

Bipolar multiplex families have an increased burden of common risk variants for psychiatric disorders

Supplementary Material

Table of Contents

Supplementary Methods: Sample description, quality control, population substructure analysis, imputation, generation and analysis of PRS.

Supplement. Discussion: Discussion of the analyses of FAM_{MDD} cases.

Supplement. References

Supplementary Fig. S1: Boxplots of PRS at different p -value thresholds for CC_{controls}, FAM_{unaffected}, and BD cases.

Supplementary Fig. S2: Association analysis comparing PRS in FAM_{BD} cases and CC_{controls}.

Supplementary Fig. S3: Association analysis comparing PRS in FAM_{BD} cases and unrelated CC_{BD} cases.

Supplementary Fig. S4: Association analysis comparing PRS in FAM_{unaffected} and CC_{controls}.

Supplementary Fig. S5: Association analysis comparing PRS in FAM_{BD} cases and FAM_{unaffected}.

Supplementary Fig. S6: Analysis of assortative mating.

Supplementary Fig. S7: Analysis of anticipation in the FAM sample.

Supplementary Fig. S8: Boxplots of PRS at different p -value thresholds, including FAM_{MDD} cases.

Supplementary Fig. S9: Association analysis comparing PRS in FAM_{MDD} cases and CC_{controls}.

Supplementary Fig. S10: Association analysis comparing PRS in FAM_{MDD} cases and FAM_{unaffected}.

Supplementary Fig. S11: Population substructure analysis.

Supplementary Fig. S12: Association analysis comparing BD PRS in unrelated CC_{BD} cases and CC_{controls}.

IGAP Supplementary Methods and Acknowledgments

Authors of the Bipolar Disorder Working Group of the PGC

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Supplementary Tables in the separate Excel file:

- Supplementary Table S1:** Test statistics of the demographic comparisons between the different samples (see Table 1).
- Supplementary Table S2:** The number of variants used for calculation of each PRS (after clumping).
- Supplementary Table S3:** Full association test results for the comparison of PRS between FAM_{BD} cases and CC_{controls}. Covariate used: Sex. Married-in family members were excluded.
- Supplementary Table S4:** Full association test results for the comparison of PRS between FAM_{BD} cases and unrelated CC_{BD} cases. Covariate used: Sex. Married-in family members were excluded.
- Supplementary Table S5:** Full association test results for the comparison of PRS between FAM_{unaffected} and CC_{controls}. Covariates used: Sex. Married-in family members were excluded.
- Supplementary Table S6:** Full association test results for the comparison of PRS between FAM_{BD} cases and FAM_{unaffected}. Fixed effects covariates: Sex, age at interview; random effects covariate: married-in status.
- Supplementary Table S7:** Full association test results for the analysis of assortative mating. Covariate used: Sex.
- Supplementary Table S8:** Full association test results for the analysis of anticipation. Covariates used: Sex, age at the interview, diagnostic group (BD/MDD/unaffected). Married-in family members were excluded.
- Supplementary Table S9:** Full association test results for the comparison of PRS between FAM_{MDD} cases and CC_{controls}. Covariate used: Sex. Married-in family members were excluded.
- Supplementary Table S10:** Full association test results for the comparison of PRS between FAM_{MDD} cases and FAM_{unaffected}. Fixed effects covariates: Sex, age at interview; random effects covariate: married-in status.
- Supplementary Table S11:** Full association test results for the comparison of BD PRS between unrelated CC_{BD} cases and CC_{controls}. Covariate used: Sex.

Supplementary Methods

Extended FAM sample description

We included 395 members of 33 families in the present analyses. 166 participants were diagnosed with BD (BD type I (BD-I), n=115; BD type II (BD-II), n=41; not otherwise specified (NOS) BD, n=10), 78 with MDD (recurrent MDD (R-MDD), n=53; single episode MDD (SE-MDD), n=17; NOS MDD, n=8), and 151 without a history of an affective disorder.

Diagnoses were assigned by two trained clinicians according to DSM IV using the best estimate approach. Diagnosis and clinical data were based on the Schedule for Affective Disorders and Schizophrenia (SADS)¹, the Operational Criteria Checklist for Psychotic and Affective Illness (OPCRIT)², the Family Informant Schedule and Criteria (FISC)³, and on clinical records.

A severe impairment during the disorder (see Table 1) corresponded to a level of 3 in the OPCRIT item 87 (no function at all in a major life role for more than two days or in-patient treatment has been required or active psychotic symptoms such as delusions or hallucinations have occurred).

Quality control (QC)

QC of genotype data was conducted in PLINK v1.90b3.36. QC was carried out first on each of both cohorts separately (FAM and CC), followed by a second round of QC on the combined dataset.

Sequence of QC steps:

1. FAM (genotyped on Infinium PsychArray BeadChip (PsychChip))
Before QC: 395 individuals and 588,454 variants
 - 1.1. Removal of SNPs with call rates <98% or a MAF <1%
 - 1.2. Check for individuals with genotyping rates <98% (*none removed*)
 - 1.3. Check for sex mismatches (*none removed*)
 - 1.4. Removal of non-autosomal variants
 - 1.5. Removal of SNPs with call rates <98%, a MAF <1%, or Hardy-Weinberg Equilibrium (HWE) test p -values < 1×10^{-6}
 - 1.6. Removal of A/T and G/C SNPs
 - 1.7. Update of variant IDs and positions to the IDs and positions in the 1000 Genomes Phase 3 reference panel
 - 1.8. Alignment of alleles to the reference panel
 - 1.9. Removal of duplicated variants and of variants not present in the reference panelAfter QC: 395 individuals and 258,046 variants

2. CC (Illumina HumanOmni1-Quad and Illumina Human610-Quad, combined and quality-controlled as previously published⁴; the QC described here was conducted on the published data)

Before QC: 547 individuals and 333,353 variants

 - 2.1. Removal of SNPs with call rates <98% or a MAF <1%
 - 2.2. Check for individuals with genotyping rates <98% (*none removed*)
 - 2.3. Check for sex mismatches (*none removed*)
 - 2.4. Check for genetic duplicates (*none removed*)
 - 2.5. Removal of individuals where the autosomal or X-chromosomal heterozygosity deviated from the mean >4 SD (*six removed*)
 - 2.6. Removal of non-autosomal variants
 - 2.7. Removal of SNPs with call rates <98%, a MAF <1%, or HWE test p -values <1×10⁻⁶
 - 2.8. Removal of A/T and G/C SNPs
 - 2.9. Update of variant IDs and positions to the IDs and positions in the 1000 Genomes Phase 3 reference panel
 - 2.10. Alignment of alleles to the reference panel
 - 2.11. Removal of duplicated variants and variants not present in the reference panel

After QC: 541 individuals and 315,634 variants

3. Combined dataset of both samples (936 individuals and 116,079 variants)
 - 3.1. Removal of SNPs with call rates <98% or a MAF <1%
 - 3.2. Removal of individuals with genotyping rates <98% (*two removed*)
 - 3.3. Removal of individuals duplicated between both datasets (*31 removed from the CC sample*)
 - 3.4. Removal of genetic outliers with a distance from the mean of >4 SD in the first eight MDS components (*15 removed from the CC sample*)
 - 3.5. Removal of individuals where the autosomal heterozygosity deviated from the mean >4 SD (*eleven removed from the FAM sample*)
 - 3.6. Removal of SNPs with call rates <98%, a MAF <1%, or HWE test p -values <1×10⁻⁶
 - 3.7. Removal of individuals from the CC sample that have been recruited as part of the FAM/ABiF cohort (*55 removed*)

After QC: 822 individuals (384 FAM and 438 CC) and 116,067 variants

Population substructure analysis

For the population substructure analyses, pre-imputation genotype data was used, after the QC steps explained above had been applied. Additional variant filtering steps were: removal of variants with a MAF <0.05 or HWE p -value <10⁻³; removal of variants mapping to the extended MHC region (chromosome 6, 25-35 Mbp) or to a typical inversion site on chromosome 8 (7-13 Mbp); LD pruning (command `--indep-pairwise 200 100 0.2`).

Next, the pairwise identity-by-state (IBS) matrix of all individuals was calculated using the command `--genome` on the filtered genotype data. Multidimensional scaling (MDS) analysis was performed on the IBS matrix using the eigendecomposition-based algorithm in PLINK v1.90b5.

In an MDS analysis, the high relatedness between family members leads to artifacts. To avoid such artifacts, only one person per family was included in population substructure analyses. For each of the 33 families, the individual with the highest absolute values in MDS components 1 and 2 was selected to represent the respective family. Afterwards, the MDS analysis was repeated, using only selected individuals from the FAM sample.

Whether MDS components differ between cohorts was analyzed with logistic regression using the following model without additional covariates:

cohort (FAM/CC) ~ MDS components.

The ten calculated MDS components showed association p -values with cohort ≥ 0.30 , except for component 3, which was associated with cohort at nominal significance with $p=0.036$. After correction for multiple testing (ten comparisons), this difference observed for component 3 was not significant.

Imputation of genotype data

Genotypes were aligned to the 1000 Genomes Phase 3 reference panel using SHAPEIT v2 (r837) and PLINK v1.90b3.36. Pre-phasing (haplotype estimation) was conducted for each chromosome separately using SHAPEIT with the *--duohmm* option. Imputation was performed using IMPUTE2 v2.3.2 in 5 Mbp chunks with 500 kbp buffers, filtering out variants that are monomorphic in the EUR samples. Chunks with <51 genotyped variants or concordance rates $<92\%$ were fused with neighboring chunks and re-imputed. Imputed variants with a MAF $<1\%$ or an INFO metric <0.8 were removed.

Imputed variants in the combined sample after QC: 6,862,461

Imputed variants in the FAM sample after QC: 8,628,089

Note that optimized imputation algorithms for pedigrees exist, for example, GIGI⁵. GIGI mainly improves imputation accuracy of rare variants but does not have a clear advantage over population-based methods regarding common variants. Moreover, GIGI can only impute one pedigree at a time and cannot impute unrelated individuals. As we were only interested in common variation (MAF $\geq 1\%$) and also wanted to analyse a mixed sample of related and unrelated subjects, we chose a population-based imputation method using SHAPEIT and IMPUTE2.

Generation and analysis of PRS

The GWAS test statistics and imputed variants in our data were merged based on chromosome, position, and alleles of each variant. Summary statistics were then clumped in PLINK v1.90b5.2, based on best-guess genotype data (hard-call threshold 0.3) using the following parameters:

```
--clump-kb 500 --clump-r2 0.1 --clump-p1 1 --clump-p2 1
```

PRS were then calculated in *R* v.3.3 based on imputed (dosage) data. Test statistics and alleles in the GWAS training data were flipped so that effect sizes were always positive. Thus, the weighted PRS represent cumulative, additive risk. PRS were scaled to represent the relative risk load (minimum possible cumulative risk load = 0, maximum = 1). For each disorder, ten PRS with different *p*-value thresholds were calculated: $<5 \times 10^{-8}$, $<1 \times 10^{-7}$, $<1 \times 10^{-6}$, $<1 \times 10^{-5}$, $<1 \times 10^{-4}$, <0.001 , <0.01 , <0.05 , <0.1 , <0.2 .

The analyses of linear mixed models using *GenABEL* were conducted in the following manner: In the first step of the PRS analyses, residuals were calculated with the *GenABEL polygenic* function using the formula *phenotype ~ covariates* (where the phenotype corresponded to the diagnosis/cohort groups contrasted in a given analysis), including the genetic relationship matrix as a random effect. Residuals from this model were then used in a second linear model with the formula *residuals ~ PRS*. Test statistics including 95% CI were calculated using bootstrapping (*R* package *boot*, nonparametric bootstrapping using ordinary resampling with 2,000 replications).

Data availability

GWAS summary statistics for PRS calculation can be obtained from the following sources:

PGC BD, MDD, and SCZ GWAS from the Psychiatric Genomics Consortium:
<https://www.med.unc.edu/pgc/results-and-downloads/>

From these summary statistics, the *Shared* and the simulated PRS can be calculated following the *R* scripts available at:
<https://gitlab.com/tillandlauer/abif-prs-analyses/>

The GWIS PRS can be calculated following this example:
<https://sites.google.com/site/mgnivard/gwis/code-example-decompose-2-traits>

The IGAP LOAD GWAS results can be obtained from IGAP:
http://web.pasteur-lille.fr/en/recherche/u744/igap/igap_download.php

For genotype and phenotype data of the CC sample, please contact the corresponding authors of the following study:
[Mühleisen TW, Leber M, Schulze TG, Strohmaier J, Degenhardt F, Treutlein J *et al.* Genome-wide association study reveals two new risk loci for bipolar disorder. *Nature Communations* 2014; 5:3339.](#)

For genotype and phenotype data of the ABiF sample, please contact the corresponding authors of the present study.

Supplementary Discussion

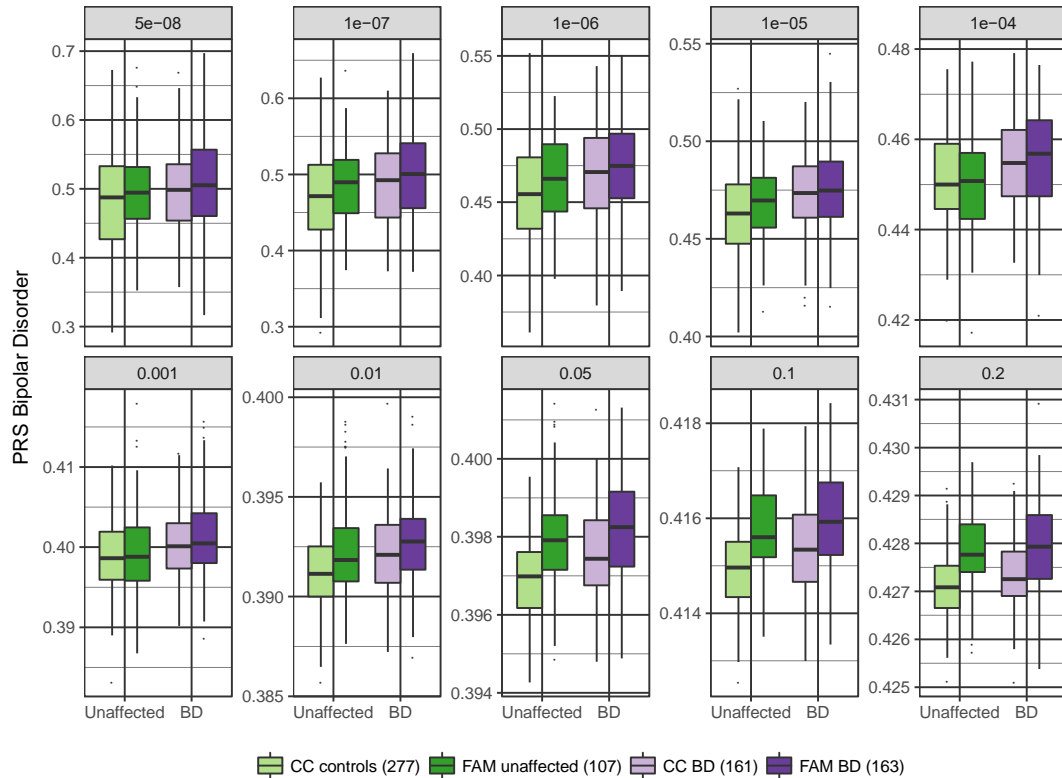
FAM_{MDD} cases had significantly higher BD and MDD than CC_{controls}. The SCZ, *Shared*, and SCZ-MDD GWIS PRS were increased at nominal significance only in FAM_{MDD}. This may be due to the lower genetic correlation of MDD and SCZ compared to the correlation of BD and SCZ⁷⁻¹⁰. However, when interpreting the results for FAM_{MDD} cases, it is important to consider that much fewer MDD than BD cases have been analysed (Table 1). The power of MDD-based analyses in the present study was thus considerably lower than for BD. This lower statistical power is also a possible explanation for why FAM_{MDD} cases only showed a nominally increased MDD PRS over FAM_{unaffected} individuals. In addition to suggesting a cross-disorder illness burden, the increased BD PRS in FAM_{MDD} cases may also indicate that, in some cases, the current MDD diagnosis constituted a prodromal stage of BD¹¹. Furthermore, in ABiF families, MDD may be more strongly driven by BD risk variants and therefore have closer etiological proximity to BD than is the case for the average MDD patient from the general population.

Supplementary References

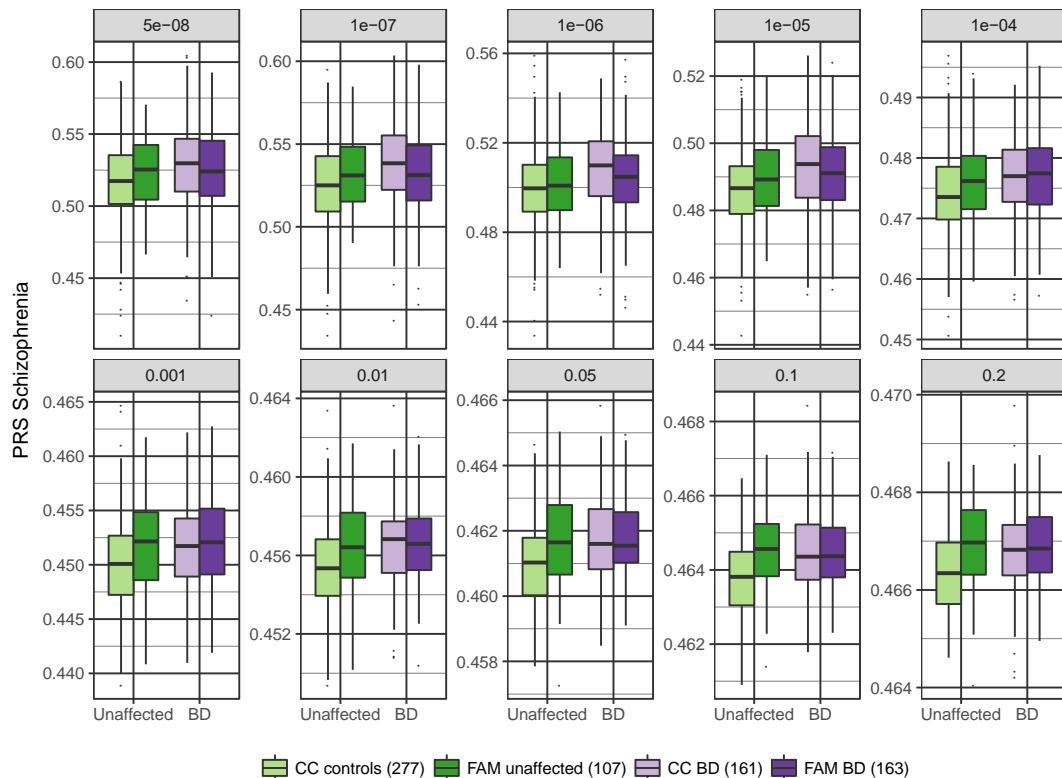
- 1 Spitzer RL. A diagnostic interview: the schedule for affective disorders and schizophrenia. *Arch Gen Psychiatry* 1978; **35**: 837–844.
- 2 McGuffin P, Farmer A, Harvey I. A polydiagnostic application of operational criteria in studies of psychotic illness. Development and reliability of the OPCRIT system. *Arch. Gen. Psychiatry*. 1991; **48**: 764–770.
- 3 Mannuzza S, Fyer AJ, Klein DF, Robins LN. Family informant schedule and criteria (FISC). *New York: Anxiety Disorder Clinic, New York State Psychiatric Institute* 1985.
- 4 Mühleisen TW, Leber M, Schulze TG, Strohmaier J, Degenhardt F, Treutlein J *et al*. Genome-wide association study reveals two new risk loci for bipolar disorder. *Nat Commun* 2014; **5**: 3339.
- 5 Cheung CYK, Thompson EA, Wijsman EM. GIGI: an approach to effective imputation of dense genotypes on large pedigrees. *Am J Hum Genet* 2013; **92**: 504–516.
- 6 Faul F, Erdfelder E, Buchner A, Lang A-G. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behav Res Methods* 2009; **41**: 1149–1160.
- 7 Wray NR, Ripke S, Mattheisen M, Trzaskowski M, Byrne EM, Abdellaoui A *et al*. Genome-wide association analyses identify 44 risk variants and refine the genetic architecture of major depression. *Nature Genetics* 2018; **50**: 668–681.
- 8 Ruderfer DM, Fanous AH, Ripke S, McQuillin A, Amdur RL, Schizophrenia Working Group of Psychiatric Genomics Consortium *et al*. Polygenic dissection of diagnosis and clinical dimensions of bipolar disorder and schizophrenia. *Mol Psychiatry* 2014; **19**: 1017–1024.
- 9 Cross-Disorder Group of the Psychiatric Genomics Consortium. Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *Lancet* 2013; **381**: 1371–1379.
- 10 Cross-Disorder Group of the Psychiatric Genomics Consortium, Lee SH, Ripke S, Neale BM, Faraone SV, Purcell SM *et al*. Genetic relationship between five psychiatric disorders estimated from genome-wide SNPs. *Nature Genetics* 2013; **45**: 984–994.
- 11 Berk M, Dodd S, Callaly P, Berk L, Fitzgerald P, de Castella AR *et al*. History of illness prior to a diagnosis of bipolar disorder or schizoaffective disorder. *J Affect Disord* 2007; **103**: 181–186.

