: MELK = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for METTL7A with decay value = 1:

METTL7A_1_Activator: METTL7A = METTL7A (Confidence: 1, TimeStep: 1)

METTL7A_2_Activator: METTL7A = !ECT2 (Confidence: 1, TimeStep: 1)

METTL7A_3_Activator: METTL7A = TMEM2 (Confidence: 1, TimeStep: 1)

METTL7A_4_Activator: METTL7A = !CENPK (Confidence: 1, TimeStep: 1)

METTL7A_5_Activator: METTL7A = ISG20 (Confidence: 1, TimeStep: 1)

METTL7A_1_Inhibitor: METTL7A = !BMF&!TRIB1 (Confidence: 1, TimeStep: 1)

METTL7A_2_Inhibitor: METTL7A = !AKAP12&LOC100505650 (Confidence: 1, TimeStep: 1)

METTL7A_3_Inhibitor: METTL7A = !C5orf24&ECT2 (Confidence: 1, TimeStep: 1)

METTL7A_4_Inhibitor: METTL7A = !C5orf24&!ID2 (Confidence: 1, TimeStep: 1)

METTL7A_5_Inhibitor: METTL7A = !C5orf24&!METTL7A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MIR4683 with decay value = 1:

MIR4683_1_Activator: MIR4683 = MIR4683 (Confidence: 1, TimeStep: 1)

MIR4683_2_Activator: MIR4683 = RPS6KA2 (Confidence: 1, TimeStep: 1)

MIR4683_3_Activator: MIR4683 = BMF&RNASET2 (Confidence: 1, TimeStep: 1)

MIR4683_4_Activator: MIR4683 = C5orf24&RNASET2 (Confidence: 1, TimeStep: 1)

MIR4683_5_Activator: MIR4683 = AKAP12&RNASET2 (Confidence: 1, TimeStep: 1)

MIR4683_1_Inhibitor: MIR4683 = ECT2 (Confidence: 1, TimeStep: 1)

MIR4683_2_Inhibitor: MIR4683 = PPIF (Confidence: 1, TimeStep: 1)

MIR4683_3_Inhibitor: MIR4683 = FOXM1 (Confidence: 1, TimeStep: 1)

MIR4683_4_Inhibitor: MIR4683 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

MIR4683_5_Inhibitor: MIR4683 = !RAG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MIR6845 with decay value = 1:

MIR6845_1_Activator: MIR6845 = MIR6845 (Confidence: 1, TimeStep: 1)

MIR6845_2_Activator: MIR6845 = !BYSL (Confidence: 1, TimeStep: 1)

MIR6845_3_Activator: MIR6845 = !PAICS (Confidence: 1, TimeStep: 1)

MIR6845_4_Activator: MIR6845 = !CRNDE (Confidence: 1, TimeStep: 1)

MIR6845_5_Activator: MIR6845 = MIR4683 (Confidence: 1, TimeStep: 1)

MIR6845_1_Inhibitor: MIR6845 = ATAD2 (Confidence: 1, TimeStep: 1)

MIR6845_2_Inhibitor: MIR6845 = BIRC5 (Confidence: 1, TimeStep: 1)

MIR6845_3_Inhibitor: MIR6845 = BUB1 (Confidence: 1, TimeStep: 1)

MIR6845_4_Inhibitor: MIR6845 = CCNA2 (Confidence: 1, TimeStep: 1)

MIR6845_5_Inhibitor: MIR6845 = CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MIR8071-1 with decay value = 1:

MIR8071-1_1_Activator: MIR8071-1 = MIR8071-1 (Confidence: 1, TimeStep: 1)

MIR8071-1_2_Activator: MIR8071-1 = F13A1 (Confidence: 1, TimeStep: 1)

MIR8071-1_3_Activator: MIR8071-1 = LGALS3 (Confidence: 1, TimeStep: 1)

MIR8071-1_4_Activator: MIR8071-1 = MNDA (Confidence: 1, TimeStep: 1)

MIR8071-1_5_Activator: MIR8071-1 = IGLC1&!TMEM97 (Confidence: 1, TimeStep: 1)

MIR8071-1_1_Inhibitor: MIR8071-1 = !ZFP36L2 (Confidence: 1, TimeStep: 1)

MIR8071-1_2_Inhibitor: MIR8071-1 = LYZ&!MIR8071-1 (Confidence: 1, TimeStep: 1)

MIR8071-1_3_Inhibitor: MIR8071-1 = !APITD1-CORT&!MS4A1 (Confidence: 1, TimeStep: 1)

MIR8071-1_4_Inhibitor: MIR8071-1 = !ASF1B&!MS4A1 (Confidence: 1, TimeStep: 1)

MIR8071-1_5_Inhibitor: MIR8071-1 = !ATAD2&!MS4A1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MKI67 with decay value = 1:

MKI67_1_Activator: MKI67 = APITD1-CORT (Confidence: 1, TimeStep: 1)

MKI67_2_Activator: MKI67 = ASF1B (Confidence: 1, TimeStep: 1)

MKI67_3_Activator: MKI67 = AURKA (Confidence: 1, TimeStep: 1)

MKI67_4_Activator: MKI67 = !BTG1 (Confidence: 1, TimeStep: 1)

MKI67_5_Activator: MKI67 = CCDC34 (Confidence: 1, TimeStep: 1)

MKI67_1_Inhibitor: MKI67 = !RRM2 (Confidence: 1, TimeStep: 1)

MKI67_2_Inhibitor: MKI67 = !CENPW (Confidence: 1, TimeStep: 1)

MKI67_3_Inhibitor: MKI67 = !TCF19 (Confidence: 1, TimeStep: 1)

MKI67_4_Inhibitor: MKI67 = !WDR76 (Confidence: 1, TimeStep: 1)

MKI67_5_Inhibitor: MKI67 = !DFNA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MND1 with decay value = 1:

MND1_1_Activator: MND1 = PTTG1 (Confidence: 1, TimeStep: 1)

MND1_2_Activator: MND1 = CDK1 (Confidence: 1, TimeStep: 1)

MND1_3_Activator: MND1 = HMMR (Confidence: 1, TimeStep: 1)

MND1_4_Activator: MND1 = KIF14 (Confidence: 1, TimeStep: 1)

MND1_5_Activator: MND1 = KIF20A (Confidence: 1, TimeStep: 1)

MND1_1_Inhibitor: MND1 = !MND1 (Confidence: 1, TimeStep: 1)

MND1_2_Inhibitor: MND1 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

MND1_3_Inhibitor: MND1 = !PAICS (Confidence: 1, TimeStep: 1)

MND1_4_Inhibitor: MND1 = !CRNDE (Confidence: 1, TimeStep: 1)

MND1_5_Inhibitor: MND1 = !SELENOI (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MNDA with decay value = 1:

MNDA_1_Activator: MNDA = !C5orf24&!ECT2 (Confidence: 1, TimeStep: 1)

MNDA_2_Activator: MNDA = IGLC1&!LEF1 (Confidence: 1, TimeStep: 1)
MNDA_3_Activator: MNDA = IGLC1&MNDA (Confidence: 1, TimeStep: 1)
MNDA_4_Activator: MNDA = ITPKB&MNDA (Confidence: 1, TimeStep: 1)
MNDA_5_Activator: MNDA = !C5orf24&FGD2 (Confidence: 1, TimeStep: 1)
MNDA_1_Inhibitor: MNDA = LOC100996643 (Confidence: 1, TimeStep: 1)
MNDA_2_Inhibitor: MNDA = !MIR8071-1 (Confidence: 1, TimeStep: 1)
MNDA_3_Inhibitor: MNDA = !CD53 (Confidence: 1, TimeStep: 1)
MNDA_4_Inhibitor: MNDA = ZWINT (Confidence: 1, TimeStep: 1)
MNDA_5_Inhibitor: MNDA = ANLN (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MPV17L with decay value = 1:

MPV17L_1_Activator: MPV17L = FABP5&!IGLL1 (Confidence: 1, TimeStep: 1)
MPV17L_2_Activator: MPV17L = FABP5&SNX29P2 (Confidence: 1, TimeStep: 1)
MPV17L_3_Activator: MPV17L = BYSL&!IGLL1 (Confidence: 1, TimeStep: 1)
MPV17L_4_Activator: MPV17L = FABP5&MPV17L (Confidence: 1, TimeStep: 1)
MPV17L_5_Activator: MPV17L = BYSL&MPV17L (Confidence: 1, TimeStep: 1)
MPV17L_1_Inhibitor: MPV17L = KIF4A (Confidence: 1, TimeStep: 1)
MPV17L_2_Inhibitor: MPV17L = RFC3 (Confidence: 1, TimeStep: 1)
MPV17L_3_Inhibitor: MPV17L = SHCBP1 (Confidence: 1, TimeStep: 1)
MPV17L_4_Inhibitor: MPV17L = FH (Confidence: 1, TimeStep: 1)
MPV17L_5_Inhibitor: MPV17L = CCNB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MS4A1 with decay value = 1:

MS4A1_1_Activator: MS4A1 = MIR8071-1 (Confidence: 1, TimeStep: 1)
MS4A1_2_Activator: MS4A1 = MTSS1 (Confidence: 1, TimeStep: 1)
MS4A1_3_Activator: MS4A1 = SERPINB9 (Confidence: 1, TimeStep: 1)
MS4A1_4_Activator: MS4A1 = IGLC1 (Confidence: 1, TimeStep: 1)
MS4A1_5_Activator: MS4A1 = ITGAM (Confidence: 1, TimeStep: 1)
MS4A1_1_Inhibitor: MS4A1 = APITD1-CORT (Confidence: 1, TimeStep: 1)
MS4A1_2_Inhibitor: MS4A1 = ASF1B (Confidence: 1, TimeStep: 1)
MS4A1_3_Inhibitor: MS4A1 = AURKA (Confidence: 1, TimeStep: 1)
MS4A1_4_Inhibitor: MS4A1 = !BTG1 (Confidence: 1, TimeStep: 1)

MS4A1_5_Inhibitor: MS4A1 = CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MS4A4A with decay value = 1:

MS4A4A_1_Activator: MS4A4A = !F13A1&FGD2 (Confidence: 1, TimeStep: 1)

MS4A4A_2_Activator: MS4A4A = !MS4A4A&TARSL2 (Confidence: 1, TimeStep: 1)

MS4A4A_3_Activator: MS4A4A = !ID2&MTSS1 (Confidence: 1, TimeStep: 1)

MS4A4A_4_Activator: MS4A4A = !MIR6845&TARSL2 (Confidence: 1, TimeStep: 1)

MS4A4A_5_Activator: MS4A4A = CENPV&HRK (Confidence: 1, TimeStep: 1)

MS4A4A_1_Inhibitor: MS4A4A = !SMAP2 (Confidence: 1, TimeStep: 1)

MS4A4A_2_Inhibitor: MS4A4A = ECT2 (Confidence: 1, TimeStep: 1)

MS4A4A_3_Inhibitor: MS4A4A = FOXM1 (Confidence: 1, TimeStep: 1)

MS4A4A_4_Inhibitor: MS4A4A = STIL (Confidence: 1, TimeStep: 1)

MS4A4A_5_Inhibitor: MS4A4A = ATAD2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MS4A7 with decay value = 1:

MS4A7_1_Activator: MS4A7 = LILRA1 (Confidence: 1, TimeStep: 1)

MS4A7_2_Activator: MS4A7 = !E2F7&MS4A7 (Confidence: 1, TimeStep: 1)

MS4A7_3_Activator: MS4A7 = !L6ST&MS4A7 (Confidence: 1, TimeStep: 1)

MS4A7_4_Activator: MS4A7 = !CCNL1&FGD2 (Confidence: 1, TimeStep: 1)

MS4A7_5_Activator: MS4A7 = !CENPV&FGD2 (Confidence: 1, TimeStep: 1)

MS4A7_1_Inhibitor: MS4A7 = APITD1-CORT (Confidence: 1, TimeStep: 1)

MS4A7_2_Inhibitor: MS4A7 = ASF1B (Confidence: 1, TimeStep: 1)

MS4A7_3_Inhibitor: MS4A7 = AURKA (Confidence: 1, TimeStep: 1)

MS4A7_4_Inhibitor: MS4A7 = !BTG1 (Confidence: 1, TimeStep: 1)

MS4A7_5_Inhibitor: MS4A7 = CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MSH6 with decay value = 1:

MSH6_1_Activator: MSH6 = HRK (Confidence: 1, TimeStep: 1)

MSH6_2_Activator: MSH6 = TENM4 (Confidence: 1, TimeStep: 1)

MSH6_3_Activator: MSH6 = AKAP12&RMI2 (Confidence: 1, TimeStep: 1)

MSH6_4_Activator: MSH6 = ABHD17B&!SNX10 (Confidence: 1, TimeStep: 1)

MSH6_5_Activator: MSH6 = GSN&RMI2 (Confidence: 1, TimeStep: 1)

MSH6_1_Inhibitor: MSH6 = !MSH6 (Confidence: 1, TimeStep: 1)

MSH6_2_Inhibitor: MSH6 = !ID3 (Confidence: 1, TimeStep: 1)

MSH6_3_Inhibitor: MSH6 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

MSH6_4_Inhibitor: MSH6 = !ABHD17B (Confidence: 1, TimeStep: 1)

MSH6_5_Inhibitor: MSH6 = LOC100130872 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MTHFD2 with decay value = 1:

MTHFD2_1_Activator: MTHFD2 = !TXNIP (Confidence: 1, TimeStep: 1)

MTHFD2_2_Activator: MTHFD2 = ATAD2 (Confidence: 1, TimeStep: 1)

MTHFD2_3_Activator: MTHFD2 = BIRC5 (Confidence: 1, TimeStep: 1)

MTHFD2_4_Activator: MTHFD2 = BUB1 (Confidence: 1, TimeStep: 1)

MTHFD2_5_Activator: MTHFD2 = CCNA2 (Confidence: 1, TimeStep: 1)

MTHFD2_1_Inhibitor: MTHFD2 = !CCDC86 (Confidence: 1, TimeStep: 1)

MTHFD2_2_Inhibitor: MTHFD2 = !PAICS (Confidence: 1, TimeStep: 1)

MTHFD2_3_Inhibitor: MTHFD2 = ITGAM (Confidence: 1, TimeStep: 1)

MTHFD2_4_Inhibitor: MTHFD2 = !SELENOI (Confidence: 1, TimeStep: 1)

MTHFD2_5_Inhibitor: MTHFD2 = CDC42EP3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MTSS1 with decay value = 1:

MTSS1_1_Activator: MTSS1 = MTSS1 (Confidence: 1, TimeStep: 1)

MTSS1_2_Activator: MTSS1 = SERPINB9 (Confidence: 1, TimeStep: 1)

MTSS1_3_Activator: MTSS1 = ITGAM (Confidence: 1, TimeStep: 1)

MTSS1_4_Activator: MTSS1 = HRK (Confidence: 1, TimeStep: 1)

MTSS1_5_Activator: MTSS1 = CCR1 (Confidence: 1, TimeStep: 1)

MTSS1_1_Inhibitor: MTSS1 = !SMAP2 (Confidence: 1, TimeStep: 1)

MTSS1_2_Inhibitor: MTSS1 = ECT2 (Confidence: 1, TimeStep: 1)

MTSS1_3_Inhibitor: MTSS1 = !MS4A1 (Confidence: 1, TimeStep: 1)

MTSS1_4_Inhibitor: MTSS1 = FOXM1 (Confidence: 1, TimeStep: 1)

MTSS1_5_Inhibitor: MTSS1 = STIL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for MYRIP with decay value = 1:

MYRIP_1_Activator: MYRIP = LOC728175 (Confidence: 1, TimeStep: 1)

MYRIP_2_Activator: MYRIP = !BIRC3&MDM2 (Confidence: 1, TimeStep: 1)

MYRIP_3_Activator: MYRIP = ELL2&MYRIP (Confidence: 1, TimeStep: 1)

MYRIP_4_Activator: MYRIP = KCNK12&!SNX10 (Confidence: 1, TimeStep: 1)

MYRIP_5_Activator: MYRIP = LOC100996643&MDM2 (Confidence: 1, TimeStep: 1)