Supplementary Fig. S1: Boxplots of PRS at different p -value thresholds for CC_{controls} , $FAM_{\text{unaffected}}$, and BD cases. FAM samples excluded from the analyses of the combined dataset are not shown in these plots, *i.e.*, family members with a history of substance abuse, married-in family members, or family members diagnosed with MDD. $CC =$ Case/control sample.

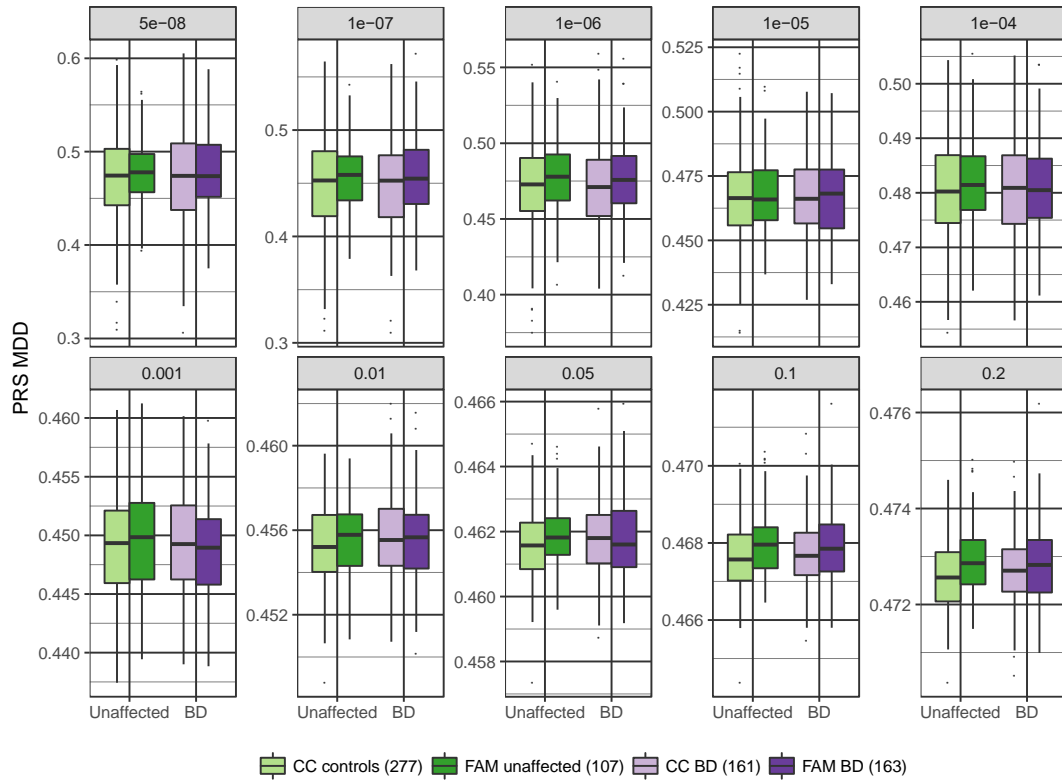
Supplementary Fig. S1A: Boxplots of BD PRS.



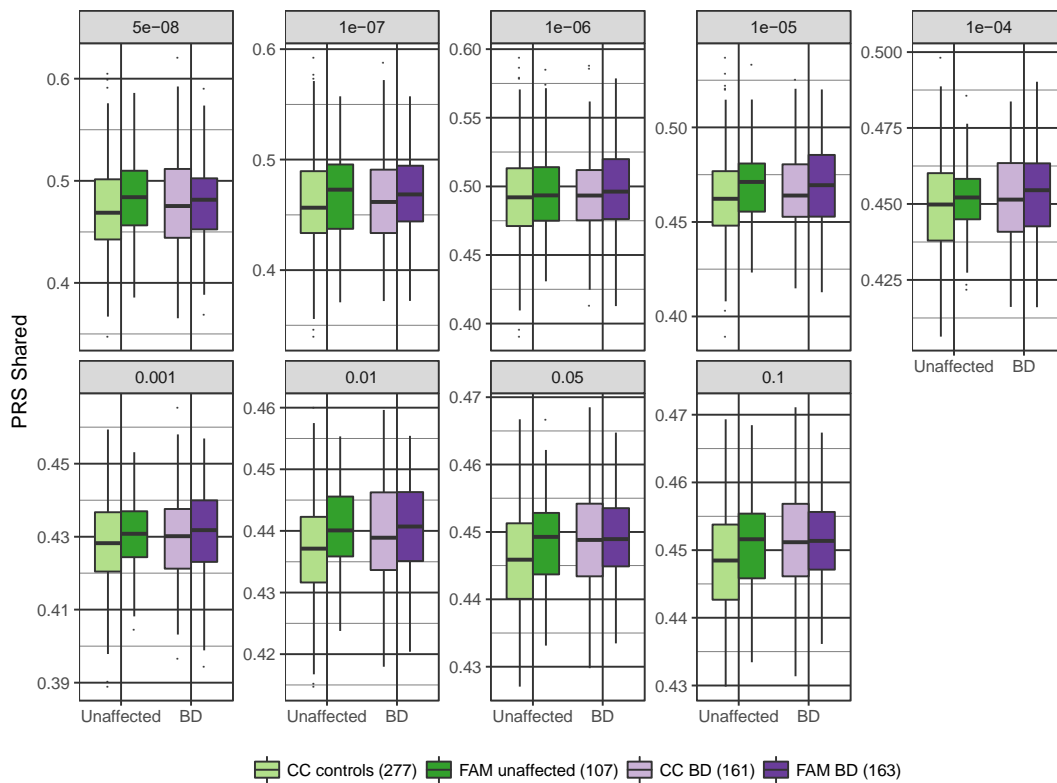
Supplementary Fig. S1B: Boxplots of SCZ PRS.



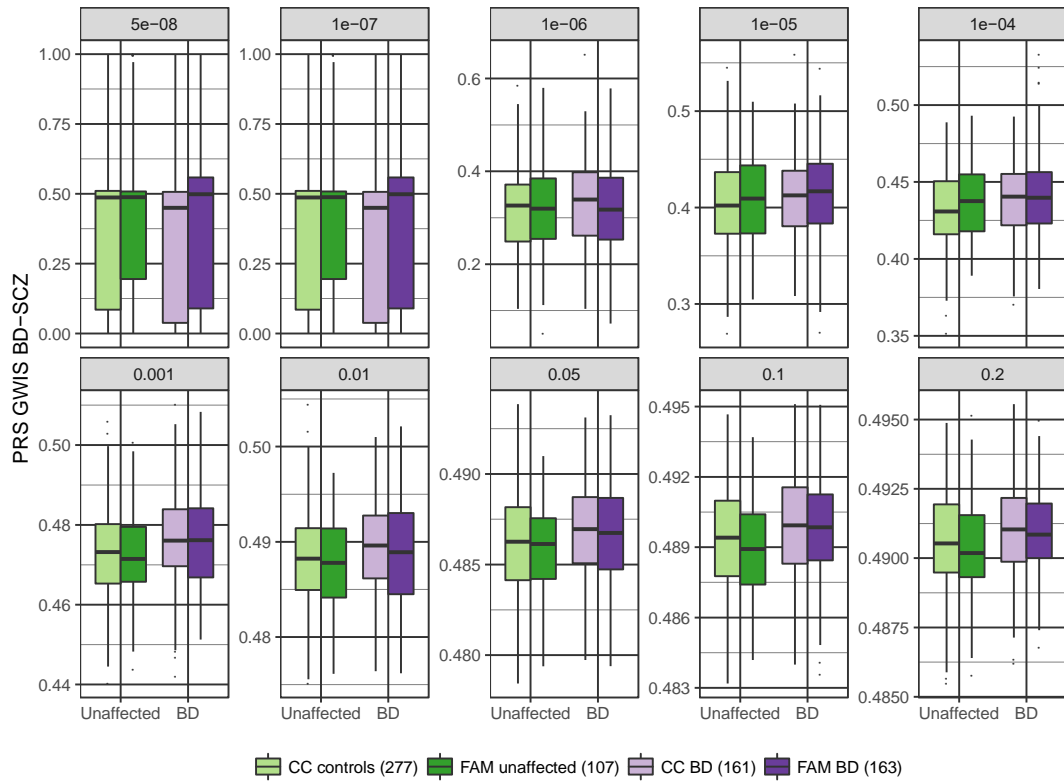
Supplementary Fig. S1C: Boxplots of MDD PRS.



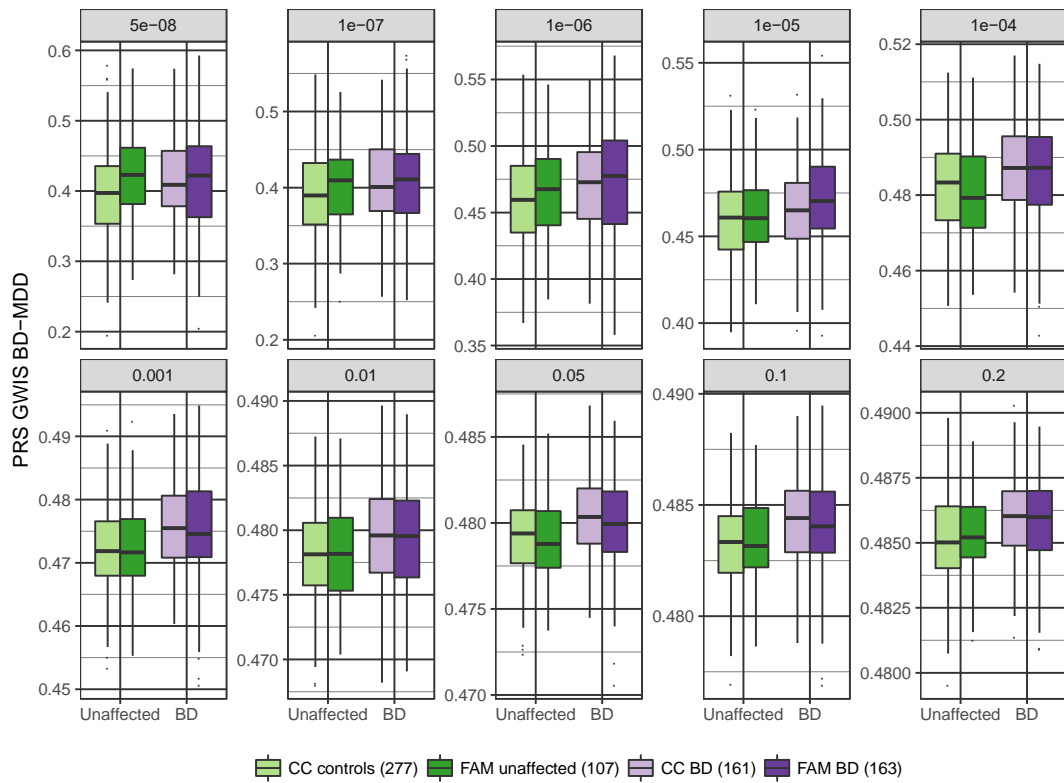
Supplementary Fig. S1D: Boxplots of the BD+SCZ+MDD *Shared* PRS. Note that because of the way this PRS was calculated, the maximum possible threshold was $p_{PRS}=0.1$.



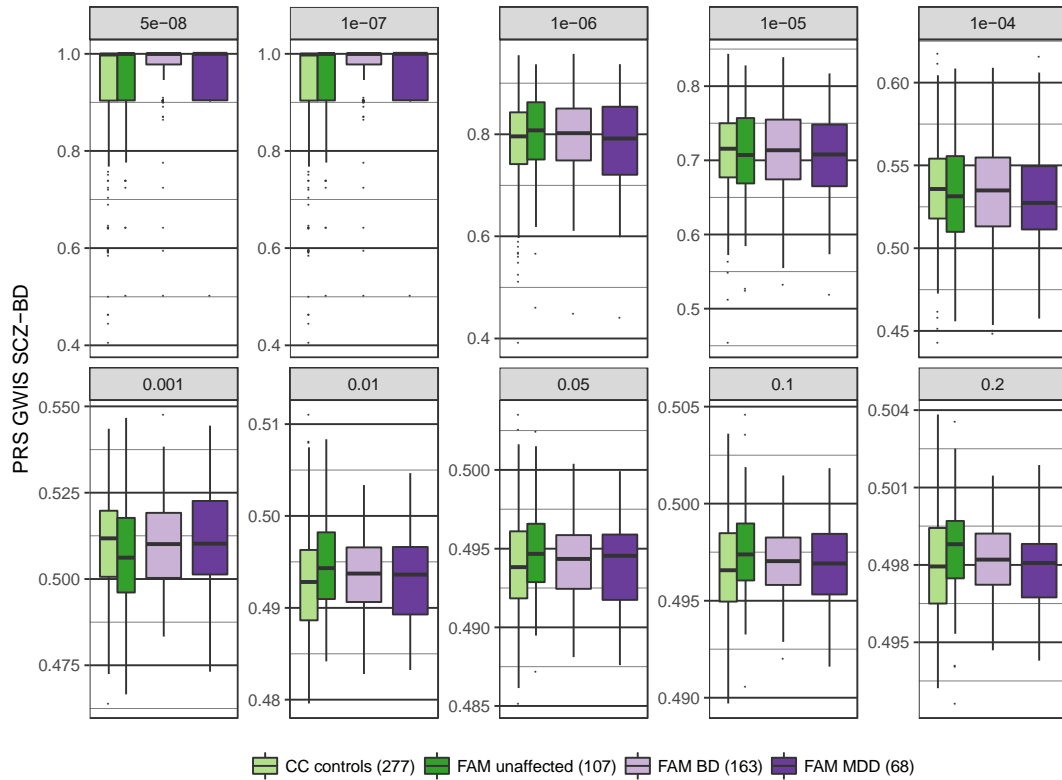
Supplementary Fig. S1E: Boxplots of the BD-SCZ GWIS PRS.



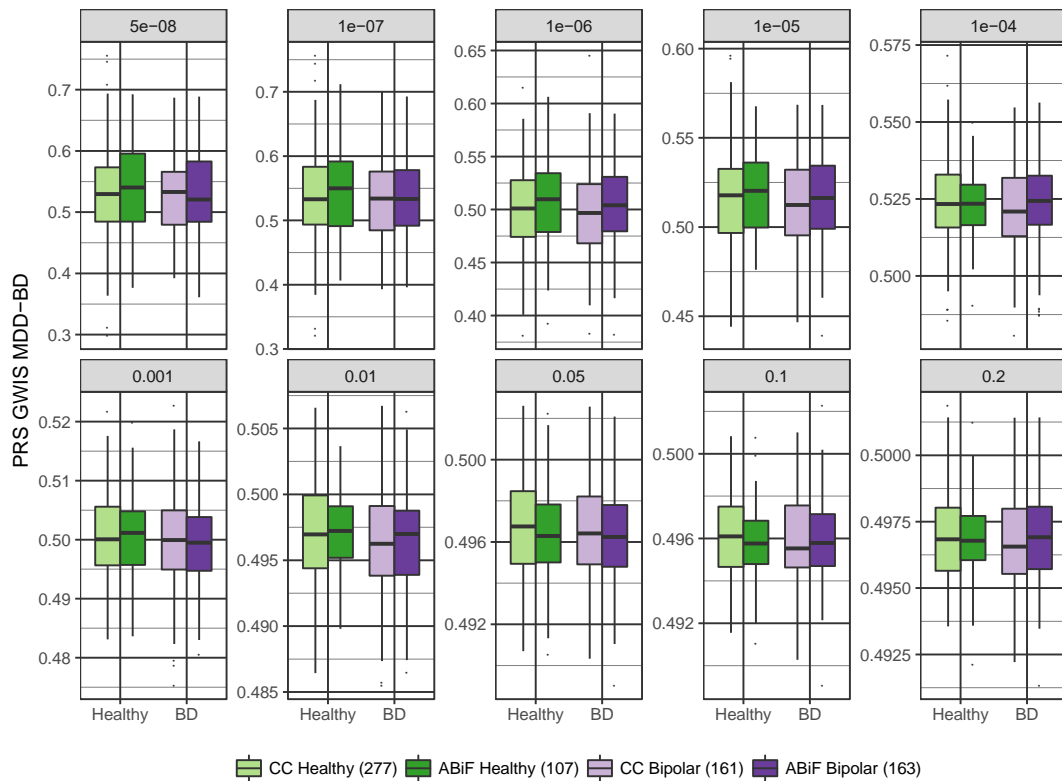
Supplementary Fig. S1F: Boxplots of the BD-MDD GWIS PRS.



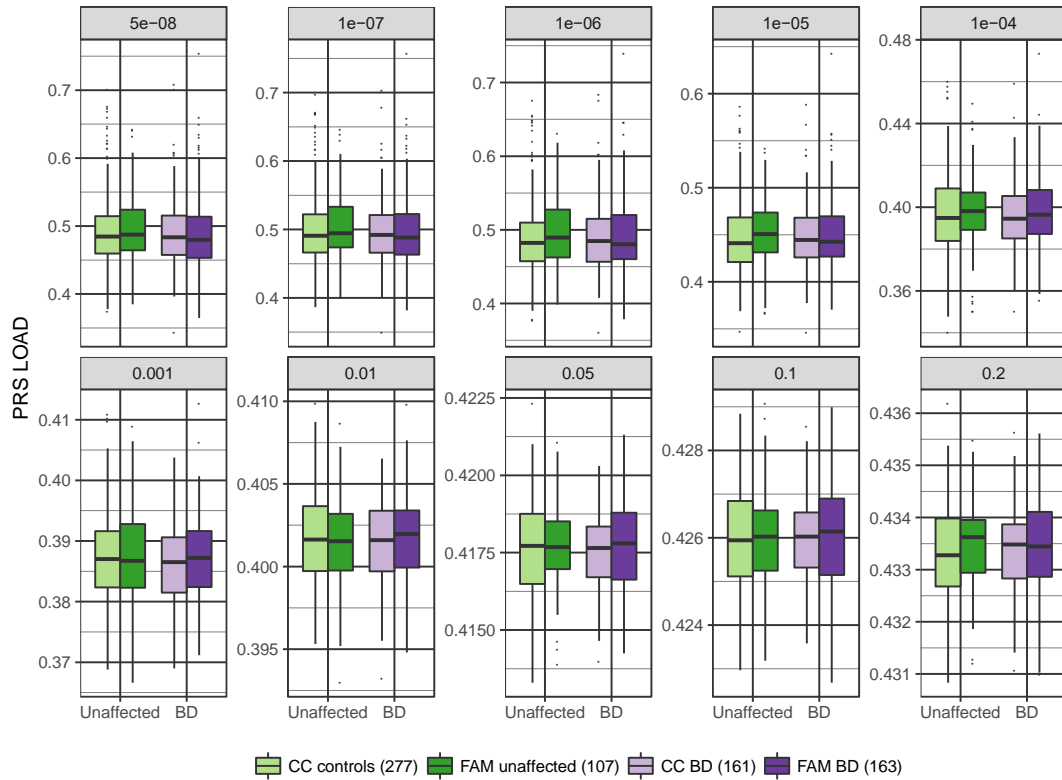
Supplementary Fig. S1G: Boxplots of the SCZ-BD GWIS PRS.



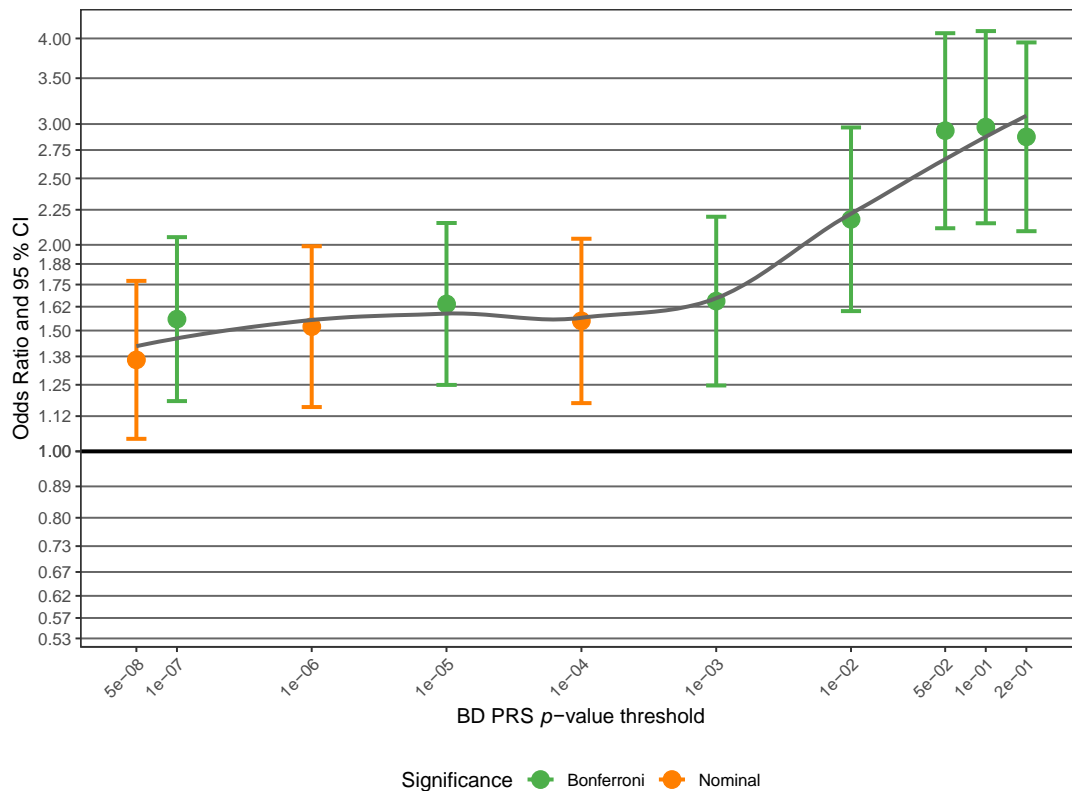
Supplementary Fig. S1H: Boxplots of the MDD-BD GWIS PRS.



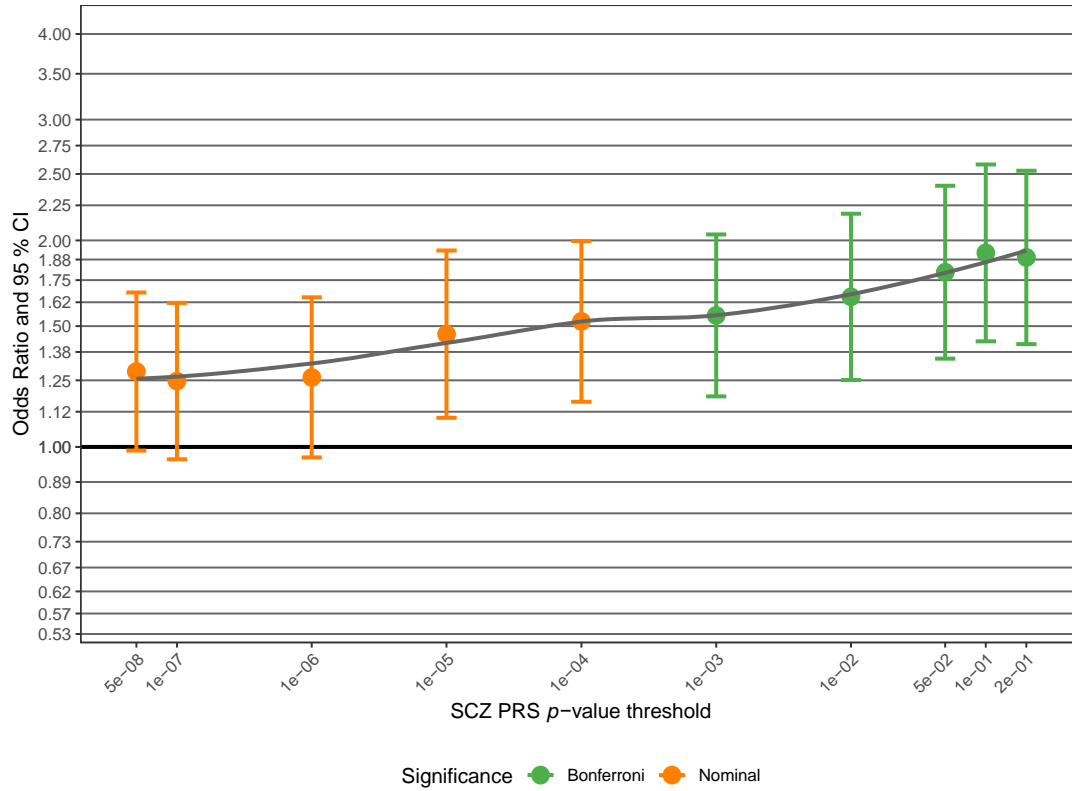
Supplementary Fig. S1I: Boxplots of the LOAD (Alzheimer) PRS.



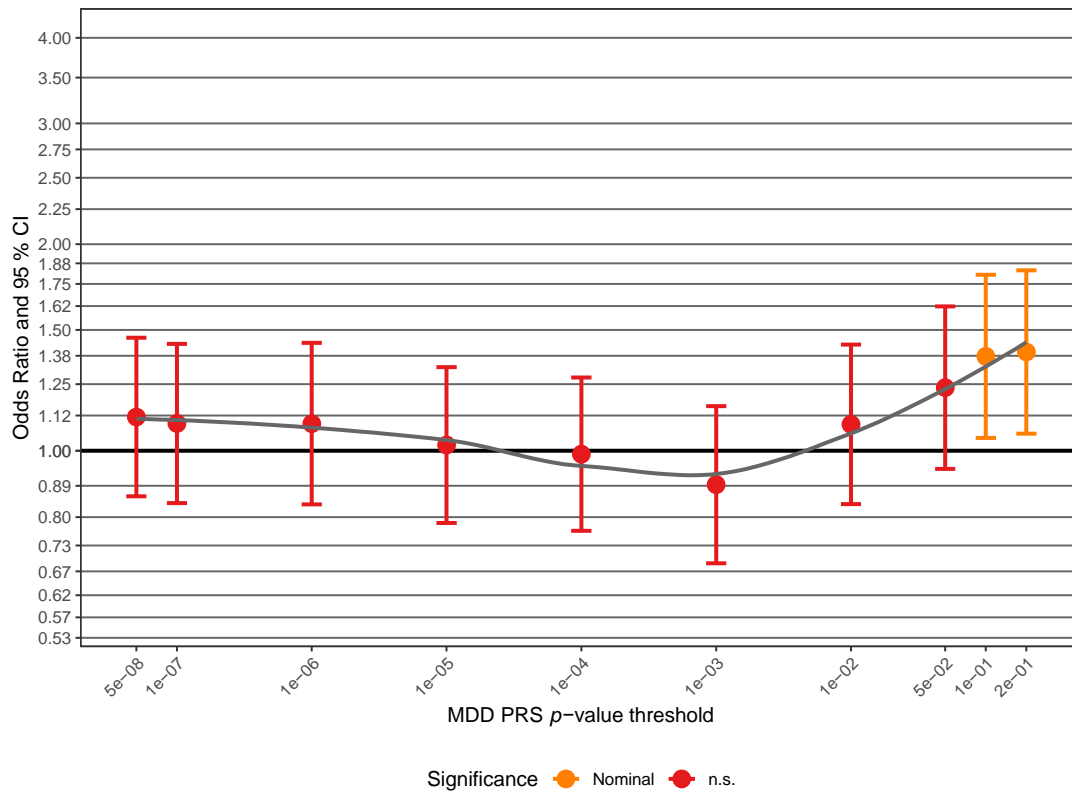
Supplementary Fig. S2: Association analysis comparing PRS in FAM_{BD} cases and CC_{controls}. Further details of the plots are described in the legend for Fig. 1. Full association test statistics including *p*-values are shown in Supplementary Table S3. **Supplementary Fig. S2A: Association of the BD PRS (data is identical to Fig. 1A).**



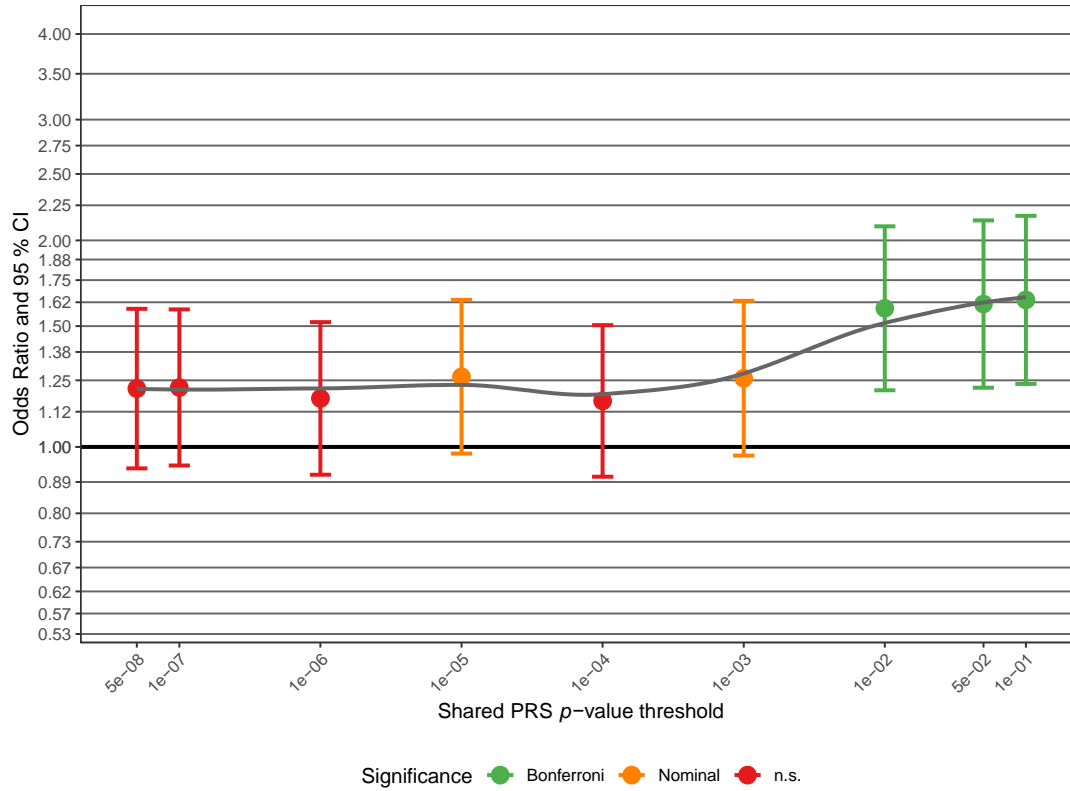
Supplementary Fig. S2B: Association of the SCZ PRS.



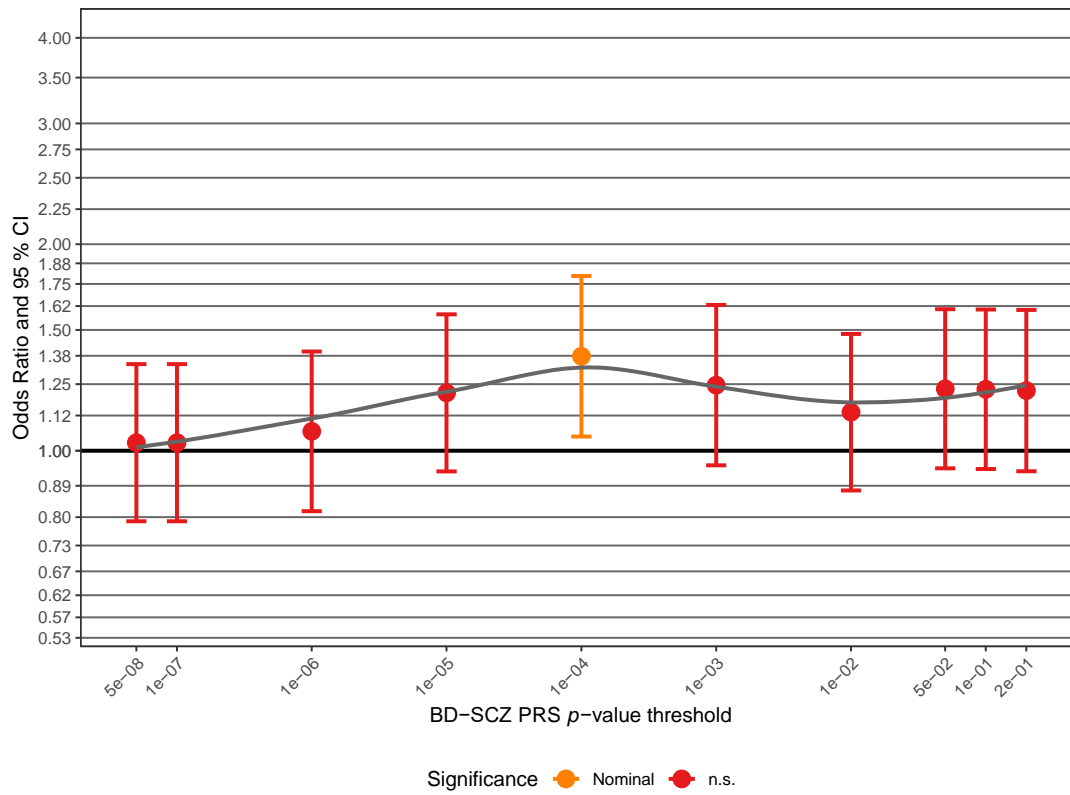
Supplementary Fig. S2C: Association of the MDD PRS.



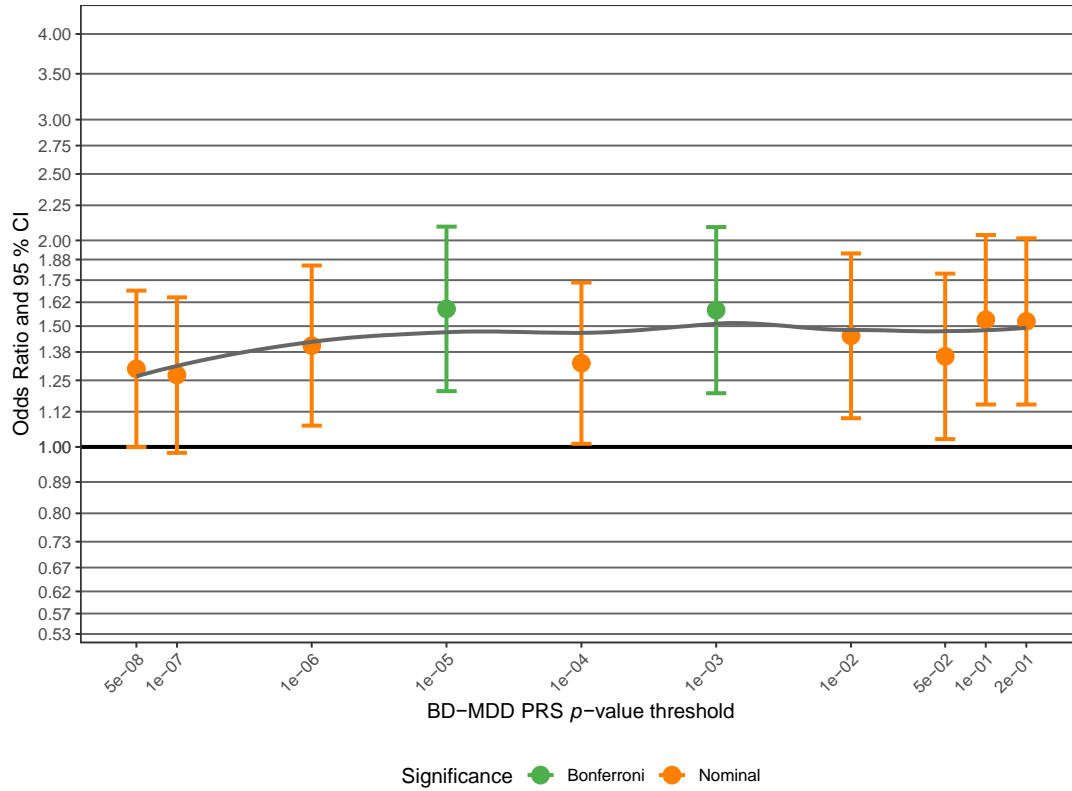
Supplementary Fig. S2D: Association of the *Shared* PRS.