MYRIP_1_Inhibitor: MYRIP = FGR (Confidence: 1, TimeStep: 1)

MYRIP_2_Inhibitor: MYRIP = !PTP4A1 (Confidence: 1, TimeStep: 1)

MYRIP_3_Inhibitor: MYRIP = !ID3 (Confidence: 1, TimeStep: 1)

MYRIP_4_Inhibitor: MYRIP = !ELL2&SNX10 (Confidence: 1, TimeStep: 1)

MYRIP_5_Inhibitor: MYRIP = !IL6ST&!LOC728175 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NCAPG with decay value = 1:

NCAPG_1_Activator: NCAPG = APITD1-CORT (Confidence: 1, TimeStep: 1)

NCAPG_2_Activator: NCAPG = ASF1B (Confidence: 1, TimeStep: 1)

NCAPG_3_Activator: NCAPG = AURKA (Confidence: 1, TimeStep: 1)

NCAPG_4_Activator: NCAPG = !BTG1 (Confidence: 1, TimeStep: 1)

NCAPG_5_Activator: NCAPG = CCDC34 (Confidence: 1, TimeStep: 1)

NCAPG_1_Inhibitor: NCAPG = !APITD1-CORT (Confidence: 1, TimeStep: 1)

NCAPG_2_Inhibitor: NCAPG = !ASF1B (Confidence: 1, TimeStep: 1)

NCAPG_3_Inhibitor: NCAPG = !AURKA (Confidence: 1, TimeStep: 1)

NCAPG_4_Inhibitor: NCAPG = BTG1 (Confidence: 1, TimeStep: 1)

NCAPG_5_Inhibitor: NCAPG = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NCAPH with decay value = 1:

NCAPH_1_Activator: NCAPH = APITD1-CORT (Confidence: 1, TimeStep: 1)

NCAPH_2_Activator: NCAPH = ASF1B (Confidence: 1, TimeStep: 1)

NCAPH_3_Activator: NCAPH = AURKA (Confidence: 1, TimeStep: 1)

NCAPH_4_Activator: NCAPH = !BTG1 (Confidence: 1, TimeStep: 1)

NCAPH_5_Activator: NCAPH = CCDC34 (Confidence: 1, TimeStep: 1)

NCAPH_1_Inhibitor: NCAPH = !APITD1-CORT (Confidence: 1, TimeStep: 1)

NCAPH_2_Inhibitor: NCAPH = !ASF1B (Confidence: 1, TimeStep: 1)

NCAPH_3_Inhibitor: NCAPH = !AURKA (Confidence: 1, TimeStep: 1)

NCAPH_4_Inhibitor: NCAPH = BTG1 (Confidence: 1, TimeStep: 1)

NCAPH_5_Inhibitor: NCAPH = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NEAT1 with decay value = 1:

NEAT1_1_Activator: NEAT1 = !ANP32E (Confidence: 1, TimeStep: 1)

NEAT1_2_Activator: NEAT1 = !FH (Confidence: 1, TimeStep: 1)

NEAT1_3_Activator: NEAT1 = NEAT1 (Confidence: 1, TimeStep: 1)

NEAT1_4_Activator: NEAT1 = !RFC3 (Confidence: 1, TimeStep: 1)

NEAT1_5_Activator: NEAT1 = !MTHFD2 (Confidence: 1, TimeStep: 1)

NEAT1_1_Inhibitor: NEAT1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

NEAT1_2_Inhibitor: NEAT1 = ASF1B (Confidence: 1, TimeStep: 1)

NEAT1_3_Inhibitor: NEAT1 = AURKA (Confidence: 1, TimeStep: 1)

NEAT1_4_Inhibitor: NEAT1 = !BTG1 (Confidence: 1, TimeStep: 1)

NEAT1_5_Inhibitor: NEAT1 = CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NEDD9 with decay value = 1:

NEDD9_1_Activator: NEDD9 = RAB31 (Confidence: 1, TimeStep: 1)

NEDD9_2_Activator: NEDD9 = !ANLN&ITGAM (Confidence: 1, TimeStep: 1)

NEDD9_3_Activator: NEDD9 = !BCAT1&NEDD9 (Confidence: 1, TimeStep: 1)

NEDD9_4_Activator: NEDD9 = !BYSL&NEDD9 (Confidence: 1, TimeStep: 1)

NEDD9_5_Activator: NEDD9 = !ASPM&ITGAM (Confidence: 1, TimeStep: 1)

NEDD9_1_Inhibitor: NEDD9 = BYSL&!WFS1 (Confidence: 1, TimeStep: 1)

NEDD9_2_Inhibitor: NEDD9 = !AURKB&!RNASET2 (Confidence: 1, TimeStep: 1)

NEDD9_3_Inhibitor: NEDD9 = !ATAD2&!RNASET2 (Confidence: 1, TimeStep: 1)

NEDD9_4_Inhibitor: NEDD9 = !BIRC5&!RNASET2 (Confidence: 1, TimeStep: 1)

NEDD9_5_Inhibitor: NEDD9 = !BUB1&!RNASET2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NEK2 with decay value = 1:

NEK2_1_Activator: NEK2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

NEK2_2_Activator: NEK2 = ASF1B (Confidence: 1, TimeStep: 1)

NEK2_3_Activator: NEK2 = AURKA (Confidence: 1, TimeStep: 1)

NEK2_4_Activator: NEK2 = !BTG1 (Confidence: 1, TimeStep: 1)

NEK2_5_Activator: NEK2 = CCDC34 (Confidence: 1, TimeStep: 1)

NEK2_1_Inhibitor: NEK2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

NEK2_2_Inhibitor: NEK2 = !ASF1B (Confidence: 1, TimeStep: 1)

NEK2_3_Inhibitor: NEK2 = !AURKA (Confidence: 1, TimeStep: 1)

NEK2_4_Inhibitor: NEK2 = BTG1 (Confidence: 1, TimeStep: 1)

NEK2_5_Inhibitor: NEK2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NME1 with decay value = 1:

NME1_1_Activator: NME1 = CENPH (Confidence: 1, TimeStep: 1)

NME1_2_Activator: NME1 = CKAP2 (Confidence: 1, TimeStep: 1)

NME1_3_Activator: NME1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

NME1_4_Activator: NME1 = ASF1B (Confidence: 1, TimeStep: 1)

NME1_5_Activator: NME1 = AURKA (Confidence: 1, TimeStep: 1)

NME1_1_Inhibitor: NME1 = LYZ (Confidence: 1, TimeStep: 1)

NME1_2_Inhibitor: NME1 = !LOC100996643 (Confidence: 1, TimeStep: 1)

NME1_3_Inhibitor: NME1 = MIR8071-1 (Confidence: 1, TimeStep: 1)

NME1_4_Inhibitor: NME1 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

NME1_5_Inhibitor: NME1 = !BYSL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NPCDR1 with decay value = 1:

NPCDR1_1_Activator: NPCDR1 = ABHD17B&CDC42EP3 (Confidence: 1, TimeStep: 1)

NPCDR1_2_Activator: NPCDR1 = !BCL2L11&CDC42EP3 (Confidence: 1, TimeStep: 1)

NPCDR1_3_Activator: NPCDR1 = !BCL2L11&!LOC100996643 (Confidence: 1, TimeStep: 1)

NPCDR1_4_Activator: NPCDR1 = !BCL2L11&!PAICS (Confidence: 1, TimeStep: 1)

NPCDR1_5_Activator: NPCDR1 = BMF&CDC42EP3 (Confidence: 1, TimeStep: 1)

NPCDR1_1_Inhibitor: NPCDR1 = CDT1 (Confidence: 1, TimeStep: 1)

NPCDR1_2_Inhibitor: NPCDR1 = BRIP1 (Confidence: 1, TimeStep: 1)

NPCDR1_3_Inhibitor: NPCDR1 = CHEK1 (Confidence: 1, TimeStep: 1)

NPCDR1_4_Inhibitor: NPCDR1 = FANCI (Confidence: 1, TimeStep: 1)

NPCDR1_5_Inhibitor: NPCDR1 = TTK (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NUF2 with decay value = 1:

NUF2_1_Activator: NUF2 = CENPH (Confidence: 1, TimeStep: 1)

NUF2_2_Activator: NUF2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

NUF2_3_Activator: NUF2 = ASF1B (Confidence: 1, TimeStep: 1)

NUF2_4_Activator: NUF2 = AURKA (Confidence: 1, TimeStep: 1)

NUF2_5_Activator: NUF2 = !BTG1 (Confidence: 1, TimeStep: 1)

NUF2_1_Inhibitor: NUF2 = !CENPF (Confidence: 1, TimeStep: 1)

NUF2_2_Inhibitor: NUF2 = !NEK2 (Confidence: 1, TimeStep: 1)

NUF2_3_Inhibitor: NUF2 = !FH (Confidence: 1, TimeStep: 1)

NUF2_4_Inhibitor: NUF2 = !C4orf46 (Confidence: 1, TimeStep: 1)

NUF2_5_Inhibitor: NUF2 = !HELLS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for NUSAP1 with decay value = 1:

NUSAP1_1_Activator: NUSAP1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

NUSAP1_2_Activator: NUSAP1 = ASF1B (Confidence: 1, TimeStep: 1)

NUSAP1_3_Activator: NUSAP1 = AURKA (Confidence: 1, TimeStep: 1)

NUSAP1_4_Activator: NUSAP1 = !BTG1 (Confidence: 1, TimeStep: 1)

NUSAP1_5_Activator: NUSAP1 = CCDC34 (Confidence: 1, TimeStep: 1)

NUSAP1_1_Inhibitor: NUSAP1 = !CENPF (Confidence: 1, TimeStep: 1)

NUSAP1_2_Inhibitor: NUSAP1 = !DEPDC1B (Confidence: 1, TimeStep: 1)

NUSAP1_3_Inhibitor: NUSAP1 = !NEK2 (Confidence: 1, TimeStep: 1)

NUSAP1_4_Inhibitor: NUSAP1 = !ANP32E (Confidence: 1, TimeStep: 1)

NUSAP1_5_Inhibitor: NUSAP1 = !FH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for OIP5 with decay value = 1:

OIP5_1_Activator: OIP5 = APITD1-CORT (Confidence: 1, TimeStep: 1)

OIP5_2_Activator: OIP5 = ASF1B (Confidence: 1, TimeStep: 1)

OIP5_3_Activator: OIP5 = AURKA (Confidence: 1, TimeStep: 1)

OIP5_4_Activator: OIP5 = !BTG1 (Confidence: 1, TimeStep: 1)

OIP5_5_Activator: OIP5 = CCDC34 (Confidence: 1, TimeStep: 1)

OIP5_1_Inhibitor: OIP5 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

OIP5_2_Inhibitor: OIP5 = !ASF1B (Confidence: 1, TimeStep: 1)

OIP5_3_Inhibitor: OIP5 = !AURKA (Confidence: 1, TimeStep: 1)

OIP5_4_Inhibitor: OIP5 = BTG1 (Confidence: 1, TimeStep: 1)

OIP5_5_Inhibitor: OIP5 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for P2RX5 with decay value = 1:

P2RX5_1_Activator: P2RX5 = CRNDE&P2RX5 (Confidence: 1, TimeStep: 1)

P2RX5_2_Activator: P2RX5 = DFNA5&P2RX5 (Confidence: 1, TimeStep: 1)

P2RX5_3_Activator: P2RX5 = KIF11&P2RX5 (Confidence: 1, TimeStep: 1)

P2RX5_4_Activator: P2RX5 = CDC20&P2RX5 (Confidence: 1, TimeStep: 1)

P2RX5_5_Activator: P2RX5 = KIF4A&P2RX5 (Confidence: 1, TimeStep: 1)

P2RX5_1_Inhibitor: P2RX5 = !DFNA5&IQGAP3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for P2RY14 with decay value = 1:

P2RY14_1_Activator: P2RY14 = C5orf24&DENND3 (Confidence: 1, TimeStep: 1)

P2RY14_2_Activator: P2RY14 = !BCL2L11&DENND3 (Confidence: 1, TimeStep: 1)

P2RY14_3_Activator: P2RY14 = DPEP1&IL1B (Confidence: 1, TimeStep: 1)

P2RY14_4_Activator: P2RY14 = BMF&DENND3 (Confidence: 1, TimeStep: 1)

P2RY14_5_Activator: P2RY14 = DPEP1&PDE4B (Confidence: 1, TimeStep: 1)

P2RY14_1_Inhibitor: P2RY14 = !METTL7A (Confidence: 1, TimeStep: 1)

P2RY14_2_Inhibitor: P2RY14 = UBE2C (Confidence: 1, TimeStep: 1)

P2RY14_3_Inhibitor: P2RY14 = CENPV&!KIF26A (Confidence: 1, TimeStep: 1)

P2RY14_4_Inhibitor: P2RY14 = !GSN&!IRAK3 (Confidence: 1, TimeStep: 1)

P2RY14_5_Inhibitor: P2RY14 = !GSN&!PDE4B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PAICS with decay value = 1:

PAICS_1_Activator: PAICS = !CD53 (Confidence: 1, TimeStep: 1)

PAICS_2_Activator: PAICS = GGH (Confidence: 1, TimeStep: 1)

PAICS_3_Activator: PAICS = FOXM1 (Confidence: 1, TimeStep: 1)

PAICS_4_Activator: PAICS = KIF2C (Confidence: 1, TimeStep: 1)

PAICS_5_Activator: PAICS = STIL (Confidence: 1, TimeStep: 1)

PAICS_1_Inhibitor: PAICS = !BYSL (Confidence: 1, TimeStep: 1)

PAICS_2_Inhibitor: PAICS = !PAICS (Confidence: 1, TimeStep: 1)

PAICS_3_Inhibitor: PAICS = !FABP5 (Confidence: 1, TimeStep: 1)

PAICS_4_Inhibitor: PAICS = F13A1 (Confidence: 1, TimeStep: 1)

PAICS_5_Inhibitor: PAICS = LGALS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PBK with decay value = 1:

PBK_1_Activator: PBK = APITD1-CORT (Confidence: 1, TimeStep: 1)

PBK_2_Activator: PBK = ASF1B (Confidence: 1, TimeStep: 1)

PBK_3_Activator: PBK = AURKA (Confidence: 1, TimeStep: 1)

PBK_4_Activator: PBK = !BTG1 (Confidence: 1, TimeStep: 1)

PBK_5_Activator: PBK = CCDC34 (Confidence: 1, TimeStep: 1)

PBK_1_Inhibitor: PBK = !APITD1-CORT (Confidence: 1, TimeStep: 1)

PBK_2_Inhibitor: PBK = !ASF1B (Confidence: 1, TimeStep: 1)

PBK_3_Inhibitor: PBK = !AURKA (Confidence: 1, TimeStep: 1)

PBK_4_Inhibitor: PBK = BTG1 (Confidence: 1, TimeStep: 1)

PBK_5_Inhibitor: PBK = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PCNA with decay value = 1:

PCNA_1_Activator: PCNA = BUB1B (Confidence: 1, TimeStep: 1)

PCNA_2_Activator: PCNA = CENPN (Confidence: 1, TimeStep: 1)

PCNA_3_Activator: PCNA = KIF15 (Confidence: 1, TimeStep: 1)

PCNA_4_Activator: PCNA = MCM10 (Confidence: 1, TimeStep: 1)

PCNA_5_Activator: PCNA = PLK4 (Confidence: 1, TimeStep: 1)

PCNA_1_Inhibitor: PCNA = !CDC20 (Confidence: 1, TimeStep: 1)

PCNA_2_Inhibitor: PCNA = !CENPW (Confidence: 1, TimeStep: 1)

PCNA_3_Inhibitor: PCNA = !DHFR (Confidence: 1, TimeStep: 1)

PCNA_4_Inhibitor: PCNA = !RAD51 (Confidence: 1, TimeStep: 1)

PCNA_5_Inhibitor: PCNA = !KIF11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PDE4B with decay value = 1:

PDE4B_1_Activator: PDE4B = IL18R1 (Confidence: 1, TimeStep: 1)

PDE4B_2_Activator: PDE4B = F13A1 (Confidence: 1, TimeStep: 1)

PDE4B_3_Activator: PDE4B = IL18RAP (Confidence: 1, TimeStep: 1)