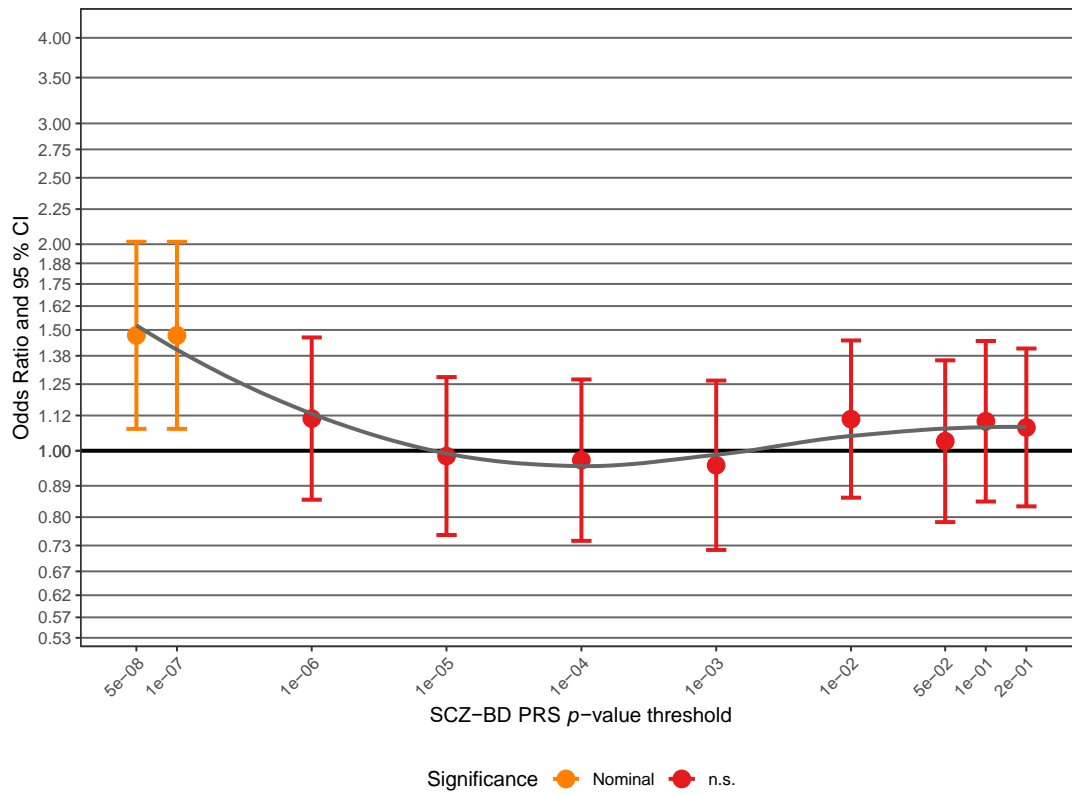
Supplementary Fig. S2E: Association of the BD-SCZ GWIS PRS.



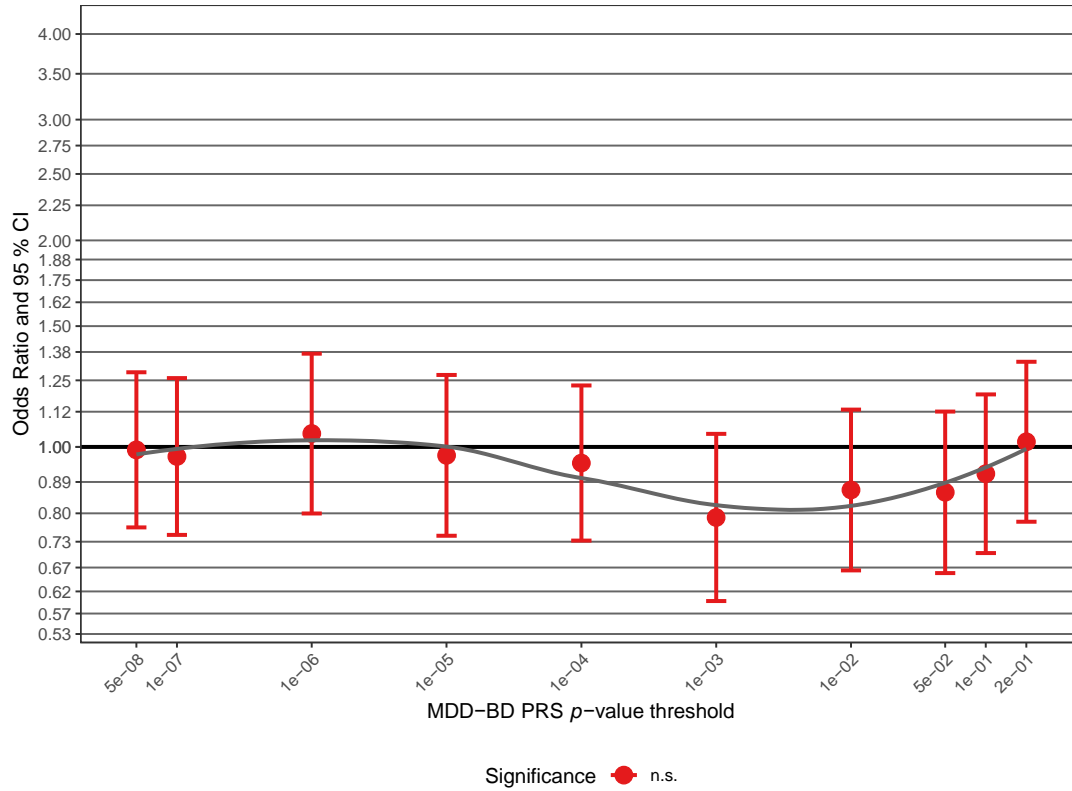
Supplementary Fig. S2F: Association of the BD-MDD GWIS PRS.



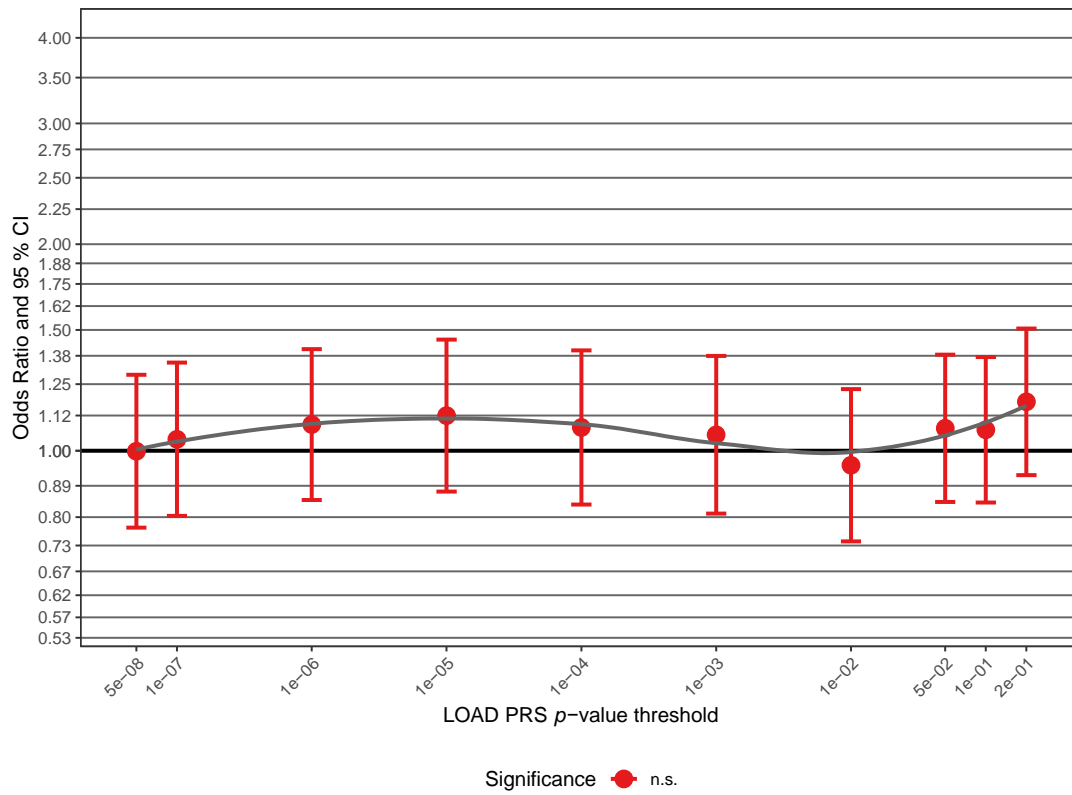
Supplementary Fig. S2G: Association of the SCZ-BD GWIS PRS.



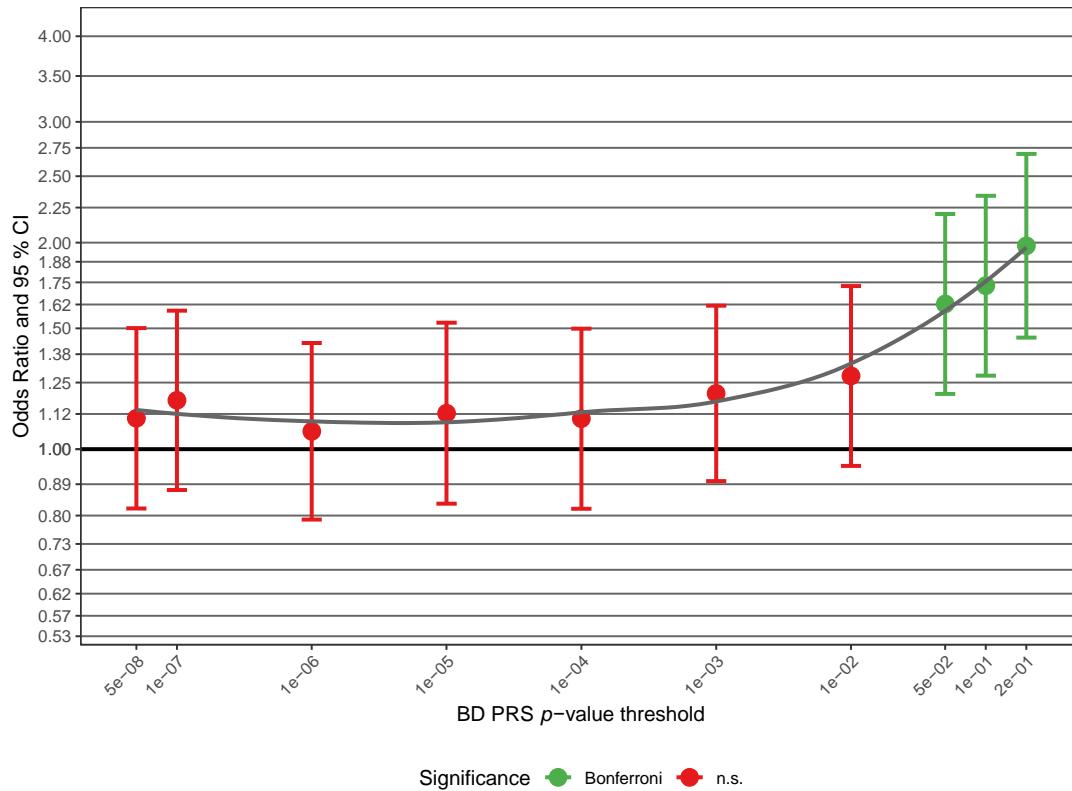
Supplementary Fig. S2H: Association of the MDD-BD GWIS PRS.



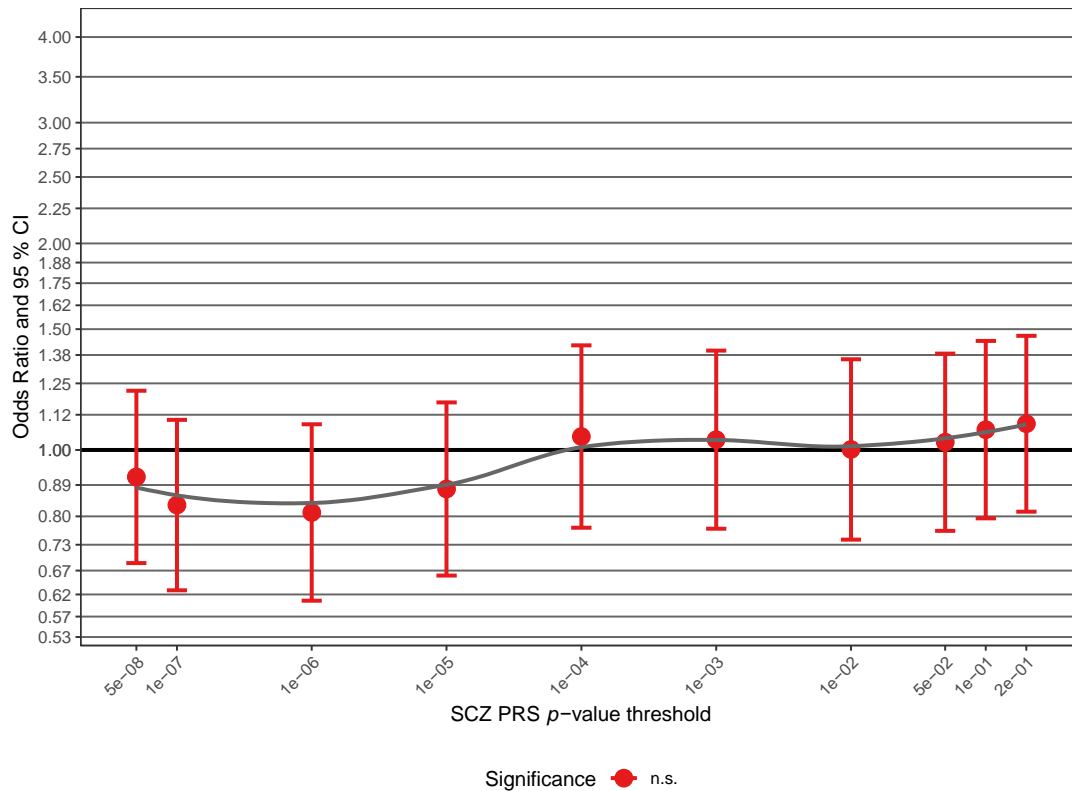
Supplementary Fig. S2I: Association of the LOAD PRS.



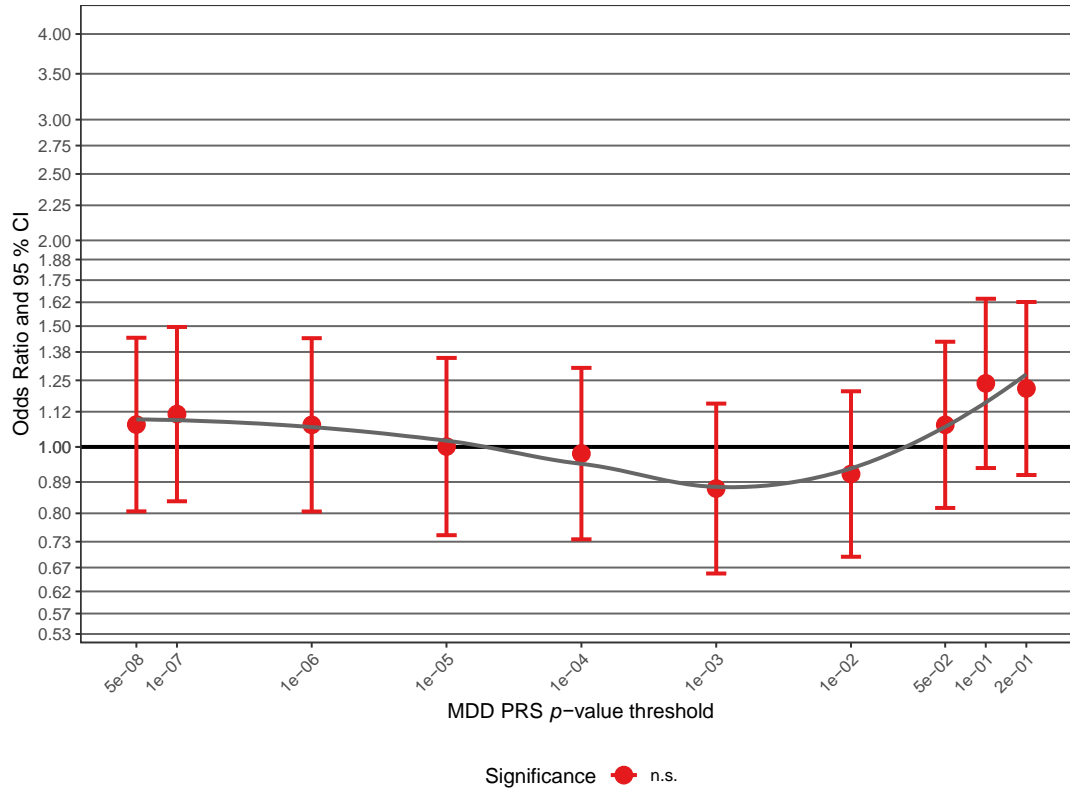
Supplementary Fig. S3: Association analysis comparing PRS in FAM_{BD} cases and unrelated CC_{BD} cases. Further details of the plots are given in the legend for Fig. 1. Full association test statistics including *p*-values are shown in Supplementary Table S4.
Supplementary Fig. S3A: Association of the BD PRS (data is identical to Fig. 1C).



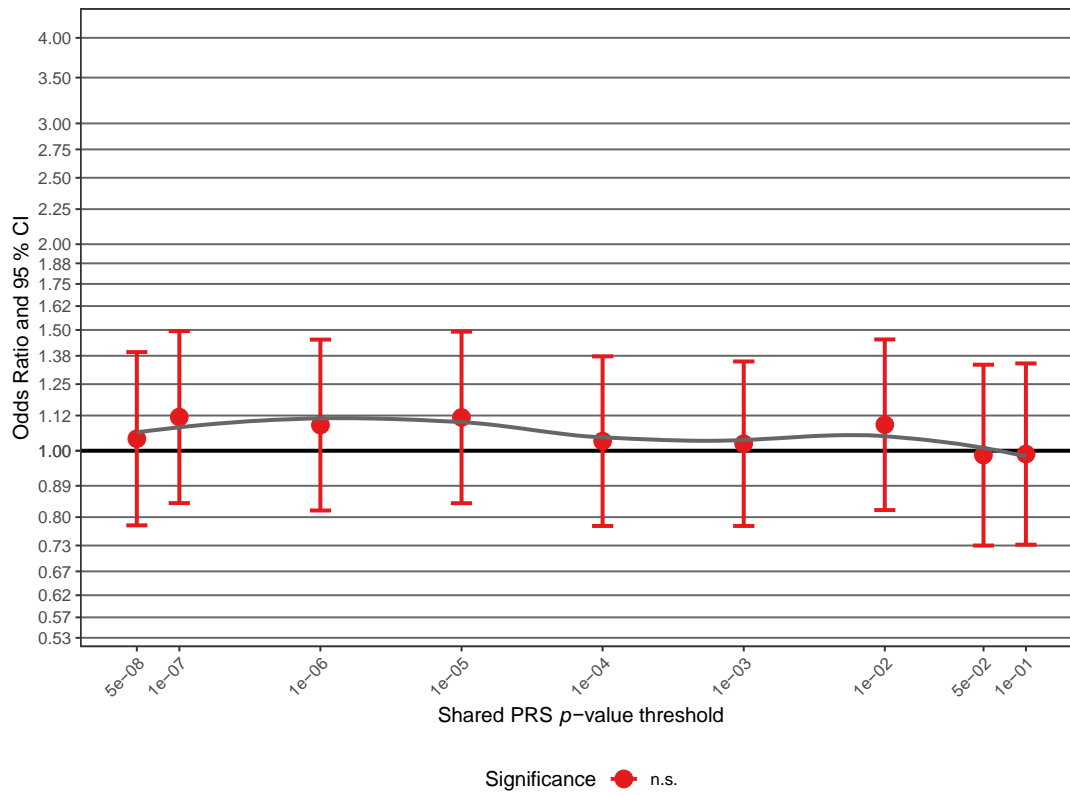
Supplementary Fig. S3B: Association of the SCZ PRS.



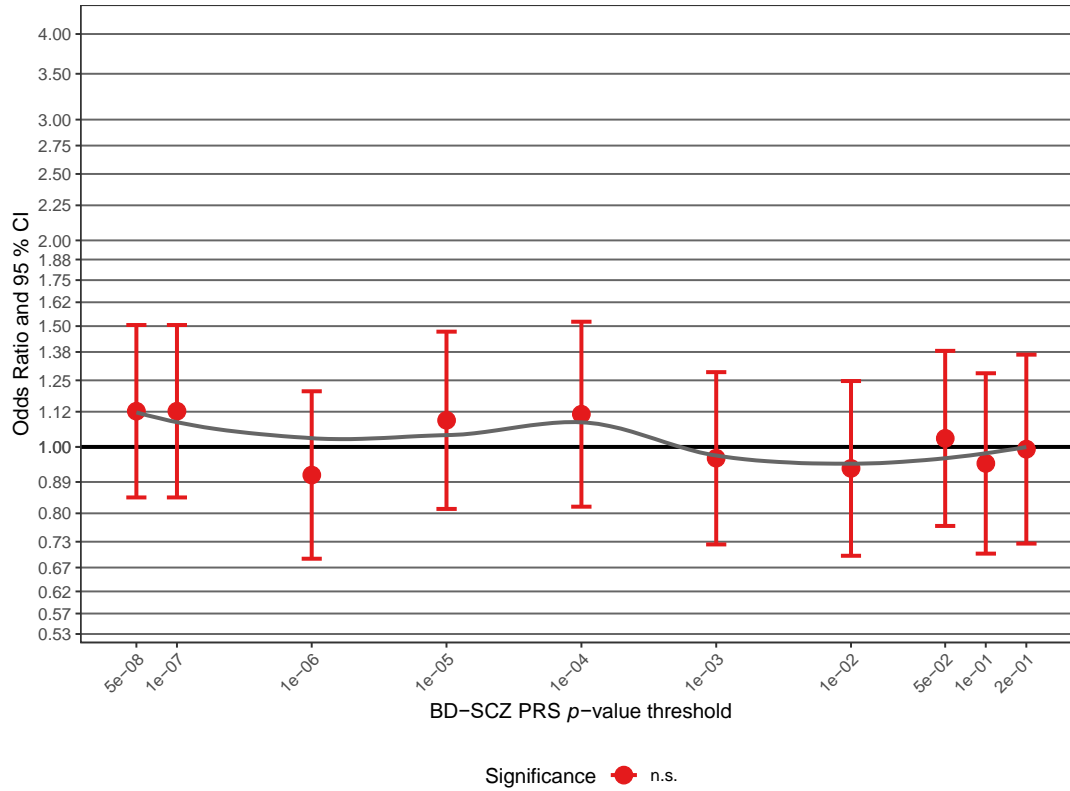
Supplementary Fig. S3C: Association of the MDD PRS.



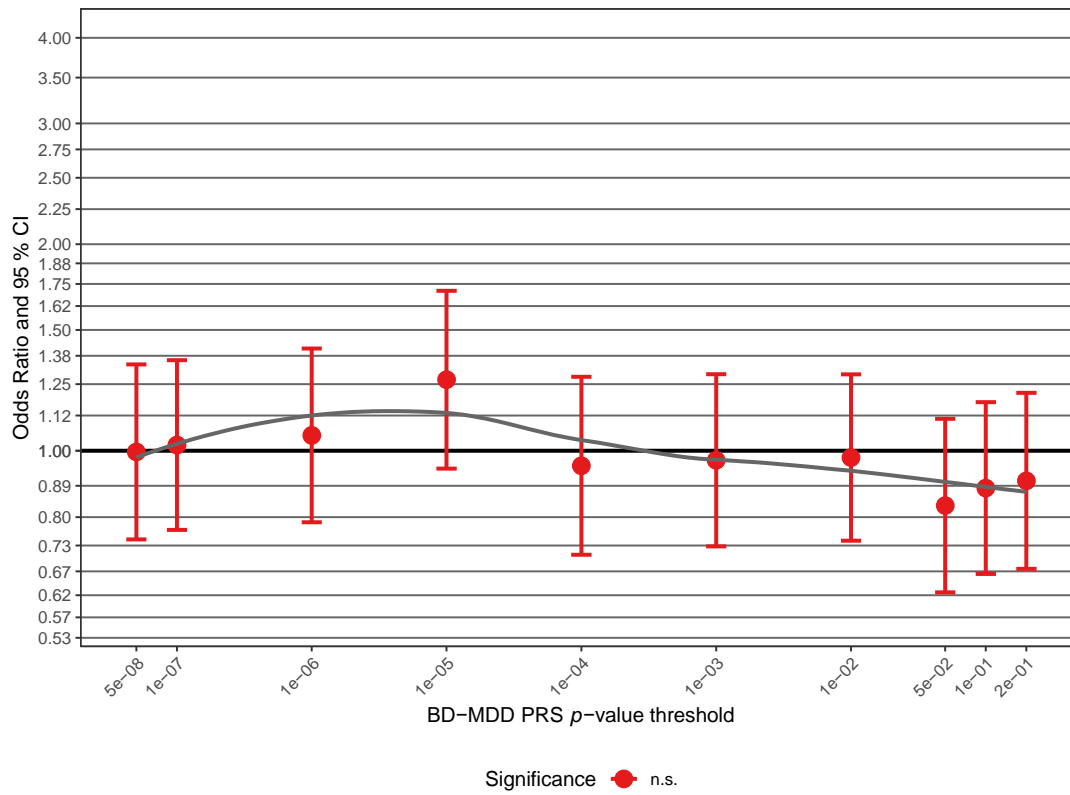
Supplementary Fig. S3D: Association of the Shared PRS.



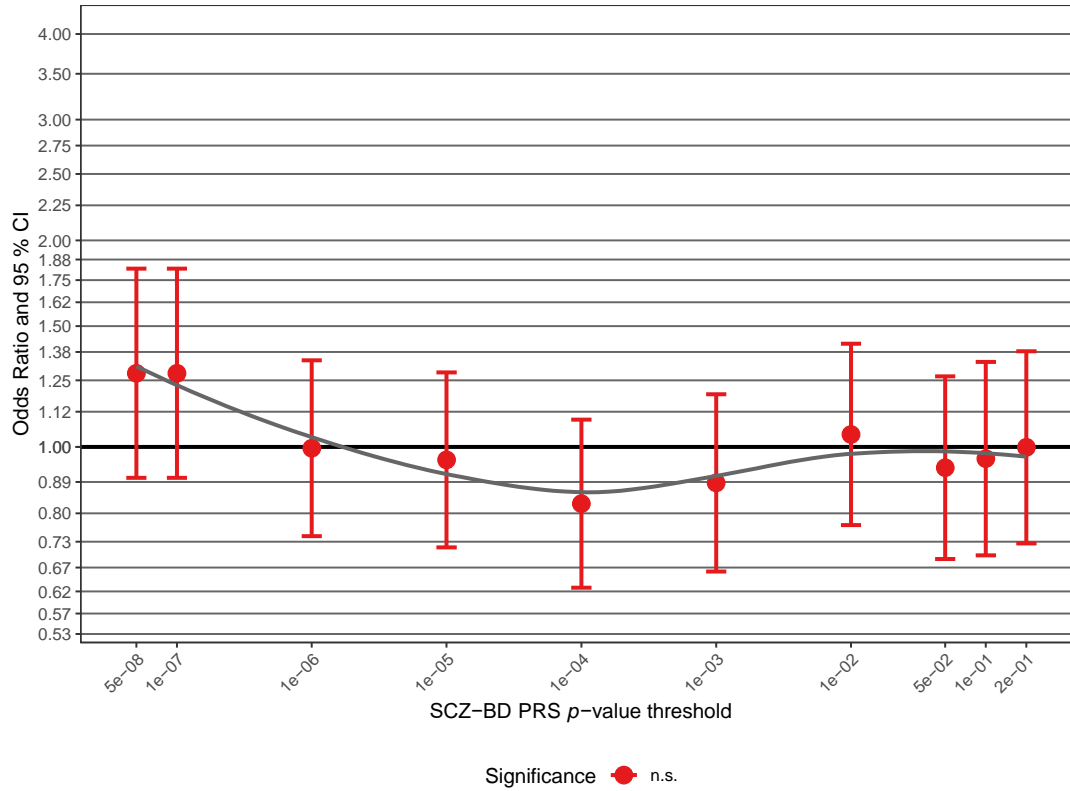
Supplementary Fig. S3E: Association of the BD-SCZ GWIS PRS.



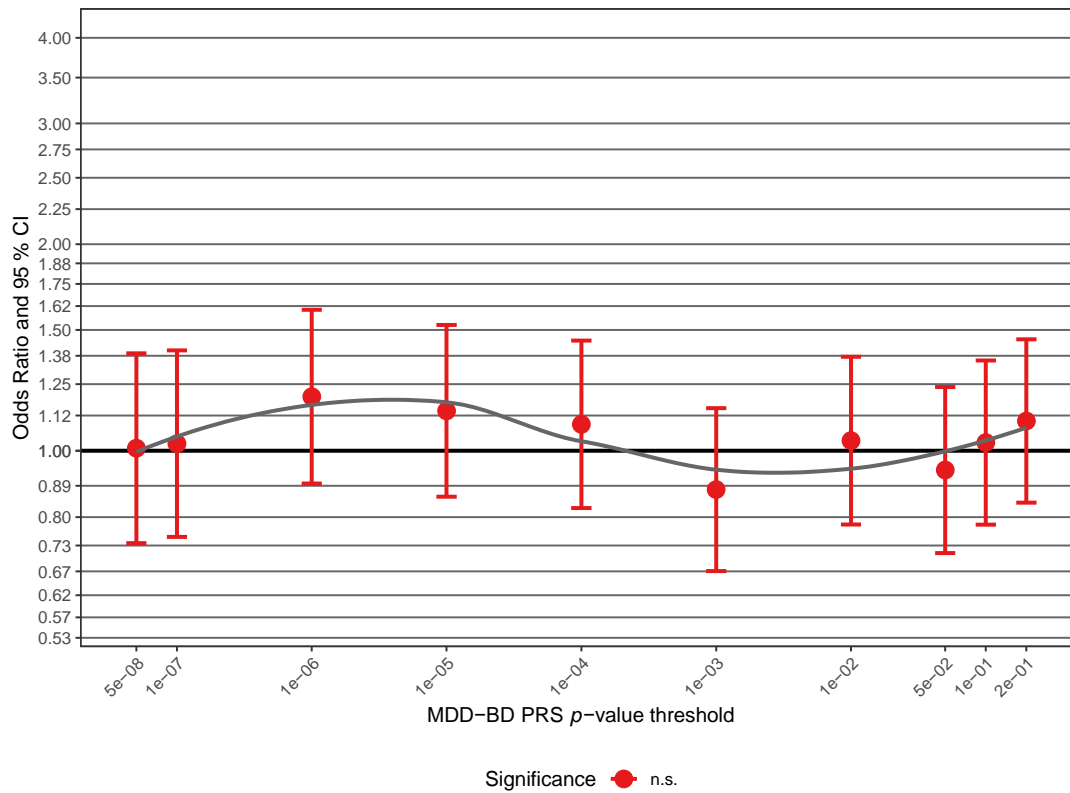
Supplementary Fig. S3F: Association of the BD-MDD GWIS PRS.



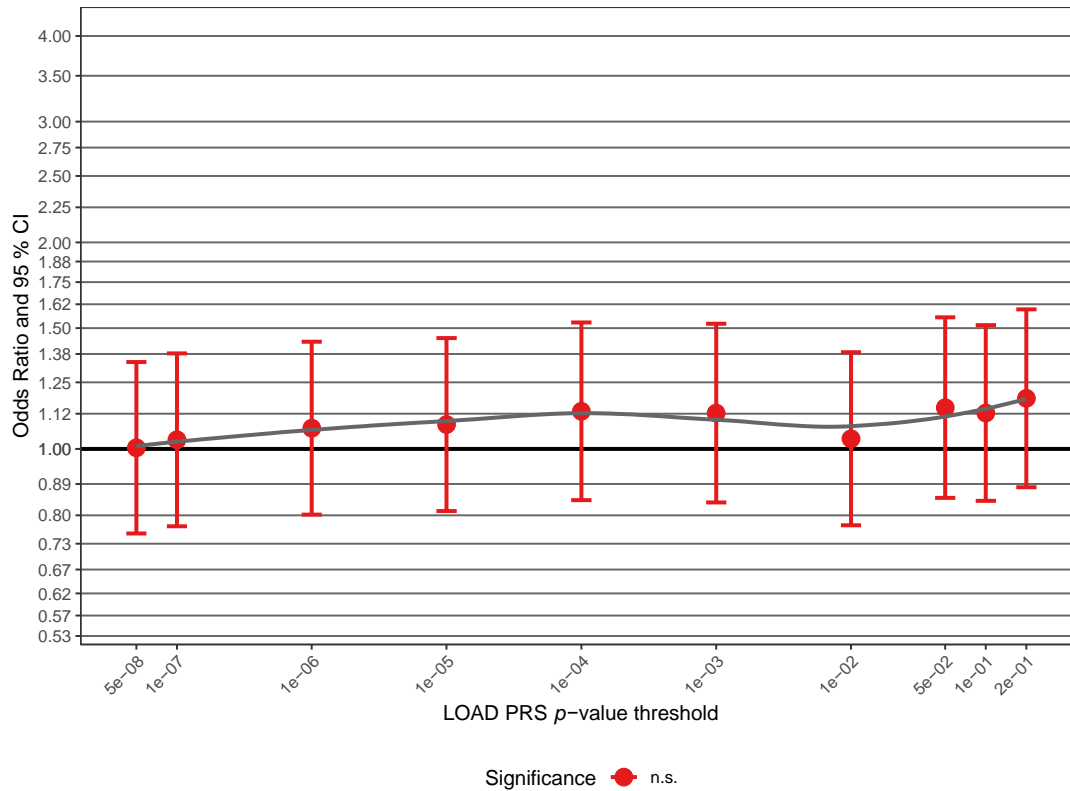
Supplementary Fig. S3G: Association of the SCZ-BD GWIS PRS.



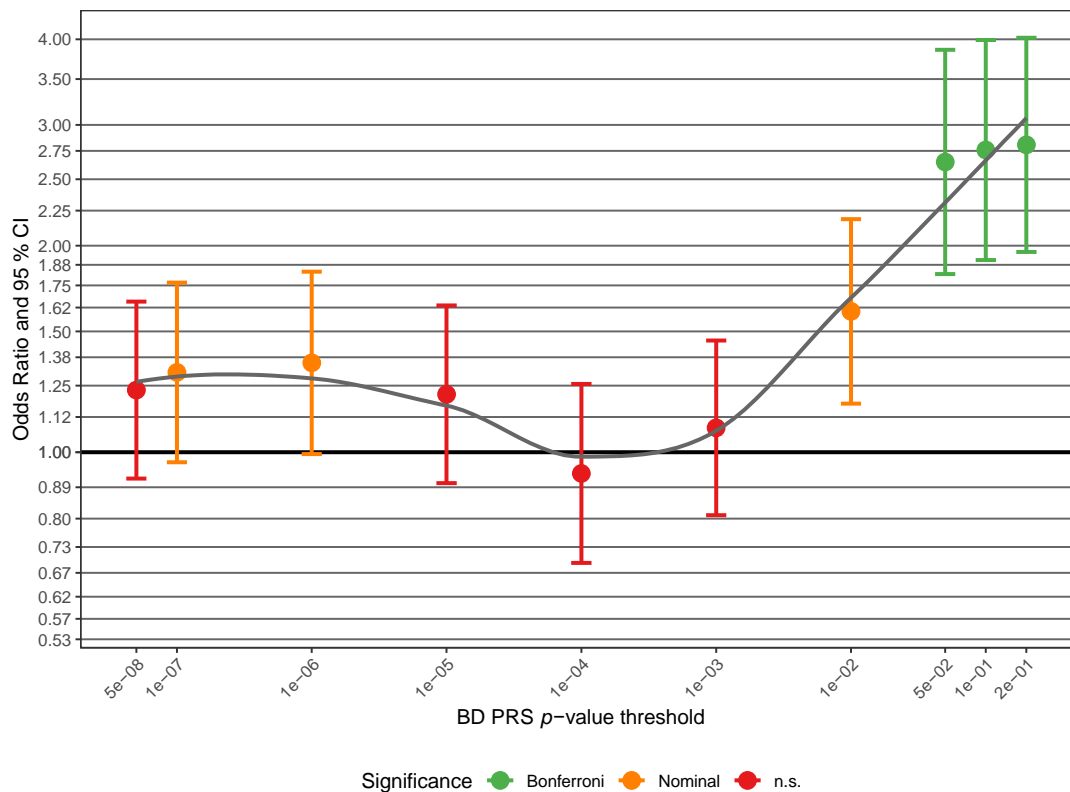
Supplementary Fig. S3H: Association of the MDD-BD GWIS PRS.