PDE4B_4_Activator: PDE4B = !ASPM&PDE4B (Confidence: 1, TimeStep: 1)

PDE4B_5_Activator: PDE4B = !ANP32E&MS4A4A (Confidence: 1, TimeStep: 1)

PDE4B_1_Inhibitor: PDE4B = !IRAK3 (Confidence: 1, TimeStep: 1)

PDE4B_2_Inhibitor: PDE4B = !TMEM2 (Confidence: 1, TimeStep: 1)

PDE4B_3_Inhibitor: PDE4B = ANP32E (Confidence: 1, TimeStep: 1)

PDE4B_4_Inhibitor: PDE4B = ECT2 (Confidence: 1, TimeStep: 1)

PDE4B_5_Inhibitor: PDE4B = FH (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PFKFB2 with decay value = 1:

PFKFB2_1_Activator: PFKFB2 = !MYRIP&PFKFB2 (Confidence: 1, TimeStep: 1)

PFKFB2_2_Activator: PFKFB2 = !LOC728175&TNFRSF21 (Confidence: 1, TimeStep: 2)

PFKFB2_3_Activator: PFKFB2 = !MYRIP&TNFRSF21 (Confidence: 1, TimeStep: 2)

PFKFB2_1_Inhibitor: PFKFB2 = LOC728175 (Confidence: 1, TimeStep: 1)

PFKFB2_2_Inhibitor: PFKFB2 = !BIRC3&LOC100996643 (Confidence: 1, TimeStep: 1)

PFKFB2_3_Inhibitor: PFKFB2 = LOC100996643&MYRIP (Confidence: 1, TimeStep: 1)

PFKFB2_4_Inhibitor: PFKFB2 = MYRIP&!PFKFB2 (Confidence: 1, TimeStep: 1)

PFKFB2_5_Inhibitor: PFKFB2 = !L6ST&LOC100996643 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PHF19 with decay value = 1:

PHF19_1_Activator: PHF19 = APITD1-CORT (Confidence: 1, TimeStep: 1)

PHF19_2_Activator: PHF19 = ASF1B (Confidence: 1, TimeStep: 1)

PHF19_3_Activator: PHF19 = AURKA (Confidence: 1, TimeStep: 1)

PHF19_4_Activator: PHF19 = !BTG1 (Confidence: 1, TimeStep: 1)

PHF19_5_Activator: PHF19 = CCDC34 (Confidence: 1, TimeStep: 1)

PHF19_1_Inhibitor: PHF19 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

PHF19_2_Inhibitor: PHF19 = !ASF1B (Confidence: 1, TimeStep: 1)

PHF19_3_Inhibitor: PHF19 = !AURKA (Confidence: 1, TimeStep: 1)

PHF19_4_Inhibitor: PHF19 = BTG1 (Confidence: 1, TimeStep: 1)

PHF19_5_Inhibitor: PHF19 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PIK3IP1 with decay value = 1:

PIK3IP1_1_Activator: PIK3IP1 = !MCM7 (Confidence: 1, TimeStep: 1)

PIK3IP1_2_Activator: PIK3IP1 = !PAICS (Confidence: 1, TimeStep: 1)

PIK3IP1_3_Activator: PIK3IP1 = DDIT4 (Confidence: 1, TimeStep: 1)

PIK3IP1_4_Activator: PIK3IP1 = FGD2 (Confidence: 1, TimeStep: 1)

PIK3IP1_5_Activator: PIK3IP1 = CCR1 (Confidence: 1, TimeStep: 1)

PIK3IP1_1_Inhibitor: PIK3IP1 = !ZFP36L2 (Confidence: 1, TimeStep: 1)

PIK3IP1_2_Inhibitor: PIK3IP1 = BRCA1&!SMIM3 (Confidence: 1, TimeStep: 1)

PIK3IP1_3_Inhibitor: PIK3IP1 = CENPU&!SMIM3 (Confidence: 1, TimeStep: 1)

PIK3IP1_4_Inhibitor: PIK3IP1 = BRIP1&!SMIM3 (Confidence: 1, TimeStep: 1)

PIK3IP1_5_Inhibitor: PIK3IP1 = CDT1&!SMIM3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PLK4 with decay value = 1:

PLK4_1_Activator: PLK4 = CENPH (Confidence: 1, TimeStep: 1)

PLK4_2_Activator: PLK4 = APITD1-CORT (Confidence: 1, TimeStep: 1)

PLK4_3_Activator: PLK4 = ASF1B (Confidence: 1, TimeStep: 1)

PLK4_4_Activator: PLK4 = AURKA (Confidence: 1, TimeStep: 1)

PLK4_5_Activator: PLK4 = !BTG1 (Confidence: 1, TimeStep: 1)

PLK4_1_Inhibitor: PLK4 = !CENPH (Confidence: 1, TimeStep: 1)

PLK4_2_Inhibitor: PLK4 = !CDK1 (Confidence: 1, TimeStep: 1)

PLK4_3_Inhibitor: PLK4 = !HMMR (Confidence: 1, TimeStep: 1)

PLK4_4_Inhibitor: PLK4 = !KIF14 (Confidence: 1, TimeStep: 1)

PLK4_5_Inhibitor: PLK4 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for POLE2 with decay value = 1:

POLE2_1_Activator: POLE2 = !BMF&MDK (Confidence: 1, TimeStep: 1)

POLE2_2_Activator: POLE2 = CENPV&MDK (Confidence: 1, TimeStep: 1)

POLE2_3_Activator: POLE2 = E2F7&MDK (Confidence: 1, TimeStep: 1)

POLE2_4_Activator: POLE2 = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

POLE2_5_Activator: POLE2 = !IL1B&MDK (Confidence: 1, TimeStep: 1)

POLE2_1_Inhibitor: POLE2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

POLE2_2_Inhibitor: POLE2 = !ASF1B (Confidence: 1, TimeStep: 1)

POLE2_3_Inhibitor: POLE2 = !AURKA (Confidence: 1, TimeStep: 1)

POLE2_4_Inhibitor: POLE2 = BTG1 (Confidence: 1, TimeStep: 1)

POLE2_5_Inhibitor: POLE2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for POLQ with decay value = 1:

POLQ_1_Activator: POLQ = !BMF&MDK (Confidence: 1, TimeStep: 1)

POLQ_2_Activator: POLQ = CENPV&MDK (Confidence: 1, TimeStep: 1)

POLQ_3_Activator: POLQ = E2F7&MDK (Confidence: 1, TimeStep: 1)

POLQ_4_Activator: POLQ = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

POLQ_5_Activator: POLQ = !IL1B&MDK (Confidence: 1, TimeStep: 1)

POLQ_1_Inhibitor: POLQ = !APITD1-CORT (Confidence: 1, TimeStep: 1)

POLQ_2_Inhibitor: POLQ = !ASF1B (Confidence: 1, TimeStep: 1)

POLQ_3_Inhibitor: POLQ = !AURKA (Confidence: 1, TimeStep: 1)

POLQ_4_Inhibitor: POLQ = BTG1 (Confidence: 1, TimeStep: 1)

POLQ_5_Inhibitor: POLQ = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for POU4F1 with decay value = 1:

POU4F1_1_Activator: POU4F1 = !BRIP1&POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_2_Activator: POU4F1 = !BUB1B&POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_3_Activator: POU4F1 = CD53&POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_4_Activator: POU4F1 = !CDT1&POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_5_Activator: POU4F1 = !CENPN&POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_1_Inhibitor: POU4F1 = !POU4F1 (Confidence: 1, TimeStep: 1)

POU4F1_2_Inhibitor: POU4F1 = !ISG20 (Confidence: 1, TimeStep: 1)

POU4F1_3_Inhibitor: POU4F1 = !CD53 (Confidence: 1, TimeStep: 1)

POU4F1_4_Inhibitor: POU4F1 = CDT1 (Confidence: 1, TimeStep: 1)

POU4F1_5_Inhibitor: POU4F1 = ZWINT (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PPBP with decay value = 1:

PPBP_1_Activator: PPBP = CELF2&F13A1 (Confidence: 1, TimeStep: 1)

PPBP_2_Activator: PPBP = !C5orf24&PIK3IP1 (Confidence: 1, TimeStep: 1)

PPBP_3_Activator: PPBP = CCR1&F13A1 (Confidence: 1, TimeStep: 1)

PPBP_4_Activator: PPBP = CDC42EP3&FGD2 (Confidence: 1, TimeStep: 1)

PPBP_5_Activator: PPBP = !CRNDE&F13A1 (Confidence: 1, TimeStep: 1)

PPBP_1_Inhibitor: PPBP = E2F8 (Confidence: 1, TimeStep: 1)

PPBP_2_Inhibitor: PPBP = !GIMAP7 (Confidence: 1, TimeStep: 1)

PPBP_3_Inhibitor: PPBP = !GIMAP4 (Confidence: 1, TimeStep: 1)

PPBP_4_Inhibitor: PPBP = KIF11 (Confidence: 1, TimeStep: 1)

PPBP_5_Inhibitor: PPBP = KNL1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PPIF with decay value = 1:

PPIF_1_Activator: PPIF = APITD1-CORT (Confidence: 1, TimeStep: 1)

PPIF_2_Activator: PPIF = ASF1B (Confidence: 1, TimeStep: 1)

PPIF_3_Activator: PPIF = AURKA (Confidence: 1, TimeStep: 1)

PPIF_4_Activator: PPIF = !BTG1 (Confidence: 1, TimeStep: 1)

PPIF_5_Activator: PPIF = CCDC34 (Confidence: 1, TimeStep: 1)

PPIF_1_Inhibitor: PPIF = !BCL2L11 (Confidence: 1, TimeStep: 1)

PPIF_2_Inhibitor: PPIF = BMF (Confidence: 1, TimeStep: 1)

PPIF_3_Inhibitor: PPIF = DPEP1 (Confidence: 1, TimeStep: 1)

PPIF_4_Inhibitor: PPIF = NPCDR1 (Confidence: 1, TimeStep: 1)

PPIF_5_Inhibitor: PPIF = IL1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PPP1R16B with decay value = 1:

PPP1R16B_1_Activator: PPP1R16B = PPP1R16B (Confidence: 1, TimeStep: 1)

PPP1R16B_2_Activator: PPP1R16B = LGALS3 (Confidence: 1, TimeStep: 1)

PPP1R16B_3_Activator: PPP1R16B = LOC100130872 (Confidence: 1, TimeStep: 1)

PPP1R16B_4_Activator: PPP1R16B = LILRA1 (Confidence: 1, TimeStep: 1)

PPP1R16B_5_Activator: PPP1R16B = !DTL&!PSPH (Confidence: 1, TimeStep: 1)

PPP1R16B_1_Inhibitor: PPP1R16B = RFC3 (Confidence: 1, TimeStep: 1)

PPP1R16B_2_Inhibitor: PPP1R16B = FH (Confidence: 1, TimeStep: 1)

PPP1R16B_3_Inhibitor: PPP1R16B = CCNB1 (Confidence: 1, TimeStep: 1)

PPP1R16B_4_Inhibitor: PPP1R16B = CENPF (Confidence: 1, TimeStep: 1)

PPP1R16B_5_Inhibitor: PPP1R16B = DEPDC1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PRDM1 with decay value = 1:

PRDM1_1_Activator: PRDM1 = PPBP (Confidence: 1, TimeStep: 1)

PRDM1_2_Activator: PRDM1 = FGL2 (Confidence: 1, TimeStep: 1)

PRDM1_3_Activator: PRDM1 = CDC42EP3&FGR (Confidence: 1, TimeStep: 1)

PRDM1_4_Activator: PRDM1 = FCER1G&!RAD51AP1 (Confidence: 1, TimeStep: 1)

PRDM1_5_Activator: PRDM1 = FCGR3B&!RAD51AP1 (Confidence: 1, TimeStep: 1)

PRDM1_1_Inhibitor: PRDM1 = RAD51AP1 (Confidence: 1, TimeStep: 1)

PRDM1_2_Inhibitor: PRDM1 = TOP2A (Confidence: 1, TimeStep: 1)

PRDM1_3_Inhibitor: PRDM1 = DTL (Confidence: 1, TimeStep: 1)

PRDM1_4_Inhibitor: PRDM1 = !ID2 (Confidence: 1, TimeStep: 1)

PRDM1_5_Inhibitor: PRDM1 = MCM7 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PRPS2 with decay value = 1:

PRPS2_1_Activator: PRPS2 = !IFNGR1 (Confidence: 1, TimeStep: 1)

PRPS2_2_Activator: PRPS2 = !SERPINB9 (Confidence: 1, TimeStep: 1)

PRPS2_3_Activator: PRPS2 = CENPV (Confidence: 1, TimeStep: 1)

PRPS2_4_Activator: PRPS2 = !IRAK3 (Confidence: 1, TimeStep: 1)

PRPS2_5_Activator: PRPS2 = !LILRB2 (Confidence: 1, TimeStep: 1)

PRPS2_1_Inhibitor: PRPS2 = !BCAT1&RPS6KA2 (Confidence: 1, TimeStep: 1)

PRPS2_2_Inhibitor: PRPS2 = FGD2&IGH (Confidence: 1, TimeStep: 1)

PRPS2_3_Inhibitor: PRPS2 = FGD2&!PRPS2 (Confidence: 1, TimeStep: 1)

PRPS2_4_Inhibitor: PRPS2 = !BCAT1&BTNL9 (Confidence: 1, TimeStep: 1)

PRPS2_5_Inhibitor: PRPS2 = CCR1&!PRPS2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PRR11 with decay value = 1:

PRR11_1_Activator: PRR11 = APITD1-CORT (Confidence: 1, TimeStep: 1)

PRR11_2_Activator: PRR11 = ASF1B (Confidence: 1, TimeStep: 1)

PRR11_3_Activator: PRR11 = AURKA (Confidence: 1, TimeStep: 1)

PRR11_4_Activator: PRR11 = !BTG1 (Confidence: 1, TimeStep: 1)

PRR11_5_Activator: PRR11 = CCDC34 (Confidence: 1, TimeStep: 1)

PRR11_1_Inhibitor: PRR11 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

PRR11_2_Inhibitor: PRR11 = !ASF1B (Confidence: 1, TimeStep: 1)

PRR11_3_Inhibitor: PRR11 = !AURKA (Confidence: 1, TimeStep: 1)

PRR11_4_Inhibitor: PRR11 = BTG1 (Confidence: 1, TimeStep: 1)

PRR11_5_Inhibitor: PRR11 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PSPH with decay value = 1:

PSPH_1_Activator: PSPH = CKAP2L (Confidence: 1, TimeStep: 1)

PSPH_2_Activator: PSPH = KIF18B (Confidence: 1, TimeStep: 1)

PSPH_3_Activator: PSPH = UBE2T (Confidence: 1, TimeStep: 1)

PSPH_4_Activator: PSPH = ATAD2 (Confidence: 1, TimeStep: 1)

PSPH_5_Activator: PSPH = AURKB (Confidence: 1, TimeStep: 1)

PSPH_1_Inhibitor: PSPH = LGALS3 (Confidence: 1, TimeStep: 1)

PSPH_2_Inhibitor: PSPH = RAB31 (Confidence: 1, TimeStep: 1)

PSPH_3_Inhibitor: PSPH = !ANP32E&PPP1R16B (Confidence: 1, TimeStep: 1)

PSPH_4_Inhibitor: PSPH = !ASPM&!RCC1 (Confidence: 1, TimeStep: 1)

PSPH_5_Inhibitor: PSPH = !ATAD2&PPP1R16B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PTP4A1 with decay value = 1:

PTP4A1_1_Activator: PTP4A1 = KIF4A (Confidence: 1, TimeStep: 1)

PTP4A1_2_Activator: PTP4A1 = RFC3 (Confidence: 1, TimeStep: 1)

PTP4A1_3_Activator: PTP4A1 = FH (Confidence: 1, TimeStep: 1)

PTP4A1_4_Activator: PTP4A1 = CCNB1 (Confidence: 1, TimeStep: 1)

PTP4A1_5_Activator: PTP4A1 = CENPF (Confidence: 1, TimeStep: 1)

PTP4A1_1_Inhibitor: PTP4A1 = !TYMS (Confidence: 1, TimeStep: 1)