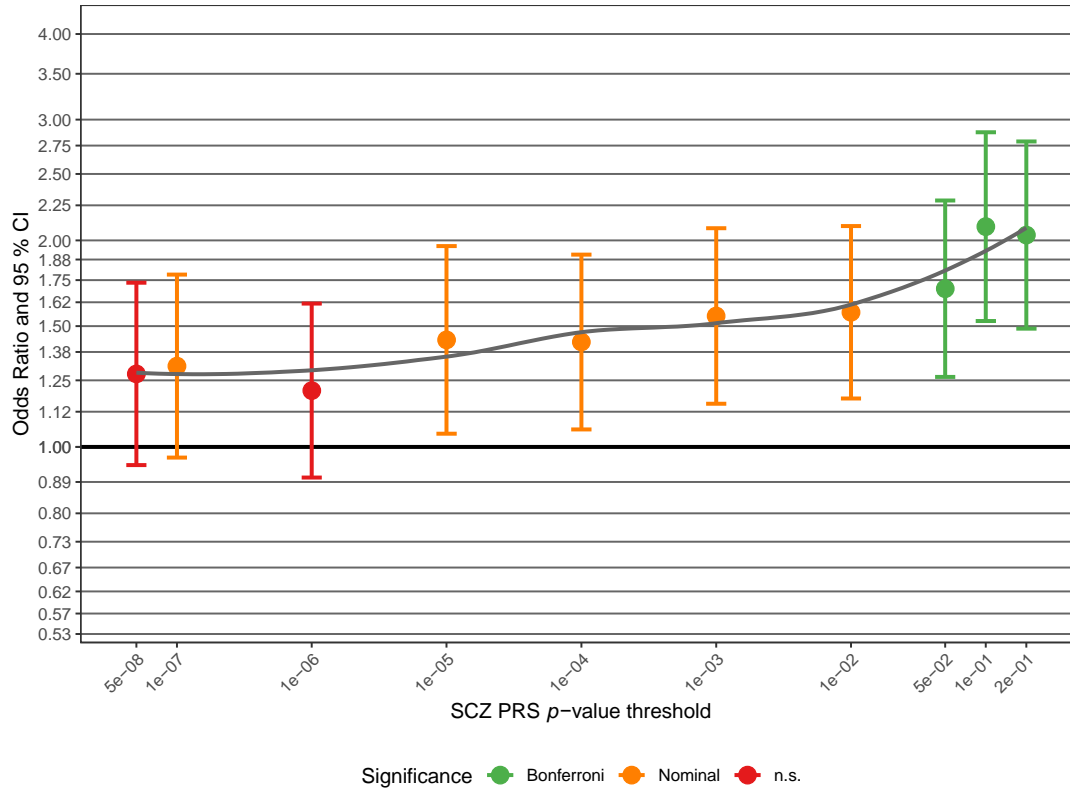
Supplementary Fig. S3I: Association of the LOAD PRS.



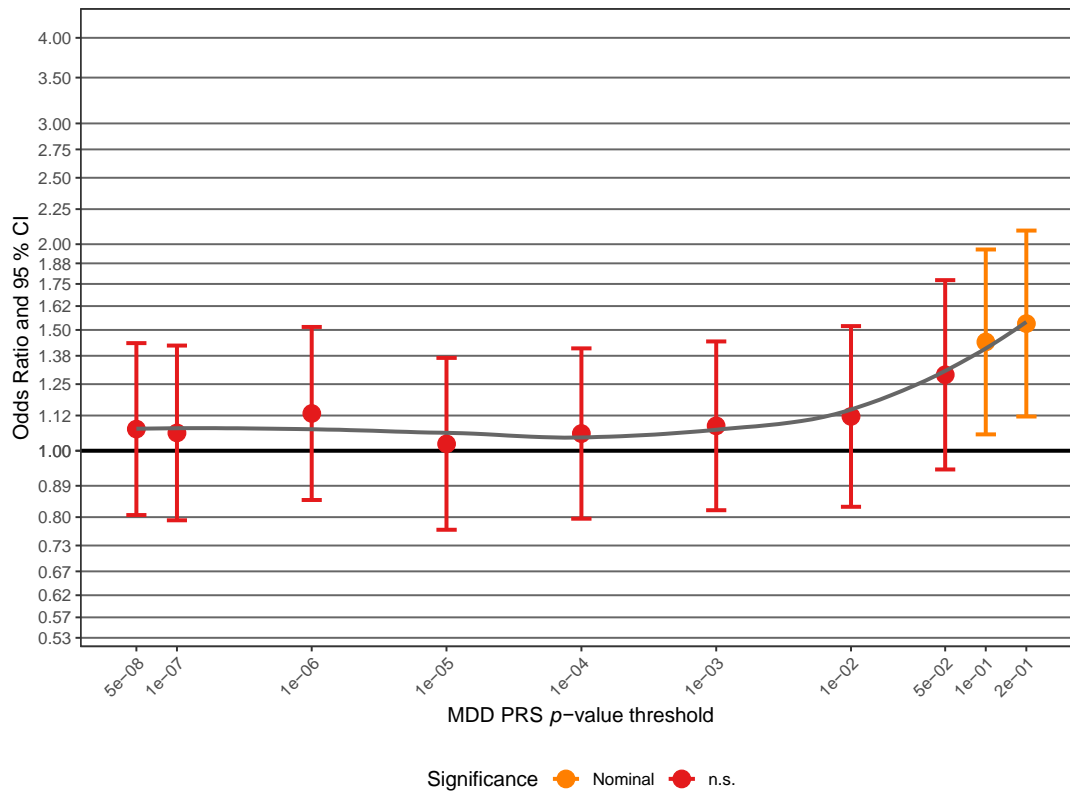
Supplementary Fig. S4: Association analysis comparing PRS in $FAM_{unaffected}$ and $CC_{controls}$. Further details of the plots are described in the legend for Fig. 1. Full association test statistics including p -values are shown in Supplementary Table S5.
Supplementary Fig. S4A: Association of the BD PRS (data is identical to Fig. 1E).



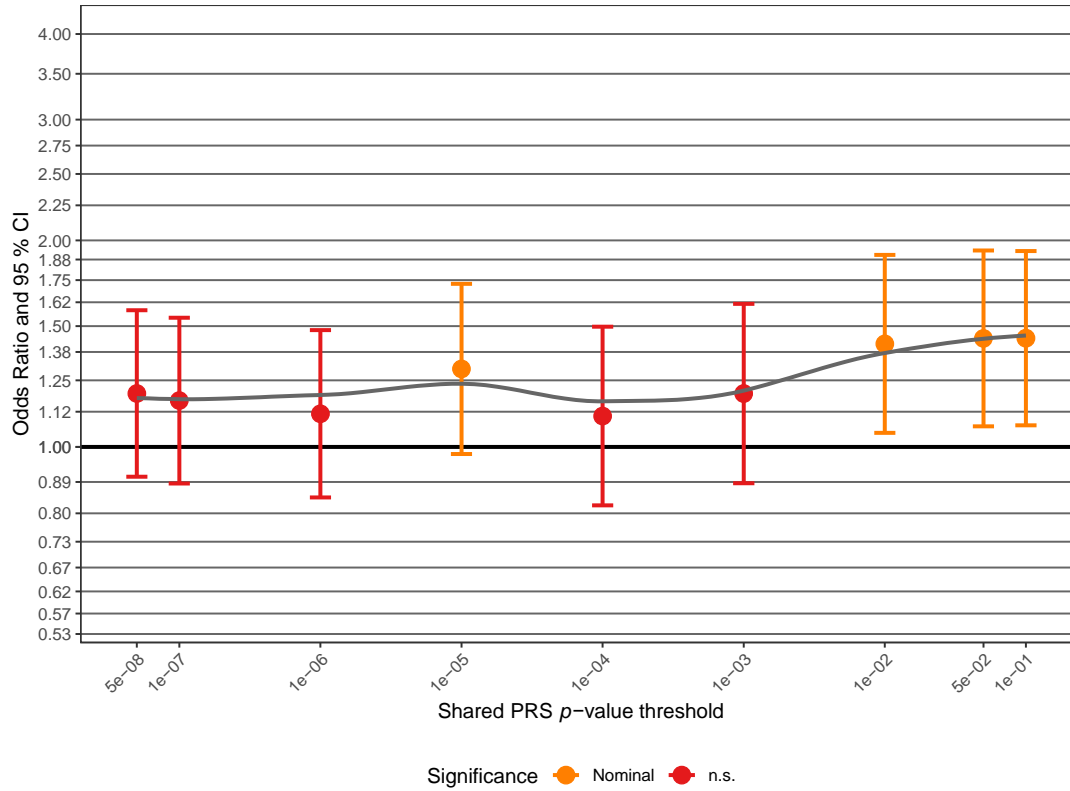
Supplementary Fig. S4B: Association of the SCZ PRS.



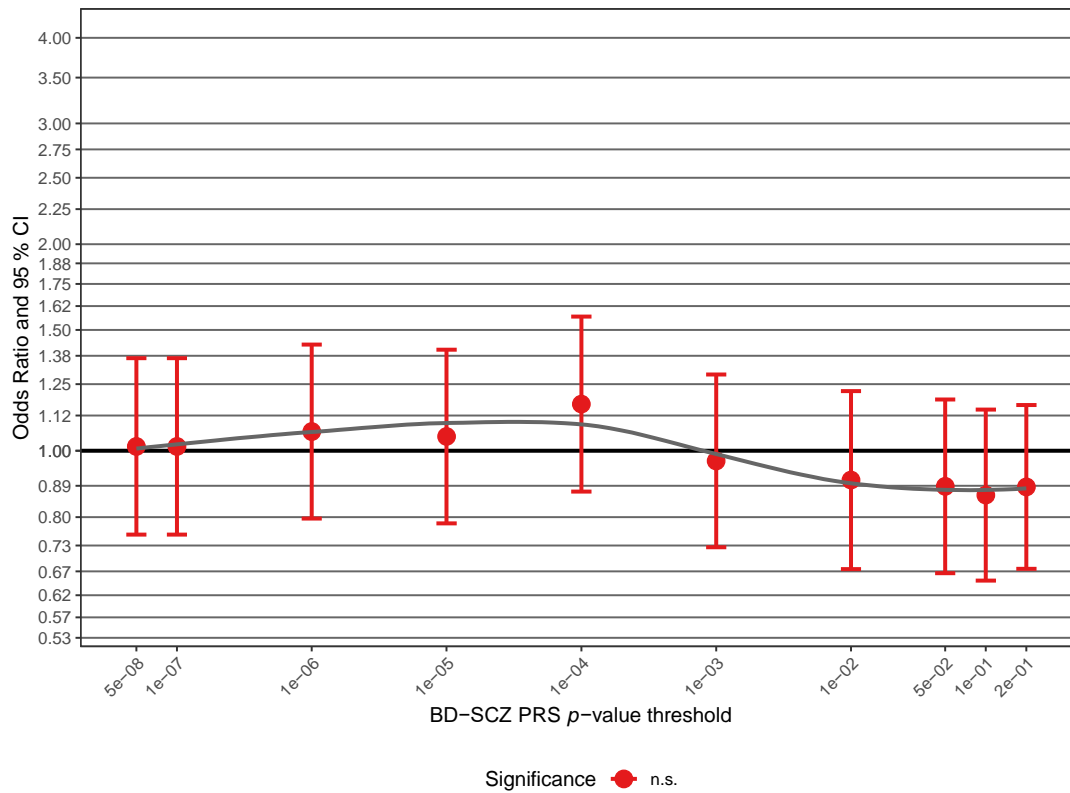
Supplementary Fig. S4C: Association of the MDD PRS.



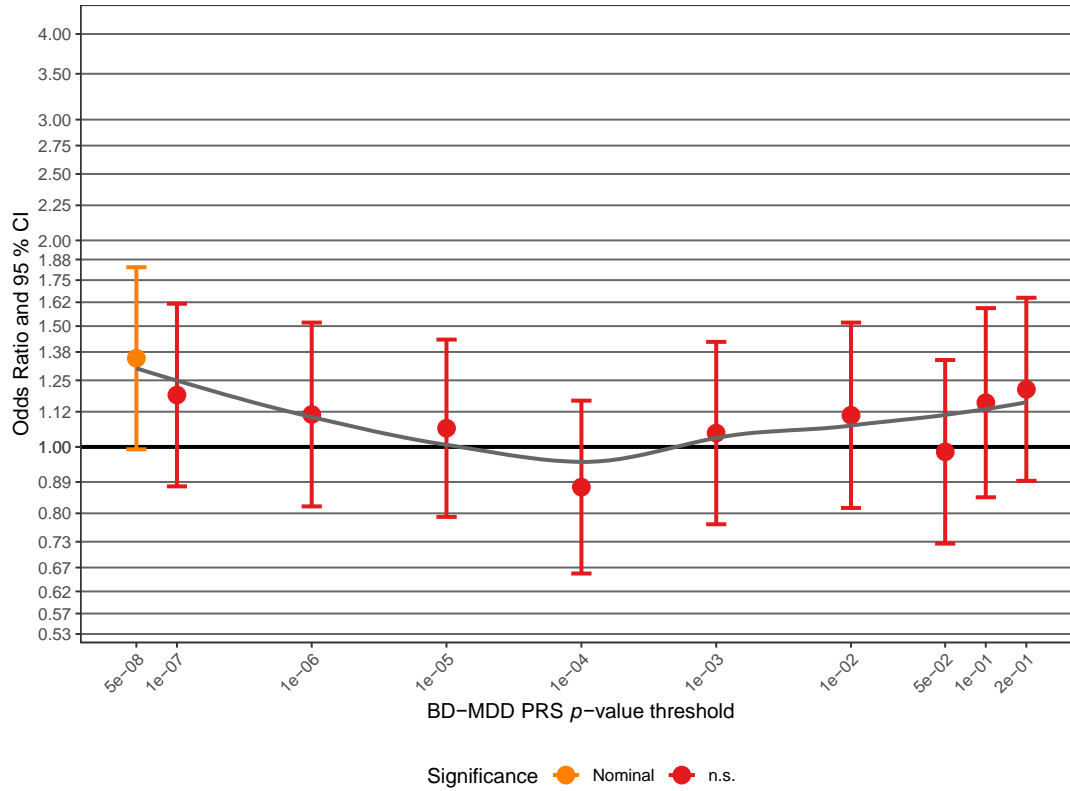
Supplementary Fig. S4D: Association of the *Shared* PRS.



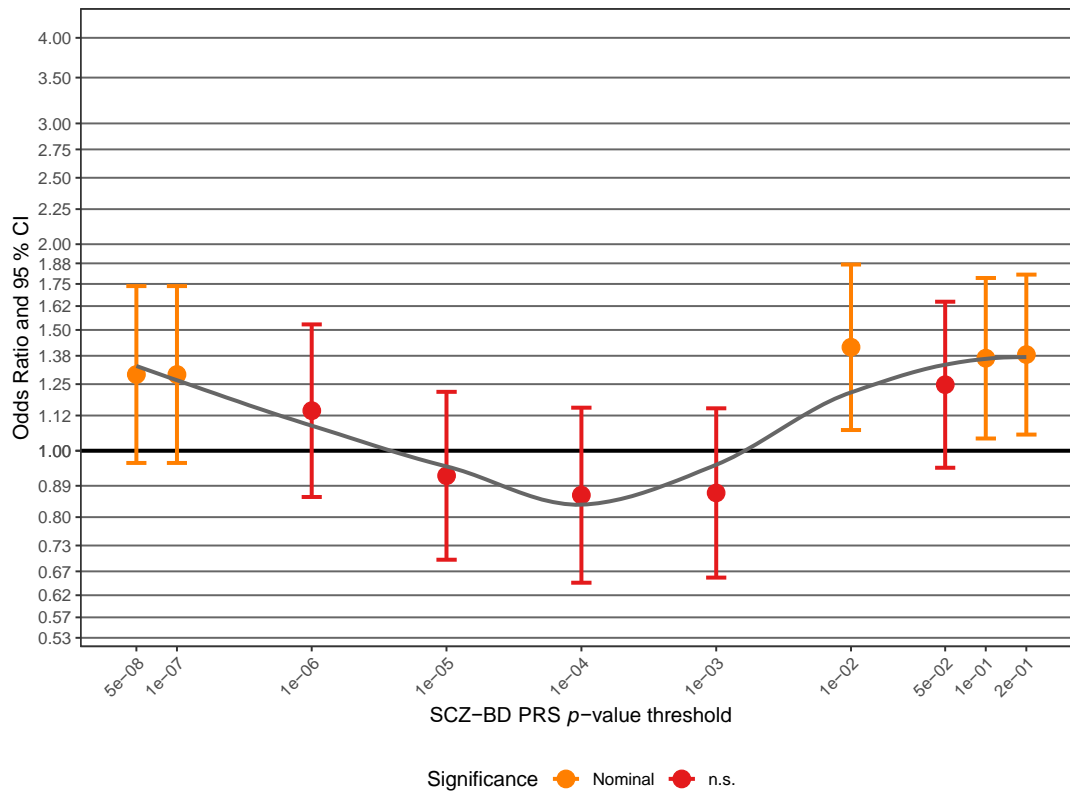
Supplementary Fig. S4E: Association of the BD-SCZ GWIS PRS.



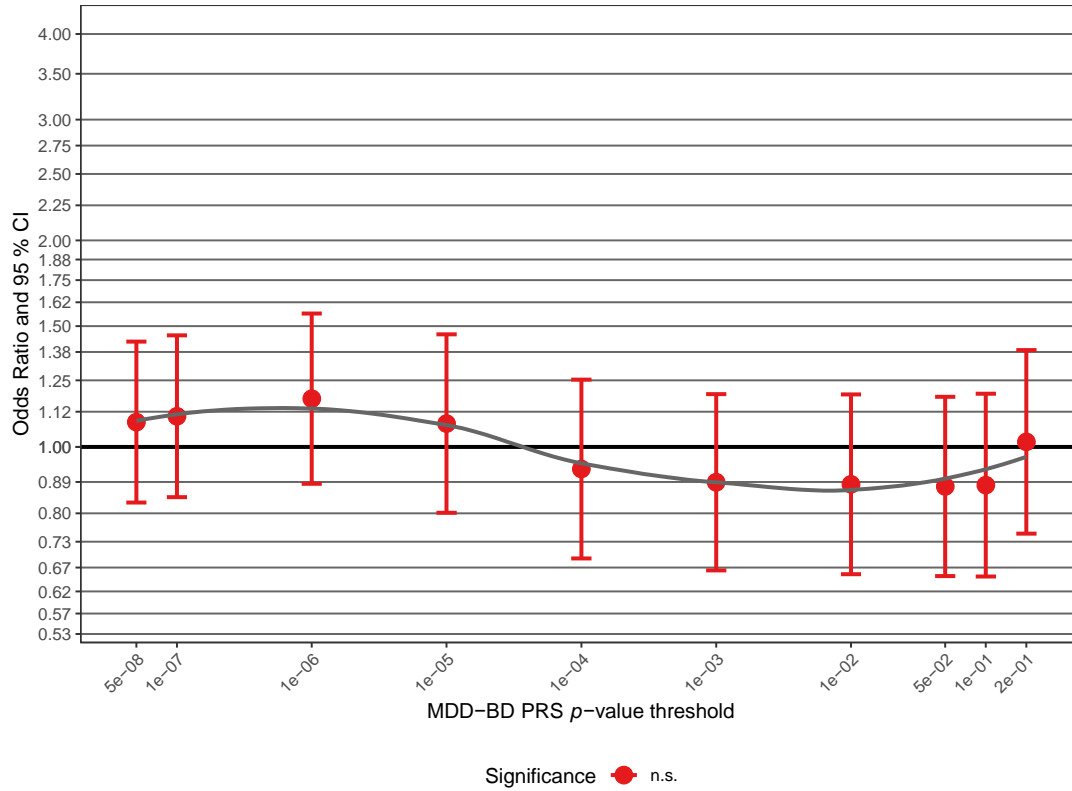
Supplementary Fig. S4F: Association of the BD-MDD GWIS PRS.



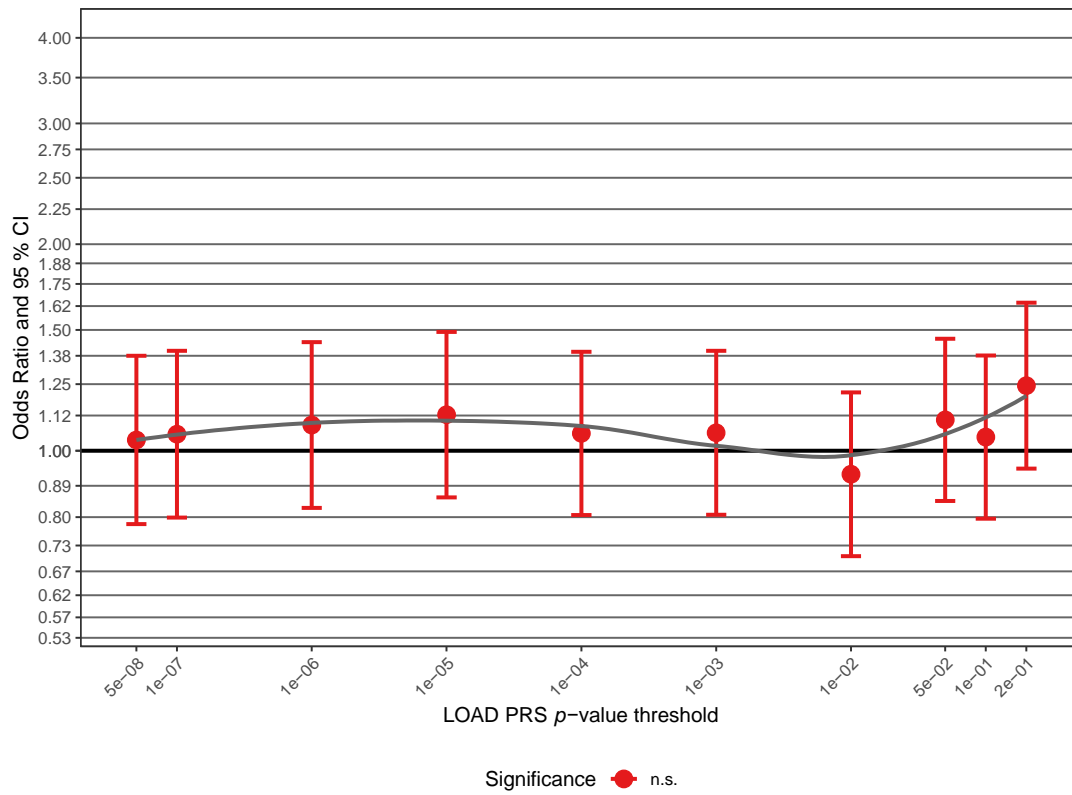
Supplementary Fig. S4G: Association of the SCZ-BD GWIS PRS.



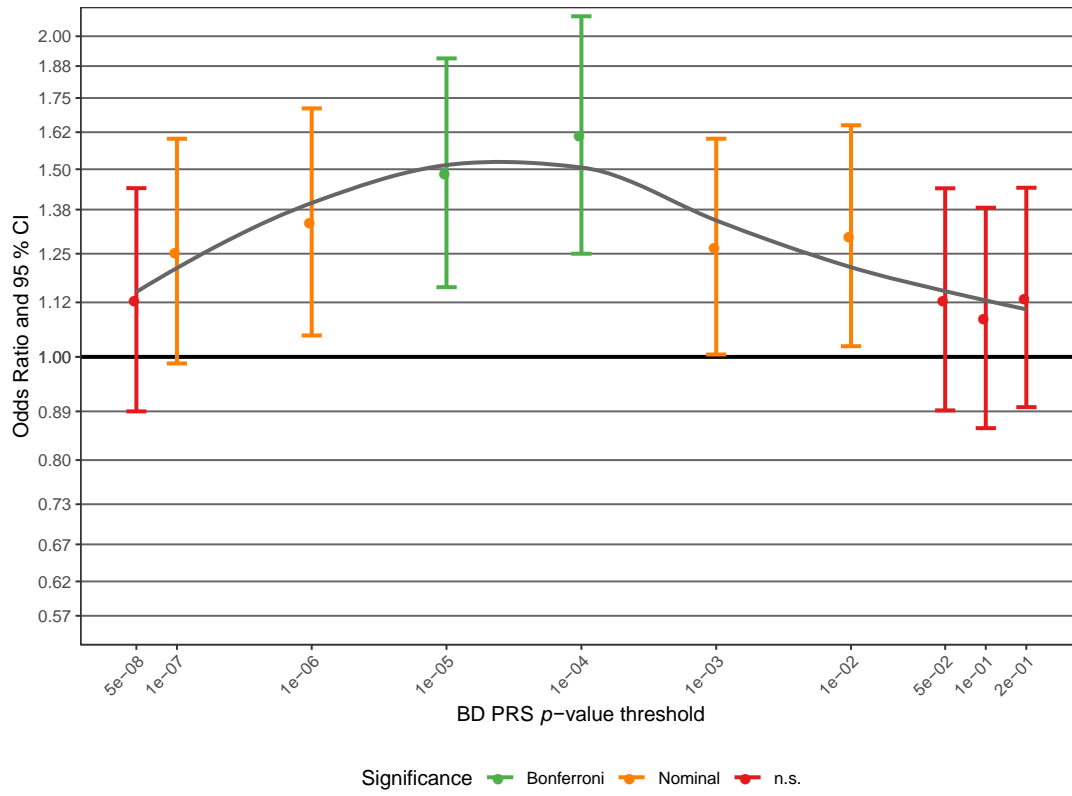
Supplementary Fig. S4H: Association of the MDD-BD GWIS PRS.



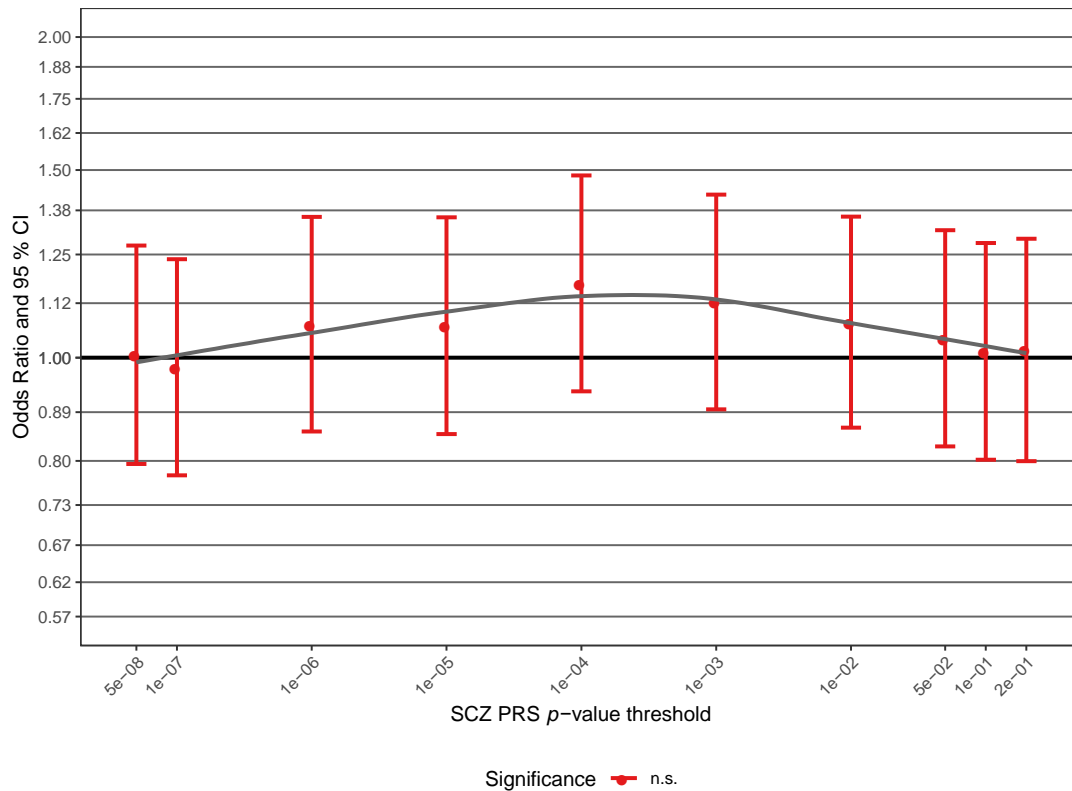
Supplementary Fig. S4I: Association of the LOAD PRS.



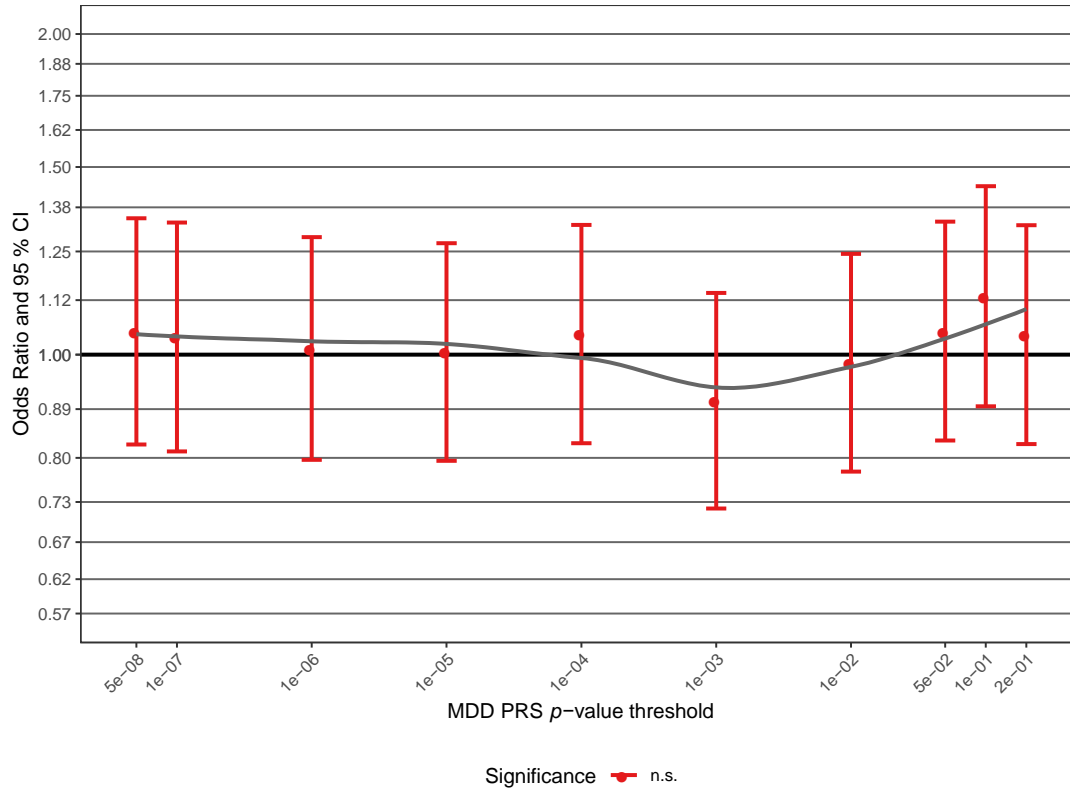
Supplementary Fig. S5: Association analysis comparing PRS in FAM_{BD} cases and FAM_{unaffected}. Further details of the plots are given in the legends for Figs. 1 and 2. Full association test statistics including *p*-values are shown in Supplementary Table S6.
Supplementary Fig. S5A: Association of the BD PRS (data is identical to Fig. 2A).



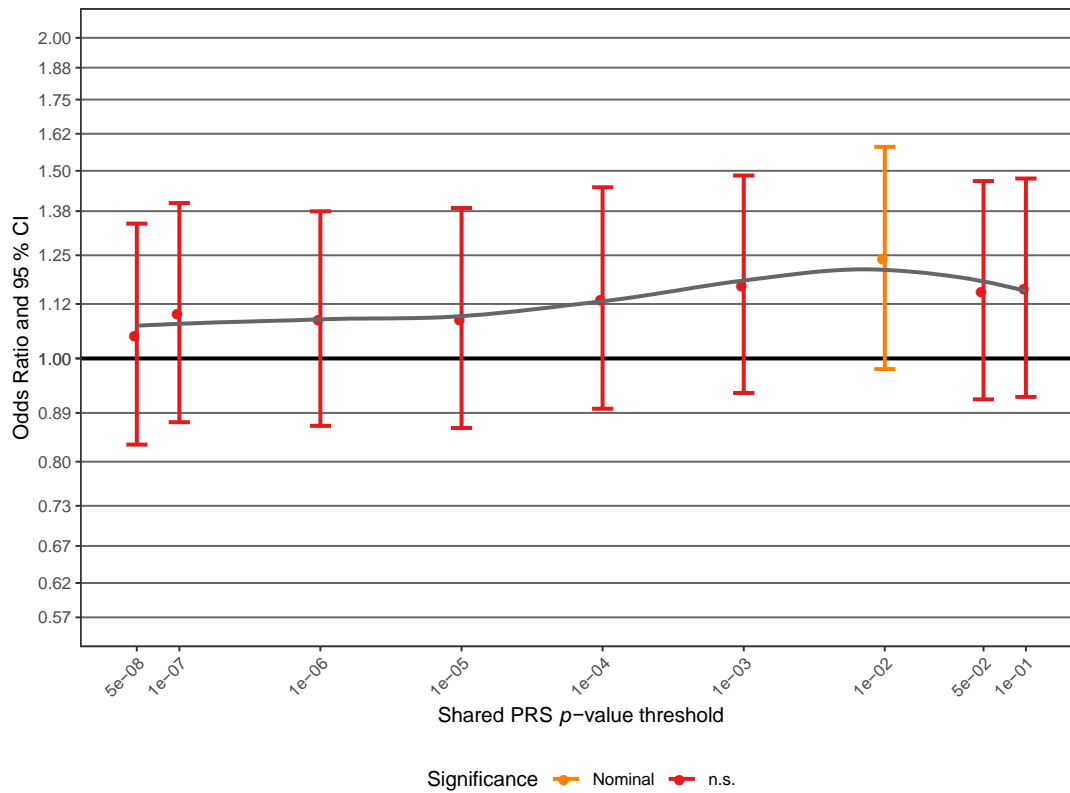
Supplementary Fig. S5B: Association of the SCZ PRS.



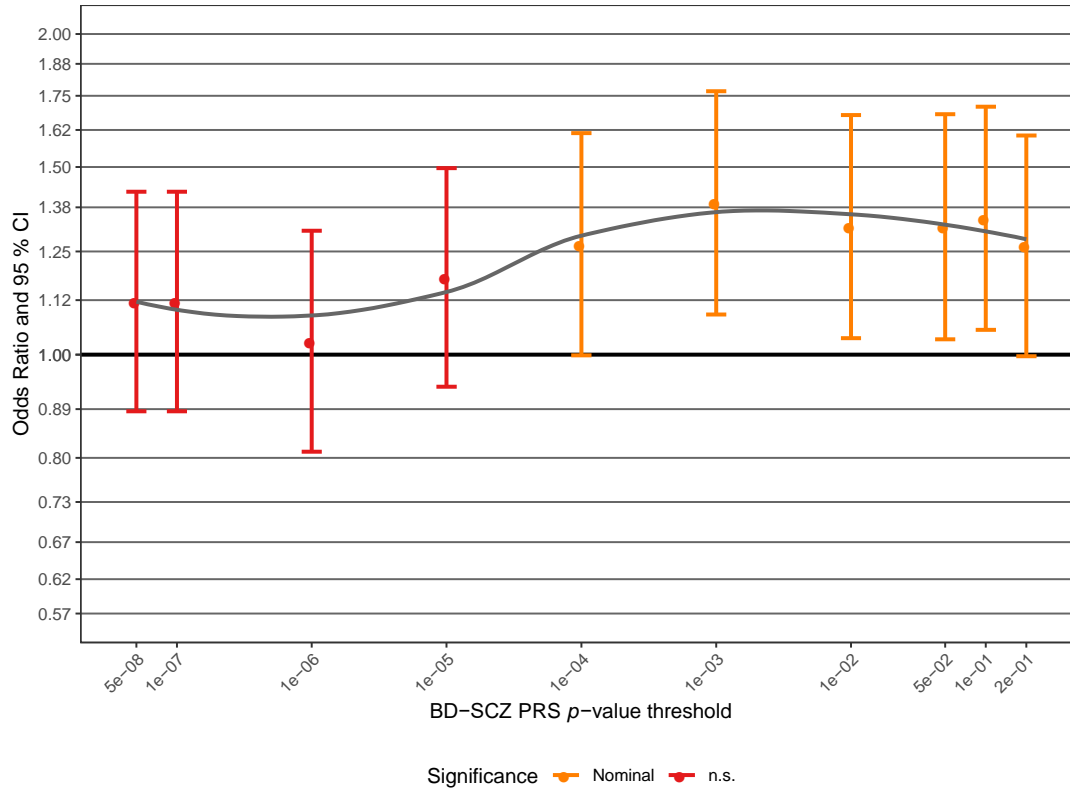
Supplementary Fig. S5C: Association of the MDD PRS.