PTP4A1_2_Inhibitor: PTP4A1 = PPP1R16B (Confidence: 1, TimeStep: 1)

PTP4A1_3_Inhibitor: PTP4A1 = !PTP4A1 (Confidence: 1, TimeStep: 1)

PTP4A1_4_Inhibitor: PTP4A1 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

PTP4A1_5_Inhibitor: PTP4A1 = LGALS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for PTTG1 with decay value = 1:

PTTG1_1_Activator: PTTG1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

PTTG1_2_Activator: PTTG1 = ASF1B (Confidence: 1, TimeStep: 1)

PTTG1_3_Activator: PTTG1 = AURKA (Confidence: 1, TimeStep: 1)

PTTG1_4_Activator: PTTG1 = !BTG1 (Confidence: 1, TimeStep: 1)

PTTG1_5_Activator: PTTG1 = CCDC34 (Confidence: 1, TimeStep: 1)

PTTG1_1_Inhibitor: PTTG1 = !PTTG1 (Confidence: 1, TimeStep: 1)

PTTG1_2_Inhibitor: PTTG1 = NEAT1 (Confidence: 1, TimeStep: 1)

PTTG1_3_Inhibitor: PTTG1 = GIMAP7 (Confidence: 1, TimeStep: 1)

PTTG1_4_Inhibitor: PTTG1 = IMND1 (Confidence: 1, TimeStep: 1)

PTTG1_5_Inhibitor: PTTG1 = IRAK3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RAB31 with decay value = 1:

RAB31_1_Activator: RAB31 = CDC42EP3&!LEF1 (Confidence: 1, TimeStep: 1)

RAB31_2_Activator: RAB31 = CDC42EP3&ZBTB16 (Confidence: 1, TimeStep: 1)

RAB31_3_Activator: RAB31 = CELF2&LGALS3 (Confidence: 1, TimeStep: 1)

RAB31_4_Activator: RAB31 = !FABP5&ZBTB16 (Confidence: 1, TimeStep: 1)

RAB31_5_Activator: RAB31 = HBG1&!LEF1 (Confidence: 1, TimeStep: 1)

RAB31_1_Inhibitor: RAB31 = CRNDE (Confidence: 1, TimeStep: 1)

RAB31_2_Inhibitor: RAB31 = !KLF9 (Confidence: 1, TimeStep: 1)

RAB31_3_Inhibitor: RAB31 = RCC1 (Confidence: 1, TimeStep: 1)

RAB31_4_Inhibitor: RAB31 = !IFNGR1 (Confidence: 1, TimeStep: 1)

RAB31_5_Inhibitor: RAB31 = LOC100996643 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RAD51 with decay value = 1:

RAD51_1_Activator: RAD51 = BUB1B (Confidence: 1, TimeStep: 1)

RAD51_2_Activator: RAD51 = CENPN (Confidence: 1, TimeStep: 1)

RAD51_3_Activator: RAD51 = KIF15 (Confidence: 1, TimeStep: 1)

RAD51_4_Activator: RAD51 = MCM10 (Confidence: 1, TimeStep: 1)

RAD51_5_Activator: RAD51 = PLK4 (Confidence: 1, TimeStep: 1)

RAD51_1_Inhibitor: RAD51 = !BUB1B (Confidence: 1, TimeStep: 1)

RAD51_2_Inhibitor: RAD51 = !CENPN (Confidence: 1, TimeStep: 1)

RAD51_3_Inhibitor: RAD51 = !KIF15 (Confidence: 1, TimeStep: 1)

RAD51_4_Inhibitor: RAD51 = !MCM10 (Confidence: 1, TimeStep: 1)

RAD51_5_Inhibitor: RAD51 = !PLK4 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RAD51AP1 with decay value = 1:

RAD51AP1_1_Activator: RAD51AP1 = CKAP2L (Confidence: 1, TimeStep: 1)

RAD51AP1_2_Activator: RAD51AP1 = KIF18B (Confidence: 1, TimeStep: 1)

RAD51AP1_3_Activator: RAD51AP1 = UBE2T (Confidence: 1, TimeStep: 1)

RAD51AP1_4_Activator: RAD51AP1 = ATAD2 (Confidence: 1, TimeStep: 1)

RAD51AP1_5_Activator: RAD51AP1 = AURKB (Confidence: 1, TimeStep: 1)

RAD51AP1_1_Inhibitor: RAD51AP1 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

RAD51AP1_2_Inhibitor: RAD51AP1 = !BYSL (Confidence: 1, TimeStep: 1)

RAD51AP1_3_Inhibitor: RAD51AP1 = !PAICS (Confidence: 1, TimeStep: 1)

RAD51AP1_4_Inhibitor: RAD51AP1 = !SELENOI (Confidence: 1, TimeStep: 1)

RAD51AP1_5_Inhibitor: RAD51AP1 = CCR1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RAG1 with decay value = 1:

RAG1_1_Activator: RAG1 = RAG1 (Confidence: 1, TimeStep: 1)

RAG1_2_Activator: RAG1 = ARPP21 (Confidence: 1, TimeStep: 1)

RAG1_3_Activator: RAG1 = IL1B (Confidence: 1, TimeStep: 1)

RAG1_4_Activator: RAG1 = KIF26A (Confidence: 1, TimeStep: 1)

RAG1_5_Activator: RAG1 = LOC285097 (Confidence: 1, TimeStep: 1)

RAG1_1_Inhibitor: RAG1 = !BRCA1&!RAG1 (Confidence: 1, TimeStep: 1)

RAG1_2_Inhibitor: RAG1 = !LOC100996643&!RAG1 (Confidence: 1, TimeStep: 1)

RAG1_3_Inhibitor: RAG1 = !AKAP12&!PAICS (Confidence: 1, TimeStep: 1)

RAG1_4_Inhibitor: RAG1 = !BYSL&!RAG1 (Confidence: 1, TimeStep: 1)

RAG1_5_Inhibitor: RAG1 = !C5orf24&!RAG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RAG2 with decay value = 1:

RAG2_1_Activator: RAG2 = !CELF2&RAG2 (Confidence: 1, TimeStep: 1)

RAG2_2_Activator: RAG2 = !CELF2&!IL27RA (Confidence: 1, TimeStep: 1)

RAG2_3_Activator: RAG2 = !FUS&!IL27RA (Confidence: 1, TimeStep: 1)

RAG2_4_Activator: RAG2 = !TGB2-AS1&!LOC100130872 (Confidence: 1, TimeStep: 1)

RAG2_5_Activator: RAG2 = C5orf24&CCR1&RAG2 (Confidence: 1, TimeStep: 1)

RAG2_1_Inhibitor: RAG2 = !IGLL1 (Confidence: 1, TimeStep: 1)

RAG2_2_Inhibitor: RAG2 = LOC100130872 (Confidence: 1, TimeStep: 1)

RAG2_3_Inhibitor: RAG2 = IL27RA&!RAG2 (Confidence: 1, TimeStep: 1)

RAG2_4_Inhibitor: RAG2 = !BCAT1&!RAG2 (Confidence: 1, TimeStep: 1)

RAG2_5_Inhibitor: RAG2 = !AKAP12&!BCAT1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RASSF4 with decay value = 1:

RASSF4_1_Activator: RASSF4 = F13A1 (Confidence: 1, TimeStep: 1)

RASSF4_2_Activator: RASSF4 = LGALS3 (Confidence: 1, TimeStep: 1)

RASSF4_3_Activator: RASSF4 = MNDA (Confidence: 1, TimeStep: 1)

RASSF4_4_Activator: RASSF4 = !CDT1&MIR8071-1 (Confidence: 1, TimeStep: 1)

RASSF4_5_Activator: RASSF4 = !CENPU&MIR8071-1 (Confidence: 1, TimeStep: 1)

RASSF4_1_Inhibitor: RASSF4 = BCL10 (Confidence: 1, TimeStep: 1)

RASSF4_2_Inhibitor: RASSF4 = CLEC2B&!MIR8071-1 (Confidence: 1, TimeStep: 1)

RASSF4_3_Inhibitor: RASSF4 = CRNDE&!TARSL2 (Confidence: 1, TimeStep: 2)

RASSF4_4_Inhibitor: RASSF4 = !CENPU&!MIR8071-1 (Confidence: 1, TimeStep: 2)

RASSF4_5_Inhibitor: RASSF4 = !BCL10&!MIR8071-1 (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for RBM14 with decay value = 1:

RBM14_1_Activator: RBM14 = UBE2C (Confidence: 1, TimeStep: 1)

RBM14_2_Activator: RBM14 = MCM5&NME1 (Confidence: 1, TimeStep: 1)

RBM14_3_Activator: RBM14 = CCDC86&MCM5 (Confidence: 1, TimeStep: 1)

RBM14_4_Activator: RBM14 = !CELF2&MCM5 (Confidence: 1, TimeStep: 1)

RBM14_5_Activator: RBM14 = CEP55&MCM5 (Confidence: 1, TimeStep: 1)

RBM14_1_Inhibitor: RBM14 = CDC42EP3 (Confidence: 1, TimeStep: 1)

RBM14_2_Inhibitor: RBM14 = !DHX9 (Confidence: 1, TimeStep: 1)

RBM14_3_Inhibitor: RBM14 = !PTP4A1 (Confidence: 1, TimeStep: 1)

RBM14_4_Inhibitor: RBM14 = !FABP5 (Confidence: 1, TimeStep: 1)

RBM14_5_Inhibitor: RBM14 = !HSP90AB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RBMS3 with decay value = 1:

RBMS3_1_Activator: RBMS3 = !PRPS2&!S100A11 (Confidence: 1, TimeStep: 1)

RBMS3_2_Activator: RBMS3 = DDIT4&SNORD3B-1 (Confidence: 1, TimeStep: 1)

RBMS3_3_Activator: RBMS3 = !E2F7&TMEM100 (Confidence: 1, TimeStep: 1)

RBMS3_4_Activator: RBMS3 = LILRB2&TMEM100 (Confidence: 1, TimeStep: 1)

RBMS3_5_Activator: RBMS3 = MIR6845&TMEM100 (Confidence: 1, TimeStep: 1)

RBMS3_1_Inhibitor: RBMS3 = !GSN (Confidence: 1, TimeStep: 1)

RBMS3_2_Inhibitor: RBMS3 = CENPV (Confidence: 1, TimeStep: 1)

RBMS3_3_Inhibitor: RBMS3 = !LILRB2 (Confidence: 1, TimeStep: 1)

RBMS3_4_Inhibitor: RBMS3 = ECT2 (Confidence: 1, TimeStep: 1)

RBMS3_5_Inhibitor: RBMS3 = !ID3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RCC1 with decay value = 1:

RCC1_1_Activator: RCC1 = FOXM1 (Confidence: 1, TimeStep: 1)

RCC1_2_Activator: RCC1 = STIL (Confidence: 1, TimeStep: 1)

RCC1_3_Activator: RCC1 = ATAD2 (Confidence: 1, TimeStep: 1)

RCC1_4_Activator: RCC1 = BIRC5 (Confidence: 1, TimeStep: 1)

RCC1_5_Activator: RCC1 = BUB1 (Confidence: 1, TimeStep: 1)

RCC1_1_Inhibitor: RCC1 = IL18R1 (Confidence: 1, TimeStep: 1)

RCC1_2_Inhibitor: RCC1 = RPS6KA2 (Confidence: 1, TimeStep: 1)

RCC1_3_Inhibitor: RCC1 = PRDM1 (Confidence: 1, TimeStep: 1)

RCC1_4_Inhibitor: RCC1 = IL18RAP (Confidence: 1, TimeStep: 1)

RCC1_5_Inhibitor: RCC1 = LGALS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RFC3 with decay value = 1:

RFC3_1_Activator: RFC3 = CENPH (Confidence: 1, TimeStep: 1)

RFC3_2_Activator: RFC3 = APITD1-CORT (Confidence: 1, TimeStep: 1)

RFC3_3_Activator: RFC3 = ASF1B (Confidence: 1, TimeStep: 1)

RFC3_4_Activator: RFC3 = AURKA (Confidence: 1, TimeStep: 1)

RFC3_5_Activator: RFC3 = !BTG1 (Confidence: 1, TimeStep: 1)

RFC3_1_Inhibitor: RFC3 = !FH (Confidence: 1, TimeStep: 1)

RFC3_2_Inhibitor: RFC3 = !NUSAP1 (Confidence: 1, TimeStep: 1)

RFC3_3_Inhibitor: RFC3 = !RFC3 (Confidence: 1, TimeStep: 1)

RFC3_4_Inhibitor: RFC3 = !NUF2 (Confidence: 1, TimeStep: 1)

RFC3_5_Inhibitor: RFC3 = !KIF11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RHOBTB3 with decay value = 1:

RHOBTB3_1_Activator: RHOBTB3 = DEPDC1B (Confidence: 1, TimeStep: 1)

RHOBTB3_2_Activator: RHOBTB3 = PTTG1 (Confidence: 1, TimeStep: 1)

RHOBTB3_3_Activator: RHOBTB3 = !TXNIP (Confidence: 1, TimeStep: 1)

RHOBTB3_4_Activator: RHOBTB3 = AURKB (Confidence: 1, TimeStep: 1)
RHOBTB3_5_Activator: RHOBTB3 = CKAP2 (Confidence: 1, TimeStep: 1)
RHOBTB3_1_Inhibitor: RHOBTB3 = !BCAT1&EMP1 (Confidence: 1, TimeStep: 1)
RHOBTB3_2_Inhibitor: RHOBTB3 = B3GNT2&F13A1 (Confidence: 1, TimeStep: 1)
RHOBTB3_3_Inhibitor: RHOBTB3 = !CCR1&F13A1 (Confidence: 1, TimeStep: 1)
RHOBTB3_4_Inhibitor: RHOBTB3 = BCAT1&F13A1 (Confidence: 1, TimeStep: 1)
RHOBTB3_5_Inhibitor: RHOBTB3 = !AURKB&EMP1&!RHOBTB3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RMI2 with decay value = 1:

RMI2_1_Activator: RMI2 = DFNA5&!P2RX5 (Confidence: 1, TimeStep: 1)
RMI2_2_Activator: RMI2 = E2F8&!P2RX5 (Confidence: 1, TimeStep: 1)
RMI2_3_Activator: RMI2 = CENPV&!P2RX5 (Confidence: 1, TimeStep: 1)
RMI2_4_Activator: RMI2 = !LILRB2&!P2RX5 (Confidence: 1, TimeStep: 1)
RMI2_5_Activator: RMI2 = !CD53&!P2RX5 (Confidence: 1, TimeStep: 1)
RMI2_1_Inhibitor: RMI2 = !FABP5 (Confidence: 1, TimeStep: 1)
RMI2_2_Inhibitor: RMI2 = IL18RAP (Confidence: 1, TimeStep: 1)
RMI2_3_Inhibitor: RMI2 = LGALS3 (Confidence: 1, TimeStep: 1)
RMI2_4_Inhibitor: RMI2 = RAB31 (Confidence: 1, TimeStep: 1)
RMI2_5_Inhibitor: RMI2 = !DFNA5&!DTL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RNASET2 with decay value = 1:

RNASET2_1_Activator: RNASET2 = LILRB2 (Confidence: 1, TimeStep: 1)
RNASET2_2_Activator: RNASET2 = RNASET2 (Confidence: 1, TimeStep: 1)
RNASET2_3_Activator: RNASET2 = !CENPV (Confidence: 1, TimeStep: 1)
RNASET2_4_Activator: RNASET2 = IRAK3 (Confidence: 1, TimeStep: 1)
RNASET2_5_Activator: RNASET2 = GSN (Confidence: 1, TimeStep: 1)
RNASET2_1_Inhibitor: RNASET2 = UBE2C (Confidence: 1, TimeStep: 1)
RNASET2_2_Inhibitor: RNASET2 = !CD53&!KIF26A (Confidence: 1, TimeStep: 1)
RNASET2_3_Inhibitor: RNASET2 = DFNA5&!GSN (Confidence: 1, TimeStep: 1)
RNASET2_4_Inhibitor: RNASET2 = DFNA5&!KIF26A (Confidence: 1, TimeStep: 1)
RNASET2_5_Inhibitor: RNASET2 = DTL&!GSN (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RPS6KA2 with decay value = 1:

RPS6KA2_1_Activator: RPS6KA2 = RPS6KA2 (Confidence: 1, TimeStep: 1)

RPS6KA2_2_Activator: RPS6KA2 = AKAP12&RBMS3 (Confidence: 1, TimeStep: 1)

RPS6KA2_3_Activator: RPS6KA2 = ID3&!PRPS2 (Confidence: 1, TimeStep: 1)

RPS6KA2_4_Activator: RPS6KA2 = RBMS3&SERPINB9 (Confidence: 1, TimeStep: 1)

RPS6KA2_5_Activator: RPS6KA2 = AKAP12&!PRPS2 (Confidence: 1, TimeStep: 1)

RPS6KA2_1_Inhibitor: RPS6KA2 = !AKAP12&!PFKFB2 (Confidence: 1, TimeStep: 1)

RPS6KA2_2_Inhibitor: RPS6KA2 = !GSN&!PFKFB2 (Confidence: 1, TimeStep: 1)

RPS6KA2_3_Inhibitor: RPS6KA2 = !MSH6&!PFKFB2 (Confidence: 1, TimeStep: 1)

RPS6KA2_4_Inhibitor: RPS6KA2 = !LOC285097&S100A11 (Confidence: 1, TimeStep: 1)

RPS6KA2_5_Inhibitor: RPS6KA2 = !PFKFB2&S100A11 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for RRM2 with decay value = 1:

RRM2_1_Activator: RRM2 = CKAP2L (Confidence: 1, TimeStep: 1)

RRM2_2_Activator: RRM2 = KIF18B (Confidence: 1, TimeStep: 1)

RRM2_3_Activator: RRM2 = UBE2T (Confidence: 1, TimeStep: 1)

RRM2_4_Activator: RRM2 = ATAD2 (Confidence: 1, TimeStep: 1)

RRM2_5_Activator: RRM2 = AURKB (Confidence: 1, TimeStep: 1)

RRM2_1_Inhibitor: RRM2 = !RRM2 (Confidence: 1, TimeStep: 1)

RRM2_2_Inhibitor: RRM2 = !CENPW (Confidence: 1, TimeStep: 1)

RRM2_3_Inhibitor: RRM2 = !TCF19 (Confidence: 1, TimeStep: 1)

RRM2_4_Inhibitor: RRM2 = !WDR76 (Confidence: 1, TimeStep: 1)

RRM2_5_Inhibitor: RRM2 = !TK1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for S100A11 with decay value = 1:

S100A11_1_Activator: S100A11 = !WASF1 (Confidence: 1, TimeStep: 1)

S100A11_2_Activator: S100A11 = LILRA1 (Confidence: 1, TimeStep: 1)

S100A11_3_Activator: S100A11 = UBE2C (Confidence: 1, TimeStep: 1)

S100A11_4_Activator: S100A11 = FCER1G&!POU4F1 (Confidence: 1, TimeStep: 1)

S100A11_5_Activator: S100A11 = SLA&TUBA4A (Confidence: 1, TimeStep: 1)

S100A11_1_Inhibitor: S100A11 = !KIF26A&MSH6 (Confidence: 1, TimeStep: 1)

S100A11_2_Inhibitor: S100A11 = !HRK&MSH6 (Confidence: 1, TimeStep: 1)

S100A11_3_Inhibitor: S100A11 = !IL1B&MSH6 (Confidence: 1, TimeStep: 1)

S100A11_4_Inhibitor: S100A11 = !SIK1&!SLA (Confidence: 1, TimeStep: 1)

S100A11_5_Inhibitor: S100A11 = !HRK&!SNX10 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for S100A8 with decay value = 1:

S100A8_1_Activator: S100A8 = F13A1 (Confidence: 1, TimeStep: 1)

S100A8_2_Activator: S100A8 = !BCAT1&!KIF11 (Confidence: 1, TimeStep: 1)

S100A8_3_Activator: S100A8 = !BCAT1&S100A8 (Confidence: 1, TimeStep: 1)

S100A8_4_Activator: S100A8 = !BCAT1&LYZ (Confidence: 1, TimeStep: 1)

S100A8_5_Activator: S100A8 = !CCNB1&IGH (Confidence: 1, TimeStep: 1)

S100A8_1_Inhibitor: S100A8 = TMEM97 (Confidence: 1, TimeStep: 1)

S100A8_2_Inhibitor: S100A8 = WDHD1 (Confidence: 1, TimeStep: 1)

S100A8_3_Inhibitor: S100A8 = CENPH (Confidence: 1, TimeStep: 1)

S100A8_4_Inhibitor: S100A8 = APITD1-CORT (Confidence: 1, TimeStep: 1)

S100A8_5_Inhibitor: S100A8 = ASF1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SCML4 with decay value = 1:

SCML4_1_Activator: SCML4 = F13A1 (Confidence: 1, TimeStep: 1)

SCML4_2_Activator: SCML4 = MNDA (Confidence: 1, TimeStep: 1)

SCML4_3_Activator: SCML4 = !BRIP1&SCML4 (Confidence: 1, TimeStep: 1)

SCML4_4_Activator: SCML4 = !BUB1B&SCML4 (Confidence: 1, TimeStep: 1)

SCML4_5_Activator: SCML4 = !C4orf46&SCML4 (Confidence: 1, TimeStep: 1)

SCML4_1_Inhibitor: SCML4 = ECT2 (Confidence: 1, TimeStep: 1)

SCML4_2_Inhibitor: SCML4 = FOXM1 (Confidence: 1, TimeStep: 1)

SCML4_3_Inhibitor: SCML4 = STIL (Confidence: 1, TimeStep: 1)

SCML4_4_Inhibitor: SCML4 = ATAD2 (Confidence: 1, TimeStep: 1)

SCML4_5_Inhibitor: SCML4 = BIRC5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SELENOI with decay value = 1:

SELENOI_1_Activator: SELENOI = !TXNIP (Confidence: 1, TimeStep: 1)

SELENOI_2_Activator: SELENOI = CENPH (Confidence: 1, TimeStep: 1)

SELENOI_3_Activator: SELENOI = CKAP2 (Confidence: 1, TimeStep: 1)

SELENOI_4_Activator: SELENOI = APITD1-CORT (Confidence: 1, TimeStep: 1)

SELENOI_5_Activator: SELENOI = ASF1B (Confidence: 1, TimeStep: 1)

SELENOI_1_Inhibitor: SELENOI = !LOC100996643 (Confidence: 1, TimeStep: 1)

SELENOI_2_Inhibitor: SELENOI = !RAD51AP1 (Confidence: 1, TimeStep: 1)

SELENOI_3_Inhibitor: SELENOI = !BYSL (Confidence: 1, TimeStep: 1)

SELENOI_4_Inhibitor: SELENOI = !SELENOI (Confidence: 1, TimeStep: 1)

SELENOI_5_Inhibitor: SELENOI = FGD2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SEMA4D with decay value = 1:

SEMA4D_1_Activator: SEMA4D = SEMA4D (Confidence: 1, TimeStep: 1)

SEMA4D_2_Activator: SEMA4D = !B3GNT2&FGR (Confidence: 1, TimeStep: 1)

SEMA4D_3_Activator: SEMA4D = !C5orf24&!PTP4A1 (Confidence: 1, TimeStep: 1)

SEMA4D_4_Activator: SEMA4D = !DHX9&TNFSF8 (Confidence: 1, TimeStep: 1)

SEMA4D_5_Activator: SEMA4D = !FABP5&SLA (Confidence: 1, TimeStep: 1)

SEMA4D_1_Inhibitor: SEMA4D = !BIRC3&!HBG1 (Confidence: 1, TimeStep: 1)

SEMA4D_2_Inhibitor: SEMA4D = ABHD17B&ASPM (Confidence: 1, TimeStep: 1)

SEMA4D_3_Inhibitor: SEMA4D = RBM14&!TNFSF8 (Confidence: 1, TimeStep: 1)

SEMA4D_4_Inhibitor: SEMA4D = ABHD17B&KIF11 (Confidence: 1, TimeStep: 1)

SEMA4D_5_Inhibitor: SEMA4D = !BIRC3&PAICS (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SERPINA1 with decay value = 1:

SERPINA1_1_Activator: SERPINA1 = PPBP (Confidence: 1, TimeStep: 1)

SERPINA1_2_Activator: SERPINA1 = FGL2 (Confidence: 1, TimeStep: 1)

SERPINA1_3_Activator: SERPINA1 = !CCDC86&!PRPS2 (Confidence: 1, TimeStep: 1)

SERPINA1_4_Activator: SERPINA1 = !CCDC86&SERPINA1 (Confidence: 1, TimeStep: 1)

SERPINA1_5_Activator: SERPINA1 = !E2F7&FCGR3B (Confidence: 1, TimeStep: 1)

SERPINA1_1_Inhibitor: SERPINA1 = APITD1-CORT (Confidence: 1, TimeStep: 1)

SERPINA1_2_Inhibitor: SERPINA1 = ASF1B (Confidence: 1, TimeStep: 1)

SERPINA1_3_Inhibitor: SERPINA1 = AURKA (Confidence: 1, TimeStep: 1)

SERPINA1_4_Inhibitor: SERPINA1 = !BTG1 (Confidence: 1, TimeStep: 1)

SERPINA1_5_Inhibitor: SERPINA1 = CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SERPINB9 with decay value = 1:

SERPINB9_1_Activator: SERPINB9 = SERPINB9 (Confidence: 1, TimeStep: 1)

SERPINB9_2_Activator: SERPINB9 = ITGAM (Confidence: 1, TimeStep: 1)

SERPINB9_3_Activator: SERPINB9 = CCR1 (Confidence: 1, TimeStep: 1)

SERPINB9_4_Activator: SERPINB9 = RPS6KA2 (Confidence: 1, TimeStep: 1)

SERPINB9_5_Activator: SERPINB9 = F13A1 (Confidence: 1, TimeStep: 1)

SERPINB9_1_Inhibitor: SERPINB9 = !LILRB2 (Confidence: 1, TimeStep: 1)

SERPINB9_2_Inhibitor: SERPINB9 = E2F7 (Confidence: 1, TimeStep: 1)

SERPINB9_3_Inhibitor: SERPINB9 = !SMAP2 (Confidence: 1, TimeStep: 1)

SERPINB9_4_Inhibitor: SERPINB9 = !TMEM2 (Confidence: 1, TimeStep: 1)

SERPINB9_5_Inhibitor: SERPINB9 = ANP32E (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SESN1 with decay value = 1:

SESN1_1_Activator: SESN1 = PPBP (Confidence: 1, TimeStep: 1)

SESN1_2_Activator: SESN1 = !CDC20&!LOC100505650 (Confidence: 1, TimeStep: 1)

SESN1_3_Activator: SESN1 = !ASPM&!ECT2 (Confidence: 1, TimeStep: 1)

SESN1_4_Activator: SESN1 = !ASPM&METTL7A (Confidence: 1, TimeStep: 1)

SESN1_5_Activator: SESN1 = !ASPM&!CENPK (Confidence: 1, TimeStep: 1)

SESN1_1_Inhibitor: SESN1 = CDT1&!FKBP5 (Confidence: 1, TimeStep: 1)

SESN1_2_Inhibitor: SESN1 = CRNDE&TENM4 (Confidence: 1, TimeStep: 1)

SESN1_3_Inhibitor: SESN1 = !DDIT4&LOC100505650 (Confidence: 1, TimeStep: 1)

SESN1_4_Inhibitor: SESN1 = !CCNB1&ECT2 (Confidence: 1, TimeStep: 1)

SESN1_5_Inhibitor: SESN1 = !CCNB1&!METTL7A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SHCBP1 with decay value = 1:

SHCBP1_1_Activator: SHCBP1 = CDK1 (Confidence: 1, TimeStep: 1)

SHCBP1_2_Activator: SHCBP1 = HMMR (Confidence: 1, TimeStep: 1)

SHCBP1_3_Activator: SHCBP1 = KIF14 (Confidence: 1, TimeStep: 1)

SHCBP1_4_Activator: SHCBP1 = KIF20A (Confidence: 1, TimeStep: 1)

SHCBP1_5_Activator: SHCBP1 = POLQ (Confidence: 1, TimeStep: 1)

SHCBP1_1_Inhibitor: SHCBP1 = !CDK1 (Confidence: 1, TimeStep: 1)

SHCBP1_2_Inhibitor: SHCBP1 = !HMMR (Confidence: 1, TimeStep: 1)

SHCBP1_3_Inhibitor: SHCBP1 = !KIF14 (Confidence: 1, TimeStep: 1)

SHCBP1_4_Inhibitor: SHCBP1 = !KIF20A (Confidence: 1, TimeStep: 1)

SHCBP1_5_Inhibitor: SHCBP1 = !POLQ (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SIK1 with decay value = 1:

SIK1_1_Activator: SIK1 = KIF26A (Confidence: 1, TimeStep: 1)

SIK1_2_Activator: SIK1 = MIR4683 (Confidence: 1, TimeStep: 1)

SIK1_3_Activator: SIK1 = CPM (Confidence: 1, TimeStep: 1)

SIK1_4_Activator: SIK1 = POU4F1 (Confidence: 1, TimeStep: 1)

SIK1_5_Activator: SIK1 = RPS6KA2 (Confidence: 1, TimeStep: 1)

SIK1_1_Inhibitor: SIK1 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

SIK1_2_Inhibitor: SIK1 = LOC100130872 (Confidence: 1, TimeStep: 1)

SIK1_3_Inhibitor: SIK1 = !METTL7A (Confidence: 1, TimeStep: 1)

SIK1_4_Inhibitor: SIK1 = SEMA4D (Confidence: 1, TimeStep: 1)

SIK1_5_Inhibitor: SIK1 = !WASF1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SKP2 with decay value = 1:

SKP2_1_Activator: SKP2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

SKP2_2_Activator: SKP2 = ASF1B (Confidence: 1, TimeStep: 1)

SKP2_3_Activator: SKP2 = AURKA (Confidence: 1, TimeStep: 1)

SKP2_4_Activator: SKP2 = !BTG1 (Confidence: 1, TimeStep: 1)

SKP2_5_Activator: SKP2 = CCDC34 (Confidence: 1, TimeStep: 1)

SKP2_1_Inhibitor: SKP2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

SKP2_2_Inhibitor: SKP2 = !ASF1B (Confidence: 1, TimeStep: 1)

SKP2_3_Inhibitor: SKP2 = !AURKA (Confidence: 1, TimeStep: 1)

SKP2_4_Inhibitor: SKP2 = BTG1 (Confidence: 1, TimeStep: 1)

SKP2_5_Inhibitor: SKP2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SLA with decay value = 1:

SLA_1_Activator: SLA = !AKAP12 (Confidence: 1, TimeStep: 1)

SLA_2_Activator: SLA = !MSH6 (Confidence: 1, TimeStep: 1)

SLA_3_Activator: SLA = ITPKB (Confidence: 1, TimeStep: 1)

SLA_4_Activator: SLA = S100A11 (Confidence: 1, TimeStep: 1)

SLA_5_Activator: SLA = TNFSF8 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SLC22A23 with decay value = 1:

SLC22A23_1_Activator: SLC22A23 = UBE2C (Confidence: 1, TimeStep: 1)

SLC22A23_2_Activator: SLC22A23 = !SCML4&WFS1 (Confidence: 1, TimeStep: 1)

SLC22A23_3_Activator: SLC22A23 = !FCER1G&SLC22A23 (Confidence: 1, TimeStep: 1)

SLC22A23_4_Activator: SLC22A23 = !FCGR3B&SLC22A23 (Confidence: 1, TimeStep: 1)

SLC22A23_5_Activator: SLC22A23 = !FGD2&SLC22A23 (Confidence: 1, TimeStep: 1)

SLC22A23_1_Inhibitor: SLC22A23 = !CENPK&FGD2 (Confidence: 1, TimeStep: 1)

SLC22A23_2_Inhibitor: SLC22A23 = TMEM2&!WFS1 (Confidence: 1, TimeStep: 1)

SLC22A23_3_Inhibitor: SLC22A23 = !MIR8071-1&SERPINA1 (Confidence: 1, TimeStep: 1)

SLC22A23_4_Inhibitor: SLC22A23 = !SCML4&SERPINA1 (Confidence: 1, TimeStep: 1)

SLC22A23_5_Inhibitor: SLC22A23 = ECT2&MS4A1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SMAP2 with decay value = 1:

SMAP2_1_Activator: SMAP2 = !CENPU (Confidence: 1, TimeStep: 1)

SMAP2_2_Activator: SMAP2 = !HIST4H4 (Confidence: 1, TimeStep: 1)

SMAP2_3_Activator: SMAP2 = !CKS1B (Confidence: 1, TimeStep: 1)

SMAP2_4_Activator: SMAP2 = GIMAP4 (Confidence: 1, TimeStep: 1)

SMAP2_5_Activator: SMAP2 = LILRB2 (Confidence: 1, TimeStep: 1)

SMAP2_1_Inhibitor: SMAP2 = HIST4H4&!IFNGR1 (Confidence: 1, TimeStep: 1)

SMAP2_2_Inhibitor: SMAP2 = HIST4H4&!LILRB2 (Confidence: 1, TimeStep: 1)

SMAP2_3_Inhibitor: SMAP2 = HIST4H4&!MIR4683 (Confidence: 1, TimeStep: 1)

SMAP2_4_Inhibitor: SMAP2 = HIST4H4&!RNASET2 (Confidence: 1, TimeStep: 1)

SMAP2_5_Inhibitor: SMAP2 = ANP32E&HIST4H4 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SMC2 with decay value = 1:

SMC2_1_Activator: SMC2 = CENPH (Confidence: 1, TimeStep: 1)

SMC2_2_Activator: SMC2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

SMC2_3_Activator: SMC2 = ASF1B (Confidence: 1, TimeStep: 1)

SMC2_4_Activator: SMC2 = AURKA (Confidence: 1, TimeStep: 1)

SMC2_5_Activator: SMC2 = !BTG1 (Confidence: 1, TimeStep: 1)

SMC2_1_Inhibitor: SMC2 = !CENPH (Confidence: 1, TimeStep: 1)

SMC2_2_Inhibitor: SMC2 = !CDK1 (Confidence: 1, TimeStep: 1)

SMC2_3_Inhibitor: SMC2 = !HMMR (Confidence: 1, TimeStep: 1)

SMC2_4_Inhibitor: SMC2 = !KIF14 (Confidence: 1, TimeStep: 1)

SMC2_5_Inhibitor: SMC2 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SMIM3 with decay value = 1:

SMIM3_1_Activator: SMIM3 = GSN (Confidence: 1, TimeStep: 1)

SMIM3_2_Activator: SMIM3 = P2RY14 (Confidence: 1, TimeStep: 1)

SMIM3_3_Activator: SMIM3 = DDIT4 (Confidence: 1, TimeStep: 1)

SMIM3_4_Activator: SMIM3 = TBXA2R (Confidence: 1, TimeStep: 1)

SMIM3_5_Activator: SMIM3 = MIR4683 (Confidence: 1, TimeStep: 1)

SMIM3_1_Inhibitor: SMIM3 = BMF&E2F7 (Confidence: 1, TimeStep: 1)

SMIM3_2_Inhibitor: SMIM3 = !CENPU&DEPDC1B (Confidence: 1, TimeStep: 1)

SMIM3_3_Inhibitor: SMIM3 = !CEP55&DEPDC1B (Confidence: 1, TimeStep: 1)

SMIM3_4_Inhibitor: SMIM3 = !DEPDC1B&FOXM1 (Confidence: 1, TimeStep: 1)

SMIM3_5_Inhibitor: SMIM3 = !DEPDC1B&STIL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SNORA21 with decay value = 1:

SNORA21_1_Activator: SNORA21 = ELL2&!NEAT1 (Confidence: 1, TimeStep: 1)

SNORA21_2_Activator: SNORA21 = !CDC42EP3&LOC728175 (Confidence: 1, TimeStep: 1)

SNORA21_3_Activator: SNORA21 = ELL2&FH (Confidence: 1, TimeStep: 1)

SNORA21_4_Activator: SNORA21 = ELL2&PTTG1 (Confidence: 1, TimeStep: 1)

SNORA21_5_Activator: SNORA21 = ELL2&RFC3 (Confidence: 1, TimeStep: 1)

SNORA21_1_Inhibitor: SNORA21 = !CRNDE (Confidence: 1, TimeStep: 1)

SNORA21_2_Inhibitor: SNORA21 = CDC42EP3 (Confidence: 1, TimeStep: 1)

SNORA21_3_Inhibitor: SNORA21 = !CDC20&IRAK3 (Confidence: 1, TimeStep: 1)

SNORA21_4_Inhibitor: SNORA21 = IRAK3&!KIF4A (Confidence: 1, TimeStep: 1)

SNORA21_5_Inhibitor: SNORA21 = IRAK3&!SHCBP1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SNORD3B-1 with decay value = 1:

SNORD3B-1_1_Inhibitor: SNORD3B-1 = !RNASET2 (Confidence: 1, TimeStep: 1)

SNORD3B-1_2_Inhibitor: SNORD3B-1 = !CD53 (Confidence: 1, TimeStep: 1)

SNORD3B-1_3_Inhibitor: SNORD3B-1 = !P2RX5&!SNORD3B-1 (Confidence: 1, TimeStep: 1)

SNORD3B-1_4_Inhibitor: SNORD3B-1 = !P2RX5&!P2RY14 (Confidence: 1, TimeStep: 1)

SNORD3B-1_5_Inhibitor: SNORD3B-1 = !P2RX5&!TBXA2R (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SNTB2 with decay value = 1:

SNTB2_1_Activator: SNTB2 = !EMP1&SNTB2 (Confidence: 1, TimeStep: 1)

SNTB2_2_Activator: SNTB2 = !DDIT4&SNTB2 (Confidence: 1, TimeStep: 1)

SNTB2_3_Activator: SNTB2 = !P2RY14&SNTB2 (Confidence: 1, TimeStep: 1)

SNTB2_4_Activator: SNTB2 = CDK6&TCF19 (Confidence: 1, TimeStep: 1)

SNTB2_5_Activator: SNTB2 = KCNK12&!RBMS3 (Confidence: 1, TimeStep: 1)

SNTB2_1_Inhibitor: SNTB2 = !DDIT4&!HBB (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for SNX10 with decay value = 1:

SNX10_1_Activator: SNX10 = !BIRC3&SNX10 (Confidence: 1, TimeStep: 1)

SNX10_2_Activator: SNX10 = !BIRC3&!RCC1 (Confidence: 1, TimeStep: 1)

SNX10_3_Activator: SNX10 = MYRIP&!PRPS2 (Confidence: 1, TimeStep: 1)

SNX10_4_Activator: SNX10 = MYRIP&!RCC1 (Confidence: 1, TimeStep: 1)

SNX10_5_Activator: SNX10 = MYRIP&SNX10 (Confidence: 1, TimeStep: 1)

SNX10_1_Inhibitor: SNX10 = RCC1&!SNX10 (Confidence: 1, TimeStep: 1)

SNX10_2_Inhibitor: SNX10 = !ITPKB&LOC728175 (Confidence: 1, TimeStep: 1)

SNX10_3_Inhibitor: SNX10 = ARRD3&!ZFP36L2 (Confidence: 1, TimeStep: 1)

SNX10_4_Inhibitor: SNX10 = !TARSL2&!ZFP36L2 (Confidence: 1, TimeStep: 1)

SNX10_5_Inhibitor: SNX10 = SNX10&!ZFP36L2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SNX29P2 with decay value = 1:

SNX29P2_1_Activator: SNX29P2 = DDIT4&KLF9 (Confidence: 1, TimeStep: 1)

SNX29P2_2_Activator: SNX29P2 = DDIT4&!RMI2 (Confidence: 1, TimeStep: 1)

SNX29P2_3_Activator: SNX29P2 = FABP5&!IGLL1 (Confidence: 1, TimeStep: 1)

SNX29P2_4_Activator: SNX29P2 = FABP5&SNX29P2 (Confidence: 1, TimeStep: 1)

SNX29P2_5_Activator: SNX29P2 = !HBG1&SNX29P2 (Confidence: 1, TimeStep: 1)

SNX29P2_1_Inhibitor: SNX29P2 = !KLF9&SHCBP1 (Confidence: 1, TimeStep: 1)

SNX29P2_2_Inhibitor: SNX29P2 = !KLF9&RFC3 (Confidence: 1, TimeStep: 1)

SNX29P2_3_Inhibitor: SNX29P2 = !CKAP2&KIF4A (Confidence: 1, TimeStep: 1)

SNX29P2_4_Inhibitor: SNX29P2 = !AURKB&KIF4A (Confidence: 1, TimeStep: 1)

SNX29P2_5_Inhibitor: SNX29P2 = BIRC3&DEPDC1B (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SOCS1 with decay value = 1:

SOCS1_1_Activator: SOCS1 = PPBP (Confidence: 1, TimeStep: 1)

SOCS1_2_Activator: SOCS1 = FGL2 (Confidence: 1, TimeStep: 1)

SOCS1_3_Activator: SOCS1 = MNDA (Confidence: 1, TimeStep: 1)

SOCS1_1_Inhibitor: SOCS1 = !SOCS1&!TXNIP (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SOS1 with decay value = 1:

SOS1_1_Activator: SOS1 = GBP4 (Confidence: 1, TimeStep: 1)

SOS1_2_Activator: SOS1 = LILRB2 (Confidence: 1, TimeStep: 1)

SOS1_3_Activator: SOS1 = !CENPV (Confidence: 1, TimeStep: 1)

SOS1_4_Activator: SOS1 = IRAK3 (Confidence: 1, TimeStep: 1)

SOS1_5_Activator: SOS1 = DENND3 (Confidence: 1, TimeStep: 1)

SOS1_1_Inhibitor: SOS1 = UBE2C (Confidence: 1, TimeStep: 1)

SOS1_2_Inhibitor: SOS1 = BCL2L1&!LY96 (Confidence: 1, TimeStep: 1)

SOS1_3_Inhibitor: SOS1 = CENPV&!LY96 (Confidence: 1, TimeStep: 1)

SOS1_4_Inhibitor: SOS1 = DEPDC1B&!LY96 (Confidence: 1, TimeStep: 1)

SOS1_5_Inhibitor: SOS1 = !GBP4&!LY96 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SQLE with decay value = 1:

SQLE_1_Activator: SQLE = CCNB2 (Confidence: 1, TimeStep: 1)

SQLE_2_Activator: SQLE = CDC45 (Confidence: 1, TimeStep: 1)

SQLE_3_Activator: SQLE = CENPA (Confidence: 1, TimeStep: 1)

SQLE_4_Activator: SQLE = DLGAP5 (Confidence: 1, TimeStep: 1)

SQLE_5_Activator: SQLE = MAD2L1 (Confidence: 1, TimeStep: 1)

SQLE_1_Inhibitor: SQLE = !BYSL (Confidence: 1, TimeStep: 1)

SQLE_2_Inhibitor: SQLE = P2RY14 (Confidence: 1, TimeStep: 1)

SQL_E_3_Inhibitor: SQL_E = !PAICS (Confidence: 1, TimeStep: 1)

SQL_E_4_Inhibitor: SQL_E = TBXA2R (Confidence: 1, TimeStep: 1)

SQL_E_5_Inhibitor: SQL_E = !TNFRSF21 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for STAB1 with decay value = 1:

STAB1_1_Activator: STAB1 = EPPK1 (Confidence: 1, TimeStep: 1)

STAB1_2_Activator: STAB1 = CDC42EP3&TBXA2R (Confidence: 1, TimeStep: 1)

STAB1_3_Activator: STAB1 = CDC42EP3&TARSL2 (Confidence: 1, TimeStep: 1)

STAB1_4_Activator: STAB1 = CRNDE&STAB1 (Confidence: 1, TimeStep: 1)

STAB1_5_Activator: STAB1 = !CLEC2B&MNDA (Confidence: 1, TimeStep: 1)

STAB1_1_Inhibitor: STAB1 = CRNDE&!WFS1 (Confidence: 1, TimeStep: 1)

STAB1_2_Inhibitor: STAB1 = !C4orf46&!IRAK3 (Confidence: 1, TimeStep: 1)

STAB1_3_Inhibitor: STAB1 = !CKS1B&!IRAK3 (Confidence: 1, TimeStep: 1)

STAB1_4_Inhibitor: STAB1 = !C4orf46&!LILRB2 (Confidence: 1, TimeStep: 1)

STAB1_5_Inhibitor: STAB1 = !C4orf46&!RNASET2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for STIL with decay value = 1:

STIL_1_Activator: STIL = !BMF&MDK (Confidence: 1, TimeStep: 1)

STIL_2_Activator: STIL = CENPV&MDK (Confidence: 1, TimeStep: 1)

STIL_3_Activator: STIL = !IFNGR1&MDK (Confidence: 1, TimeStep: 1)

STIL_4_Activator: STIL = !IL1B&MDK (Confidence: 1, TimeStep: 1)

STIL_5_Activator: STIL = !IRAK3&MDK (Confidence: 1, TimeStep: 1)

STIL_1_Inhibitor: STIL = !ATAD2 (Confidence: 1, TimeStep: 1)

STIL_2_Inhibitor: STIL = !BIRC5 (Confidence: 1, TimeStep: 1)

STIL_3_Inhibitor: STIL = !BUB1 (Confidence: 1, TimeStep: 1)

STIL_4_Inhibitor: STIL = !CCNA2 (Confidence: 1, TimeStep: 1)

STIL_5_Inhibitor: STIL = !CDCA5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for SUV39H2 with decay value = 1:

SUV39H2_1_Activator: SUV39H2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

SUV39H2_2_Activator: SUV39H2 = ASF1B (Confidence: 1, TimeStep: 1)

SUV39H2_3_Activator: SUV39H2 = AURKA (Confidence: 1, TimeStep: 1)

SUV39H2_4_Activator: SUV39H2 = !BTG1 (Confidence: 1, TimeStep: 1)
SUV39H2_5_Activator: SUV39H2 = CCDC34 (Confidence: 1, TimeStep: 1)
SUV39H2_1_Inhibitor: SUV39H2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)
SUV39H2_2_Inhibitor: SUV39H2 = !ASF1B (Confidence: 1, TimeStep: 1)
SUV39H2_3_Inhibitor: SUV39H2 = !AURKA (Confidence: 1, TimeStep: 1)
SUV39H2_4_Inhibitor: SUV39H2 = BTG1 (Confidence: 1, TimeStep: 1)
SUV39H2_5_Inhibitor: SUV39H2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TARSL2 with decay value = 1:

TARSL2_1_Activator: TARSL2 = TBXA2R (Confidence: 1, TimeStep: 1)
TARSL2_2_Activator: TARSL2 = STAB1 (Confidence: 1, TimeStep: 1)
TARSL2_3_Activator: TARSL2 = F13A1 (Confidence: 1, TimeStep: 1)
TARSL2_4_Activator: TARSL2 = !BRIP1&TARSL2 (Confidence: 1, TimeStep: 1)
TARSL2_5_Activator: TARSL2 = !BUB1B&TARSL2 (Confidence: 1, TimeStep: 1)
TARSL2_1_Inhibitor: TARSL2 = !KIF2C&TTK (Confidence: 1, TimeStep: 1)
TARSL2_2_Inhibitor: TARSL2 = !CCNB1&CHEK1 (Confidence: 1, TimeStep: 1)
TARSL2_3_Inhibitor: TARSL2 = !CCNB1&FANCI (Confidence: 1, TimeStep: 1)
TARSL2_4_Inhibitor: TARSL2 = !CCNB1&FOXM1 (Confidence: 1, TimeStep: 1)
TARSL2_5_Inhibitor: TARSL2 = !CCNB1&STIL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TBXA2R with decay value = 1:

TBXA2R_1_Activator: TBXA2R = TBXA2R (Confidence: 1, TimeStep: 1)
TBXA2R_2_Activator: TBXA2R = F13A1 (Confidence: 1, TimeStep: 1)
TBXA2R_3_Activator: TBXA2R = RAB31 (Confidence: 1, TimeStep: 1)
TBXA2R_4_Activator: TBXA2R = !ANP32E&RASSF4 (Confidence: 1, TimeStep: 1)
TBXA2R_5_Activator: TBXA2R = !ASPM&IFNGR1 (Confidence: 1, TimeStep: 1)
TBXA2R_1_Inhibitor: TBXA2R = !LILRB2 (Confidence: 1, TimeStep: 1)
TBXA2R_2_Inhibitor: TBXA2R = ECT2 (Confidence: 1, TimeStep: 1)
TBXA2R_3_Inhibitor: TBXA2R = CCNB2 (Confidence: 1, TimeStep: 1)
TBXA2R_4_Inhibitor: TBXA2R = CDC45 (Confidence: 1, TimeStep: 1)
TBXA2R_5_Inhibitor: TBXA2R = CENPA (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TCF19 with decay value = 1:

TCF19_1_Activator: TCF19 = !P2RX5&PHF19 (Confidence: 1, TimeStep: 1)

TCF19_2_Activator: TCF19 = !P2RX5&POLE2 (Confidence: 1, TimeStep: 1)

TCF19_3_Activator: TCF19 = !P2RX5&TIPIN (Confidence: 1, TimeStep: 1)

TCF19_4_Activator: TCF19 = !P2RX5&TPX2 (Confidence: 1, TimeStep: 1)

TCF19_5_Activator: TCF19 = !P2RX5&UBE2T (Confidence: 1, TimeStep: 1)

TCF19_1_Inhibitor: TCF19 = !WDR76 (Confidence: 1, TimeStep: 1)

TCF19_2_Inhibitor: TCF19 = !E2F8 (Confidence: 1, TimeStep: 1)

TCF19_3_Inhibitor: TCF19 = !GINS2 (Confidence: 1, TimeStep: 1)

TCF19_4_Inhibitor: TCF19 = !DTL (Confidence: 1, TimeStep: 1)

TCF19_5_Inhibitor: TCF19 = !TOP2A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TENM4 with decay value = 1:

TENM4_1_Activator: TENM4 = AURKB&MDM2 (Confidence: 1, TimeStep: 1)

TENM4_2_Activator: TENM4 = AURKB&TENM4 (Confidence: 1, TimeStep: 1)

TENM4_3_Activator: TENM4 = CENPU&MDM2 (Confidence: 1, TimeStep: 1)

TENM4_4_Activator: TENM4 = CEP55&TENM4 (Confidence: 1, TimeStep: 1)

TENM4_5_Activator: TENM4 = CKAP2L&TENM4 (Confidence: 1, TimeStep: 1)

TENM4_1_Inhibitor: TENM4 = !TENM4 (Confidence: 1, TimeStep: 1)

TENM4_2_Inhibitor: TENM4 = !AURKB (Confidence: 1, TimeStep: 1)

TENM4_3_Inhibitor: TENM4 = !KIF18A (Confidence: 1, TimeStep: 1)

TENM4_4_Inhibitor: TENM4 = !OIP5 (Confidence: 1, TimeStep: 1)

TENM4_5_Inhibitor: TENM4 = !TRIP13 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TIMELESS with decay value = 1:

TIMELESS_1_Activator: TIMELESS = ANLN&MDK (Confidence: 1, TimeStep: 1)

TIMELESS_2_Activator: TIMELESS = BCAT1&MDK (Confidence: 1, TimeStep: 1)

TIMELESS_3_Activator: TIMELESS = !CD53&MDK (Confidence: 1, TimeStep: 1)

TIMELESS_4_Activator: TIMELESS = !ANP32E&BUB1B (Confidence: 1, TimeStep: 1)

TIMELESS_5_Activator: TIMELESS = !ANP32E&C4orf46 (Confidence: 1, TimeStep: 1)

TIMELESS_1_Inhibitor: TIMELESS = !CKS1B (Confidence: 1, TimeStep: 1)

TIMELESS_2_Inhibitor: TIMELESS = !TIMELESS (Confidence: 1, TimeStep: 1)

TIMELESS_3_Inhibitor: TIMELESS = !BRCA1 (Confidence: 1, TimeStep: 1)

TIMELESS_4_Inhibitor: TIMELESS = !MCM7 (Confidence: 1, TimeStep: 1)

TIMELESS_5_Inhibitor: TIMELESS = !BYSL (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TIPIN with decay value = 1:

TIPIN_1_Activator: TIPIN = APITD1-CORT (Confidence: 1, TimeStep: 1)

TIPIN_2_Activator: TIPIN = ASF1B (Confidence: 1, TimeStep: 1)

TIPIN_3_Activator: TIPIN = AURKA (Confidence: 1, TimeStep: 1)

TIPIN_4_Activator: TIPIN = !BTG1 (Confidence: 1, TimeStep: 1)

TIPIN_5_Activator: TIPIN = CCDC34 (Confidence: 1, TimeStep: 1)

TIPIN_1_Inhibitor: TIPIN = !APITD1-CORT (Confidence: 1, TimeStep: 1)

TIPIN_2_Inhibitor: TIPIN = !ASF1B (Confidence: 1, TimeStep: 1)

TIPIN_3_Inhibitor: TIPIN = !AURKA (Confidence: 1, TimeStep: 1)

TIPIN_4_Inhibitor: TIPIN = BTG1 (Confidence: 1, TimeStep: 1)

TIPIN_5_Inhibitor: TIPIN = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TK1 with decay value = 1:

TK1_1_Activator: TK1 = CCNB1 (Confidence: 1, TimeStep: 1)

TK1_2_Activator: TK1 = IQGAP3 (Confidence: 1, TimeStep: 1)

TK1_3_Activator: TK1 = CDK1 (Confidence: 1, TimeStep: 1)

TK1_4_Activator: TK1 = CKAP2L (Confidence: 1, TimeStep: 1)

TK1_5_Activator: TK1 = HMMR (Confidence: 1, TimeStep: 1)

TK1_1_Inhibitor: TK1 = !CDC20 (Confidence: 1, TimeStep: 1)

TK1_2_Inhibitor: TK1 = !KIF4A (Confidence: 1, TimeStep: 1)

TK1_3_Inhibitor: TK1 = !CENPW (Confidence: 1, TimeStep: 1)

TK1_4_Inhibitor: TK1 = !DHFR (Confidence: 1, TimeStep: 1)

TK1_5_Inhibitor: TK1 = !RAD51 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TMEM100 with decay value = 1:

TMEM100_1_Activator: TMEM100 = !CDC42EP3&RBMS3 (Confidence: 1, TimeStep: 1)

TMEM100_2_Activator: TMEM100 = !CDC42EP3&TMEM100 (Confidence: 1, TimeStep: 1)

TMEM100_3_Activator: TMEM100 = DTL&TMEM100 (Confidence: 1, TimeStep: 1)

TMEM100_4_Activator: TMEM100 = !MDK&RFC3 (Confidence: 1, TimeStep: 1)
TMEM100_5_Activator: TMEM100 = !MDK&PTTG1 (Confidence: 1, TimeStep: 1)
TMEM100_1_Inhibitor: TMEM100 = !IGLL1 (Confidence: 1, TimeStep: 1)
TMEM100_2_Inhibitor: TMEM100 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)
TMEM100_3_Inhibitor: TMEM100 = !DEPDC1B&!RBMS3 (Confidence: 1, TimeStep: 1)
TMEM100_4_Inhibitor: TMEM100 = !FH&!RBMS3 (Confidence: 1, TimeStep: 1)
TMEM100_5_Inhibitor: TMEM100 = GIMAP7&!RBMS3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TMEM2 with decay value = 1:

TMEM2_1_Activator: TMEM2 = LILRB2 (Confidence: 1, TimeStep: 1)
TMEM2_2_Activator: TMEM2 = !CENPV (Confidence: 1, TimeStep: 1)
TMEM2_3_Activator: TMEM2 = IRAK3 (Confidence: 1, TimeStep: 1)
TMEM2_4_Activator: TMEM2 = GSN (Confidence: 1, TimeStep: 1)
TMEM2_5_Activator: TMEM2 = AKAP12 (Confidence: 1, TimeStep: 1)
TMEM2_1_Inhibitor: TMEM2 = !METTL7A (Confidence: 1, TimeStep: 1)
TMEM2_2_Inhibitor: TMEM2 = UBE2C (Confidence: 1, TimeStep: 1)
TMEM2_3_Inhibitor: TMEM2 = !AKAP12&!LILRB2 (Confidence: 1, TimeStep: 1)
TMEM2_4_Inhibitor: TMEM2 = !AKAP12&!IL6ST (Confidence: 1, TimeStep: 1)
TMEM2_5_Inhibitor: TMEM2 = !AKAP12&!KLF9 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TMEM97 with decay value = 1:

TMEM97_1_Activator: TMEM97 = CENPH (Confidence: 1, TimeStep: 1)
TMEM97_2_Activator: TMEM97 = APITD1-CORT (Confidence: 1, TimeStep: 1)
TMEM97_3_Activator: TMEM97 = ASF1B (Confidence: 1, TimeStep: 1)
TMEM97_4_Activator: TMEM97 = AURKA (Confidence: 1, TimeStep: 1)
TMEM97_5_Activator: TMEM97 = !BTG1 (Confidence: 1, TimeStep: 1)
TMEM97_1_Inhibitor: TMEM97 = !CENPH (Confidence: 1, TimeStep: 1)
TMEM97_2_Inhibitor: TMEM97 = !CDK1 (Confidence: 1, TimeStep: 1)
TMEM97_3_Inhibitor: TMEM97 = !HMMR (Confidence: 1, TimeStep: 1)
TMEM97_4_Inhibitor: TMEM97 = !KIF14 (Confidence: 1, TimeStep: 1)
TMEM97_5_Inhibitor: TMEM97 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TNFRSF21 with decay value = 1:

TNFRSF21_1_Activator: TNFRSF21 = BYSL&!RASSF4 (Confidence: 1, TimeStep: 1)

TNFRSF21_2_Activator: TNFRSF21 = DTL&!STAB1 (Confidence: 1, TimeStep: 1)

TNFRSF21_3_Activator: TNFRSF21 = BRCA1&!STAB1 (Confidence: 1, TimeStep: 1)

TNFRSF21_4_Activator: TNFRSF21 = CKS1B&!STAB1 (Confidence: 1, TimeStep: 1)

TNFRSF21_5_Activator: TNFRSF21 = GINS2&!STAB1 (Confidence: 1, TimeStep: 1)

TNFRSF21_1_Inhibitor: TNFRSF21 = LILRA1 (Confidence: 1, TimeStep: 1)

TNFRSF21_2_Inhibitor: TNFRSF21 = !ANP32E&!TNFRSF21 (Confidence: 1, TimeStep: 1)

TNFRSF21_3_Inhibitor: TNFRSF21 = !FH&!TNFRSF21 (Confidence: 1, TimeStep: 1)

TNFRSF21_4_Inhibitor: TNFRSF21 = !DHFR&!TNFRSF21 (Confidence: 1, TimeStep: 1)

TNFRSF21_5_Inhibitor: TNFRSF21 = HBB&RNASET2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TNFSF8 with decay value = 1:

TNFSF8_1_Activator: TNFSF8 = TNFSF8 (Confidence: 1, TimeStep: 1)

TNFSF8_2_Activator: TNFSF8 = ITGB2-AS1 (Confidence: 1, TimeStep: 1)

TNFSF8_3_Activator: TNFSF8 = !RAG1 (Confidence: 1, TimeStep: 1)

TNFSF8_4_Activator: TNFSF8 = LOC100130872 (Confidence: 1, TimeStep: 1)

TNFSF8_5_Activator: TNFSF8 = SEMA4D (Confidence: 1, TimeStep: 1)

TNFSF8_1_Inhibitor: TNFSF8 = !SNX10 (Confidence: 1, TimeStep: 1)

TNFSF8_2_Inhibitor: TNFSF8 = LOC728175 (Confidence: 1, TimeStep: 1)

TNFSF8_3_Inhibitor: TNFSF8 = LOC100996643&!TNFSF8 (Confidence: 1, TimeStep: 1)

TNFSF8_4_Inhibitor: TNFSF8 = !BIRC3&MYRIP (Confidence: 1, TimeStep: 1)

TNFSF8_5_Inhibitor: TNFSF8 = LOC100996643&TMEM2 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TOP2A with decay value = 1:

TOP2A_1_Activator: TOP2A = CENPF (Confidence: 1, TimeStep: 1)

TOP2A_2_Activator: TOP2A = DEPDC1B (Confidence: 1, TimeStep: 1)

TOP2A_3_Activator: TOP2A = NEK2 (Confidence: 1, TimeStep: 1)

TOP2A_4_Activator: TOP2A = CDK1 (Confidence: 1, TimeStep: 1)

TOP2A_5_Activator: TOP2A = CKAP2L (Confidence: 1, TimeStep: 1)

TOP2A_1_Inhibitor: TOP2A = !E2F8 (Confidence: 1, TimeStep: 1)

TOP2A_2_Inhibitor: TOP2A = !TYMS (Confidence: 1, TimeStep: 1)

TOP2A_3_Inhibitor: TOP2A = PPP1R16B (Confidence: 1, TimeStep: 1)

TOP2A_4_Inhibitor: TOP2A = !PTP4A1 (Confidence: 1, TimeStep: 1)

TOP2A_5_Inhibitor: TOP2A = !FABP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TPX2 with decay value = 1:

TPX2_1_Activator: TPX2 = APITD1-CORT (Confidence: 1, TimeStep: 1)

TPX2_2_Activator: TPX2 = ASF1B (Confidence: 1, TimeStep: 1)

TPX2_3_Activator: TPX2 = AURKA (Confidence: 1, TimeStep: 1)

TPX2_4_Activator: TPX2 = !BTG1 (Confidence: 1, TimeStep: 1)

TPX2_5_Activator: TPX2 = CCDC34 (Confidence: 1, TimeStep: 1)

TPX2_1_Inhibitor: TPX2 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

TPX2_2_Inhibitor: TPX2 = !ASF1B (Confidence: 1, TimeStep: 1)

TPX2_3_Inhibitor: TPX2 = !AURKA (Confidence: 1, TimeStep: 1)

TPX2_4_Inhibitor: TPX2 = BTG1 (Confidence: 1, TimeStep: 1)

TPX2_5_Inhibitor: TPX2 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TRIB1 with decay value = 1:

TRIB1_1_Activator: TRIB1 = !ABHD17B (Confidence: 1, TimeStep: 1)

TRIB1_2_Activator: TRIB1 = IL18RAP (Confidence: 1, TimeStep: 1)

TRIB1_3_Activator: TRIB1 = LOC728175 (Confidence: 1, TimeStep: 1)

TRIB1_4_Activator: TRIB1 = SCML4&TRIB1 (Confidence: 1, TimeStep: 1)

TRIB1_5_Activator: TRIB1 = IL6ST&!PRPS2 (Confidence: 1, TimeStep: 1)

TRIB1_1_Inhibitor: TRIB1 = PRPS2&!TRIB1 (Confidence: 1, TimeStep: 1)

TRIB1_2_Inhibitor: TRIB1 = !IL18R1&!TRIB1 (Confidence: 1, TimeStep: 1)

TRIB1_3_Inhibitor: TRIB1 = CENPV&!TRIB1 (Confidence: 1, TimeStep: 1)

TRIB1_4_Inhibitor: TRIB1 = !IL6ST&!TRIB1 (Confidence: 1, TimeStep: 1)

TRIB1_5_Inhibitor: TRIB1 = !TMEM2&!TRIB1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TRIP13 with decay value = 1:

TRIP13_1_Activator: TRIP13 = APITD1-CORT (Confidence: 1, TimeStep: 1)

TRIP13_2_Activator: TRIP13 = ASF1B (Confidence: 1, TimeStep: 1)

TRIP13_3_Activator: TRIP13 = AURKA (Confidence: 1, TimeStep: 1)

TRIP13_4_Activator: TRIP13 = !BTG1 (Confidence: 1, TimeStep: 1)

TRIP13_5_Activator: TRIP13 = CCDC34 (Confidence: 1, TimeStep: 1)

TRIP13_1_Inhibitor: TRIP13 = !APITD1-CORT (Confidence: 1, TimeStep: 1)

TRIP13_2_Inhibitor: TRIP13 = !ASF1B (Confidence: 1, TimeStep: 1)

TRIP13_3_Inhibitor: TRIP13 = !AURKA (Confidence: 1, TimeStep: 1)

TRIP13_4_Inhibitor: TRIP13 = BTG1 (Confidence: 1, TimeStep: 1)

TRIP13_5_Inhibitor: TRIP13 = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TTK with decay value = 1:

TTK_1_Activator: TTK = KIF2C (Confidence: 1, TimeStep: 1)

TTK_2_Activator: TTK = ATAD2 (Confidence: 1, TimeStep: 1)

TTK_3_Activator: TTK = BIRC5 (Confidence: 1, TimeStep: 1)

TTK_4_Activator: TTK = BUB1 (Confidence: 1, TimeStep: 1)

TTK_5_Activator: TTK = CCNA2 (Confidence: 1, TimeStep: 1)

TTK_1_Inhibitor: TTK = !KIF2C (Confidence: 1, TimeStep: 1)

TTK_2_Inhibitor: TTK = !CCNB2 (Confidence: 1, TimeStep: 1)

TTK_3_Inhibitor: TTK = !CDC45 (Confidence: 1, TimeStep: 1)

TTK_4_Inhibitor: TTK = !CENPA (Confidence: 1, TimeStep: 1)

TTK_5_Inhibitor: TTK = !DLGAP5 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TUBA4A with decay value = 1:

TUBA4A_1_Activator: TUBA4A = TUBA4A (Confidence: 1, TimeStep: 1)

TUBA4A_2_Activator: TUBA4A = STAB1 (Confidence: 1, TimeStep: 1)

TUBA4A_3_Activator: TUBA4A = !LILRB2&SMIM3 (Confidence: 1, TimeStep: 1)

TUBA4A_4_Activator: TUBA4A = GBP4&P2RY14&!TENM4 (Confidence: 1, TimeStep: 1)

TUBA4A_5_Activator: TUBA4A = !DENND3&!LOC100505650&SMIM3 (Confidence: 1, TimeStep: 1)

TUBA4A_1_Inhibitor: TUBA4A = LOC100505650 (Confidence: 1, TimeStep: 1)

TUBA4A_2_Inhibitor: TUBA4A = BCL10 (Confidence: 1, TimeStep: 1)

TUBA4A_3_Inhibitor: TUBA4A = ASPM&!TUBA4A (Confidence: 1, TimeStep: 1)

TUBA4A_4_Inhibitor: TUBA4A = NUF2&!TUBA4A (Confidence: 1, TimeStep: 1)

TUBA4A_5_Inhibitor: TUBA4A = HELLS&!TUBA4A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TXNIP with decay value = 1:

TXNIP_1_Activator: TXNIP = !CKAP2 (Confidence: 1, TimeStep: 1)

TXNIP_2_Activator: TXNIP = TXNIP (Confidence: 1, TimeStep: 1)

TXNIP_3_Activator: TXNIP = !CENPU (Confidence: 1, TimeStep: 1)

TXNIP_4_Activator: TXNIP = !HIST4H4 (Confidence: 1, TimeStep: 1)

TXNIP_5_Activator: TXNIP = NEAT1 (Confidence: 1, TimeStep: 1)

TXNIP_1_Inhibitor: TXNIP = ANP32E&MDK (Confidence: 1, TimeStep: 1)

TXNIP_2_Inhibitor: TXNIP = !BMF&MDK (Confidence: 1, TimeStep: 1)

TXNIP_3_Inhibitor: TXNIP = CENPV&MDK (Confidence: 1, TimeStep: 1)

TXNIP_4_Inhibitor: TXNIP = E2F7&MDK (Confidence: 1, TimeStep: 1)

TXNIP_5_Inhibitor: TXNIP = ECT2&MDK (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for TYMS with decay value = 1:

TYMS_1_Activator: TYMS = WDR76 (Confidence: 1, TimeStep: 1)

TYMS_2_Activator: TYMS = MKI67 (Confidence: 1, TimeStep: 1)

TYMS_3_Activator: TYMS = ZWINT (Confidence: 1, TimeStep: 1)

TYMS_4_Activator: TYMS = ANLN (Confidence: 1, TimeStep: 1)

TYMS_5_Activator: TYMS = BRIP1 (Confidence: 1, TimeStep: 1)

TYMS_1_Inhibitor: TYMS = !TYMS (Confidence: 1, TimeStep: 1)

TYMS_2_Inhibitor: TYMS = PRDM1 (Confidence: 1, TimeStep: 1)

TYMS_3_Inhibitor: TYMS = LGALS3 (Confidence: 1, TimeStep: 1)

TYMS_4_Inhibitor: TYMS = RAB31 (Confidence: 1, TimeStep: 1)

TYMS_5_Inhibitor: TYMS = !ANLN&BIRC3 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for UBE2C with decay value = 1:

UBE2C_1_Activator: UBE2C = UBE2C (Confidence: 1, TimeStep: 1)

UBE2C_2_Activator: UBE2C = !AKAP12&APITD1-CORT (Confidence: 1, TimeStep: 1)

UBE2C_3_Activator: UBE2C = !AKAP12&ASF1B (Confidence: 1, TimeStep: 1)

UBE2C_4_Activator: UBE2C = !AKAP12&AURKA (Confidence: 1, TimeStep: 1)

UBE2C_5_Activator: UBE2C = !AKAP12&AURKB (Confidence: 1, TimeStep: 1)

UBE2C_1_Inhibitor: UBE2C = !UBE2C (Confidence: 1, TimeStep: 1)

UBE2C_2_Inhibitor: UBE2C = !APITD1-CORT (Confidence: 1, TimeStep: 1)

UBE2C_3_Inhibitor: UBE2C = !ASF1B (Confidence: 1, TimeStep: 1)

UBE2C_4_Inhibitor: UBE2C = !AURKA (Confidence: 1, TimeStep: 1)

UBE2C_5_Inhibitor: UBE2C = BTG1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for UBE2T with decay value = 1:

UBE2T_1_Activator: UBE2T = APITD1-CORT (Confidence: 1, TimeStep: 1)

UBE2T_2_Activator: UBE2T = ASF1B (Confidence: 1, TimeStep: 1)

UBE2T_3_Activator: UBE2T = AURKA (Confidence: 1, TimeStep: 1)

UBE2T_4_Activator: UBE2T = !BTG1 (Confidence: 1, TimeStep: 1)

UBE2T_5_Activator: UBE2T = CCDC34 (Confidence: 1, TimeStep: 1)

UBE2T_1_Inhibitor: UBE2T = !APITD1-CORT (Confidence: 1, TimeStep: 1)

UBE2T_2_Inhibitor: UBE2T = !ASF1B (Confidence: 1, TimeStep: 1)

UBE2T_3_Inhibitor: UBE2T = !AURKA (Confidence: 1, TimeStep: 1)

UBE2T_4_Inhibitor: UBE2T = BTG1 (Confidence: 1, TimeStep: 1)

UBE2T_5_Inhibitor: UBE2T = !CCDC34 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for WASF1 with decay value = 1:

WASF1_1_Activator: WASF1 = P2RX5 (Confidence: 1, TimeStep: 1)

WASF1_2_Activator: WASF1 = !HBG1&WASF1 (Confidence: 1, TimeStep: 1)

WASF1_3_Activator: WASF1 = !HBB&WASF1 (Confidence: 1, TimeStep: 1)

WASF1_4_Activator: WASF1 = !IGH&WASF1 (Confidence: 1, TimeStep: 1)

WASF1_5_Activator: WASF1 = !F13A1&!S100A11 (Confidence: 1, TimeStep: 1)

WASF1_1_Inhibitor: WASF1 = !WASF1 (Confidence: 1, TimeStep: 1)

WASF1_2_Inhibitor: WASF1 = FCGR3B&HBG1 (Confidence: 1, TimeStep: 1)

WASF1_3_Inhibitor: WASF1 = FCGR3B&!IL1B (Confidence: 1, TimeStep: 1)

WASF1_4_Inhibitor: WASF1 = FCGR3B&ITGB2-AS1 (Confidence: 1, TimeStep: 1)

WASF1_5_Inhibitor: WASF1 = ITGB2-AS1&!SNORA21 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for WDHD1 with decay value = 1:

WDHD1_1_Activator: WDHD1 = !CD53&MDK (Confidence: 1, TimeStep: 1)

WDHD1_2_Activator: WDHD1 = CENPK&MDK (Confidence: 1, TimeStep: 1)

WDHD1_3_Activator: WDHD1 = !IGH&MDK (Confidence: 1, TimeStep: 1)

WDHD1_4_Activator: WDHD1 = !IGLC1&MDK (Confidence: 1, TimeStep: 1)

WDHD1_5_Activator: WDHD1 = !BMF&MDK (Confidence: 1, TimeStep: 1)

WDHD1_1_Inhibitor: WDHD1 = !CENPH (Confidence: 1, TimeStep: 1)

WDHD1_2_Inhibitor: WDHD1 = !CDK1 (Confidence: 1, TimeStep: 1)

WDHD1_3_Inhibitor: WDHD1 = !HMMR (Confidence: 1, TimeStep: 1)

WDHD1_4_Inhibitor: WDHD1 = !KIF14 (Confidence: 1, TimeStep: 1)

WDHD1_5_Inhibitor: WDHD1 = !KIF20A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for WDR76 with decay value = 1:

WDR76_1_Activator: WDR76 = CENPH (Confidence: 1, TimeStep: 1)

WDR76_2_Activator: WDR76 = APITD1-CORT (Confidence: 1, TimeStep: 1)

WDR76_3_Activator: WDR76 = ASF1B (Confidence: 1, TimeStep: 1)

WDR76_4_Activator: WDR76 = AURKA (Confidence: 1, TimeStep: 1)

WDR76_5_Activator: WDR76 = !BTG1 (Confidence: 1, TimeStep: 1)

WDR76_1_Inhibitor: WDR76 = !IQGAP3 (Confidence: 1, TimeStep: 1)

WDR76_2_Inhibitor: WDR76 = !ANLN (Confidence: 1, TimeStep: 1)

WDR76_3_Inhibitor: WDR76 = !PRR11 (Confidence: 1, TimeStep: 1)

WDR76_4_Inhibitor: WDR76 = !CDC20 (Confidence: 1, TimeStep: 1)

WDR76_5_Inhibitor: WDR76 = !KIF4A (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for WFS1 with decay value = 1:

WFS1_1_Activator: WFS1 = PDE4B&PTP4A1 (Confidence: 1, TimeStep: 1)

WFS1_2_Activator: WFS1 = !SERPINA1&WFS1 (Confidence: 1, TimeStep: 1)

WFS1_3_Activator: WFS1 = ARPP21&WFS1 (Confidence: 1, TimeStep: 1)

WFS1_4_Activator: WFS1 = !HBG1&PDE4B (Confidence: 1, TimeStep: 1)

WFS1_5_Activator: WFS1 = PDE4B&WFS1 (Confidence: 1, TimeStep: 1)

WFS1_1_Inhibitor: WFS1 = !TGB2-AS1 (Confidence: 1, TimeStep: 1)

WFS1_2_Inhibitor: WFS1 = LOC100130872 (Confidence: 1, TimeStep: 1)

WFS1_3_Inhibitor: WFS1 = LILRA1 (Confidence: 1, TimeStep: 1)

WFS1_4_Inhibitor: WFS1 = !KIF26A&!WFS1 (Confidence: 1, TimeStep: 1)

WFS1_5_Inhibitor: WFS1 = !L1B&!WFS1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ZBTB16 with decay value = 1:

ZBTB16_1_Activator: ZBTB16 = LILRA1 (Confidence: 1, TimeStep: 1)

ZBTB16_2_Activator: ZBTB16 = !CCR1&ZBTB16 (Confidence: 1, TimeStep: 1)

ZBTB16_3_Activator: ZBTB16 = !GSN&LILRB2 (Confidence: 1, TimeStep: 1)

ZBTB16_4_Activator: ZBTB16 = !GSN&SERPINB9 (Confidence: 1, TimeStep: 1)

ZBTB16_5_Activator: ZBTB16 = !GSN&ZBTB16 (Confidence: 1, TimeStep: 1)

ZBTB16_1_Inhibitor: ZBTB16 = !LILRB2&!ZBTB16 (Confidence: 1, TimeStep: 1)

ZBTB16_2_Inhibitor: ZBTB16 = !RBMS3&SNORA21 (Confidence: 1, TimeStep: 1)

ZBTB16_3_Inhibitor: ZBTB16 = DFNA5&!ZBTB16 (Confidence: 1, TimeStep: 1)

ZBTB16_4_Inhibitor: ZBTB16 = !GBP4&SNORA21 (Confidence: 1, TimeStep: 1)

ZBTB16_5_Inhibitor: ZBTB16 = !GBP4&!ZBTB16 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ZFP36L2 with decay value = 1:

ZFP36L2_1_Activator: ZFP36L2 = ZFP36L2 (Confidence: 1, TimeStep: 1)

ZFP36L2_2_Activator: ZFP36L2 = TXNIP (Confidence: 1, TimeStep: 1)

ZFP36L2_3_Activator: ZFP36L2 = LEF1 (Confidence: 1, TimeStep: 1)

ZFP36L2_4_Activator: ZFP36L2 = !HIST4H4 (Confidence: 1, TimeStep: 1)

ZFP36L2_5_Activator: ZFP36L2 = !MTHFD2 (Confidence: 1, TimeStep: 1)

ZFP36L2_1_Inhibitor: ZFP36L2 = CKAP2 (Confidence: 1, TimeStep: 2)

ZFP36L2_2_Inhibitor: ZFP36L2 = !TXNIP (Confidence: 1, TimeStep: 2)

ZFP36L2_3_Inhibitor: ZFP36L2 = !ZFP36L2 (Confidence: 1, TimeStep: 2)

Multiple Transition Functions for ZNF367 with decay value = 1:

ZNF367_1_Activator: ZNF367 = MDK&MKI67 (Confidence: 1, TimeStep: 1)

ZNF367_2_Activator: ZNF367 = MDK&NCAPG (Confidence: 1, TimeStep: 1)

ZNF367_3_Activator: ZNF367 = MDK&NEK2 (Confidence: 1, TimeStep: 1)

ZNF367_4_Activator: ZNF367 = MDK&NUSAP1 (Confidence: 1, TimeStep: 1)

ZNF367_5_Activator: ZNF367 = MDK&POLQ (Confidence: 1, TimeStep: 1)

ZNF367_1_Inhibitor: ZNF367 = !CKS1B (Confidence: 1, TimeStep: 1)

ZNF367_2_Inhibitor: ZNF367 = !TIMELESS (Confidence: 1, TimeStep: 1)

ZNF367_3_Inhibitor: ZNF367 = !BRCA1 (Confidence: 1, TimeStep: 1)

ZNF367_4_Inhibitor: ZNF367 = !MCM7 (Confidence: 1, TimeStep: 1)

ZNF367_5_Inhibitor: ZNF367 = !RAD51AP1 (Confidence: 1, TimeStep: 1)

Multiple Transition Functions for ZWINT with decay value = 1:

ZWINT_1_Activator: ZWINT = KIF2C (Confidence: 1, TimeStep: 1)

ZWINT_2_Activator: ZWINT = ATAD2 (Confidence: 1, TimeStep: 1)

ZWINT_3_Activator: ZWINT = BIRC5 (Confidence: 1, TimeStep: 1)

ZWINT_4_Activator: ZWINT = BUB1 (Confidence: 1, TimeStep: 1)

ZWINT_5_Activator: ZWINT = CCNA2 (Confidence: 1, TimeStep: 1)

ZWINT_1_Inhibitor: ZWINT = !KIF2C (Confidence: 1, TimeStep: 1)

ZWINT_2_Inhibitor: ZWINT = !CCNB2 (Confidence: 1, TimeStep: 1)

ZWINT_3_Inhibitor: ZWINT = !CDC45 (Confidence: 1, TimeStep: 1)

ZWINT_4_Inhibitor: ZWINT = !CENPA (Confidence: 1, TimeStep: 1)

ZWINT_5_Inhibitor: ZWINT = !DLGAP5 (Confidence: 1, TimeStep: 1)

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