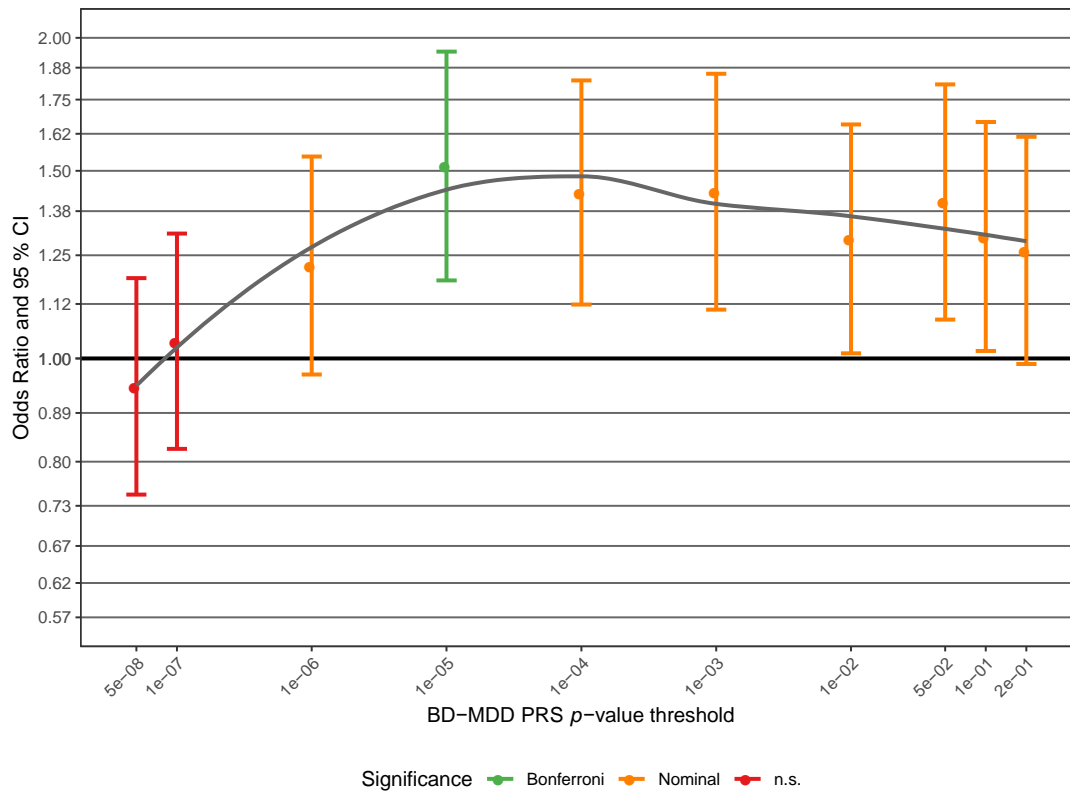
Supplementary Fig. S5D: Association of the Shared PRS.



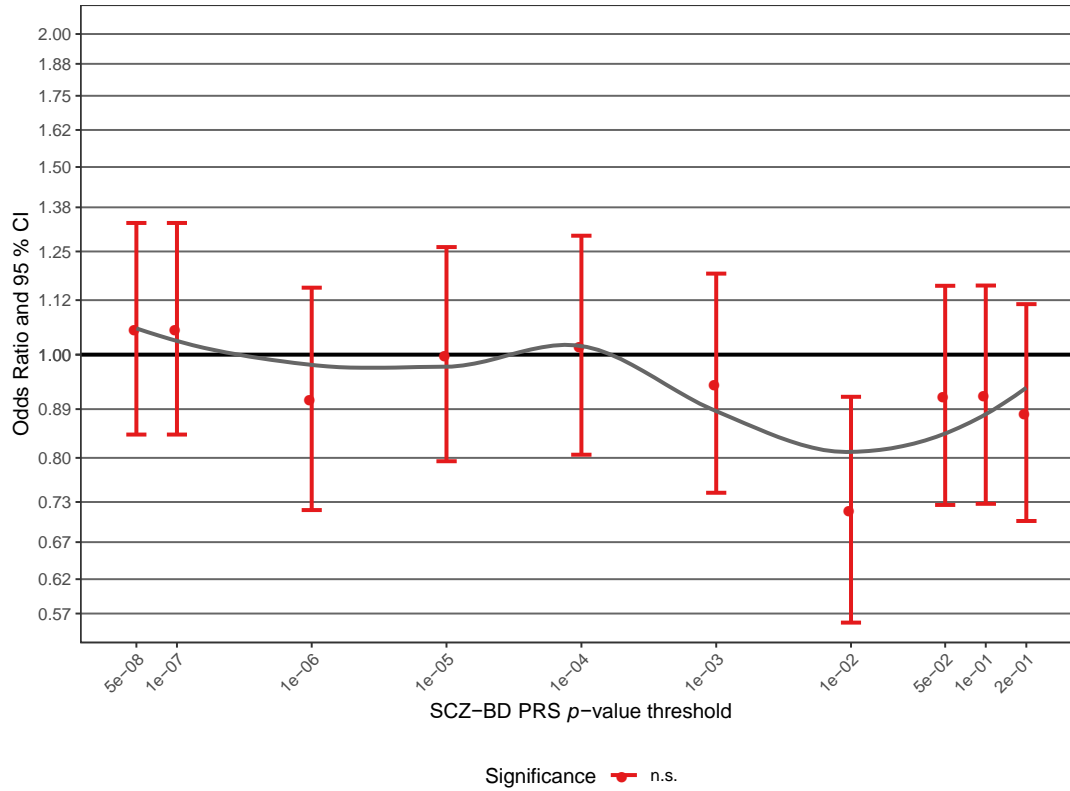
Supplementary Fig. S5E: Association of the BD-SCZ GWIS PRS.



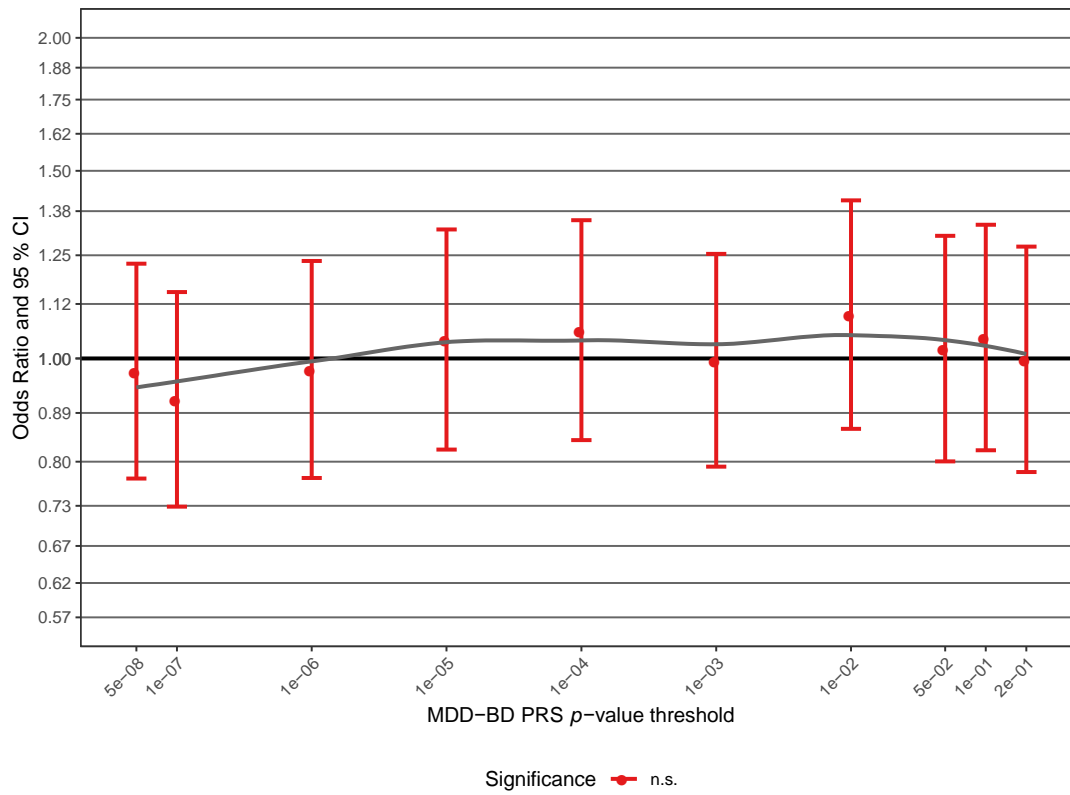
Supplementary Fig. S5F: Association of the BD-MDD GWIS PRS.



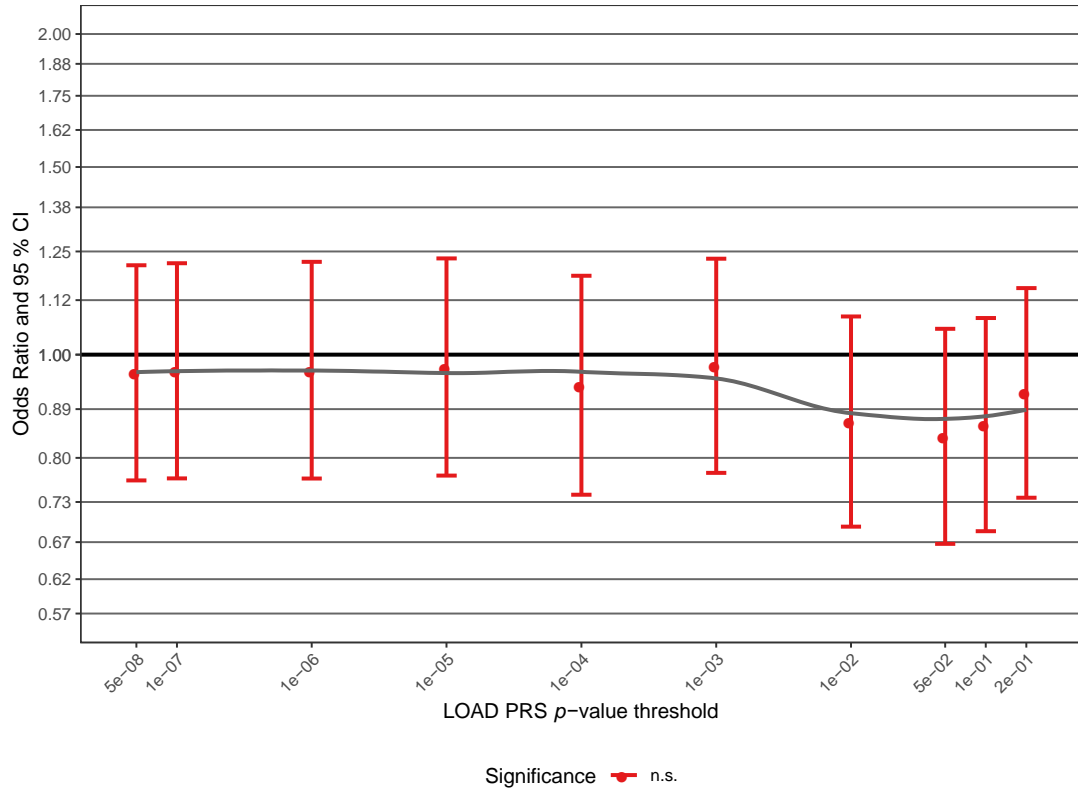
Supplementary Fig. S5G: Association of the SCZ-BD GWIS PRS.



Supplementary Fig. S5H: Association of the MDD-BD GWIS PRS.

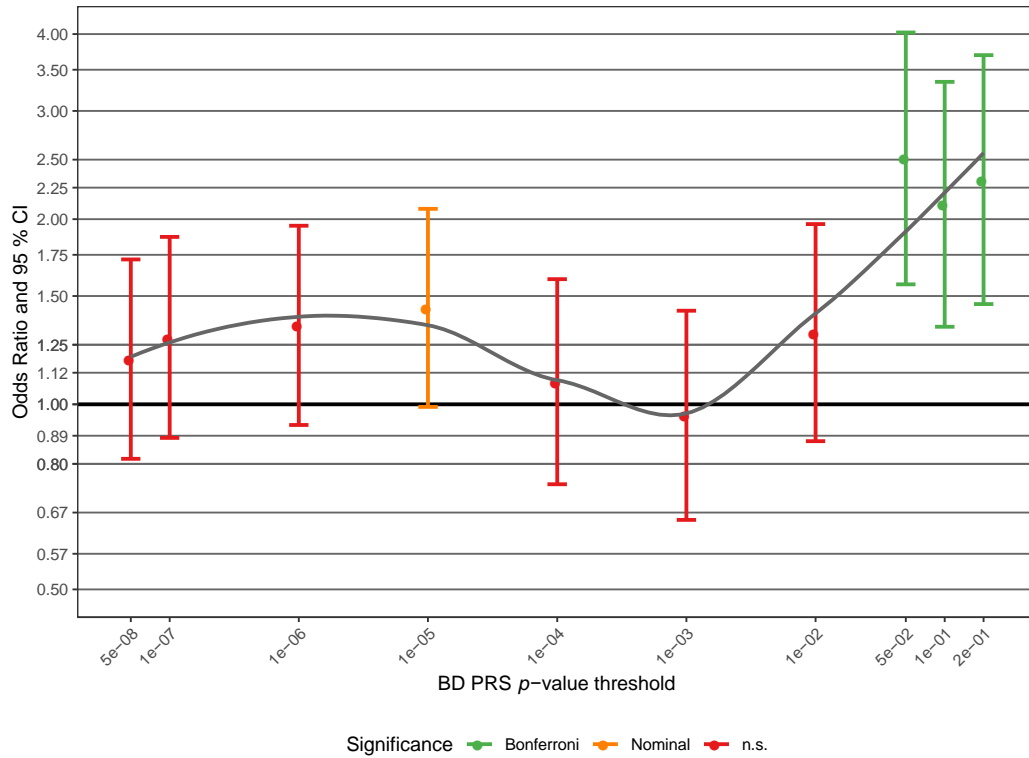


Supplementary Fig. S5I: Association of the LOAD PRS.

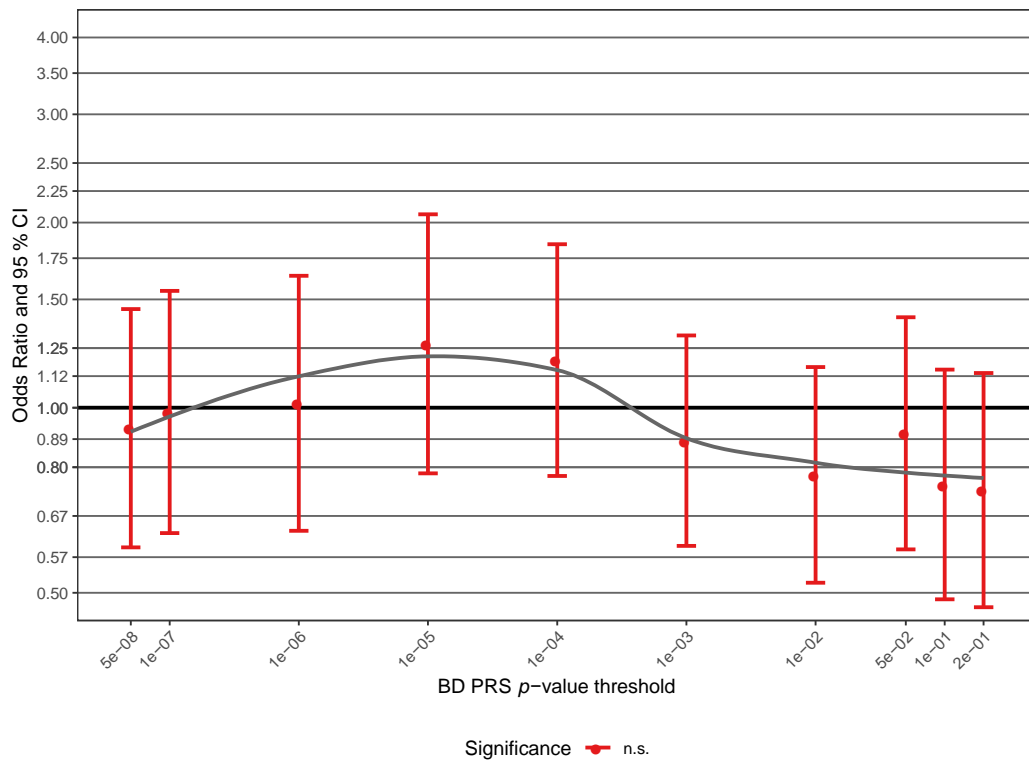


Supplementary Fig. S6: Analysis of assortative mating. Further details regarding the analysis and plots are described in the legend for Fig. 2C. Full association test statistics including p -values are shown in Supplementary Table S7. Significance threshold: $\alpha=0.05/20=0.0025$.

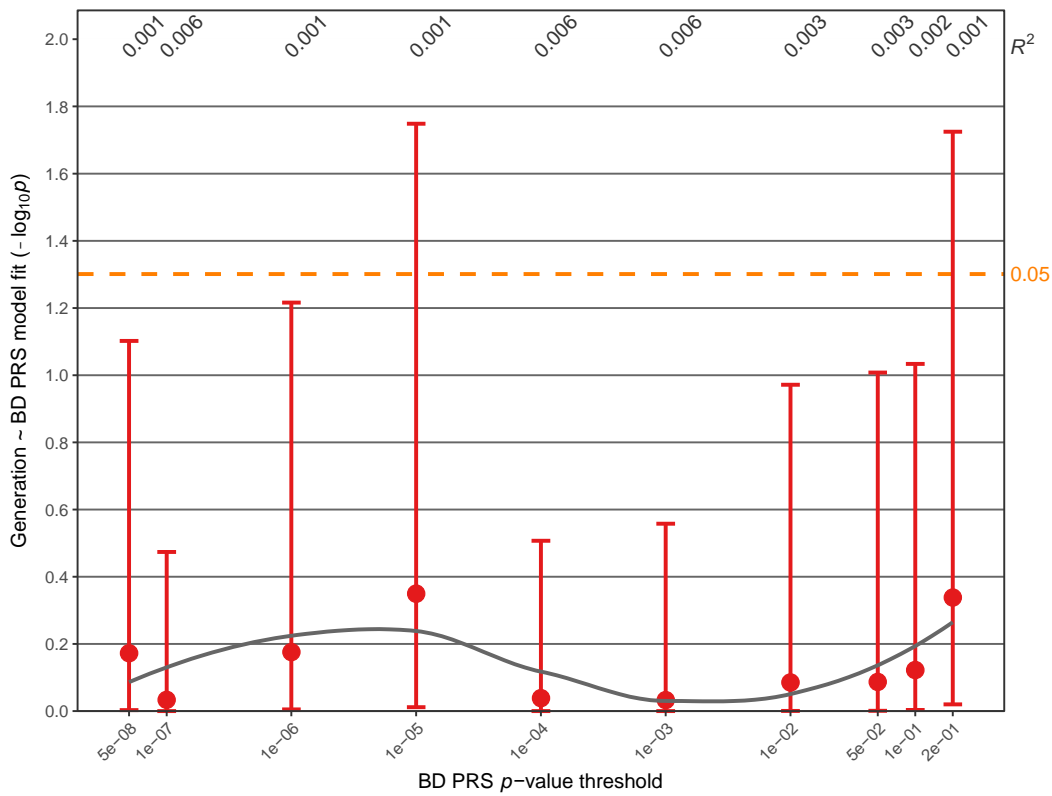
Supplementary Fig. S6A: Association analysis comparing the BD PRS in unaffected married-in family members and $CC_{controls}$.



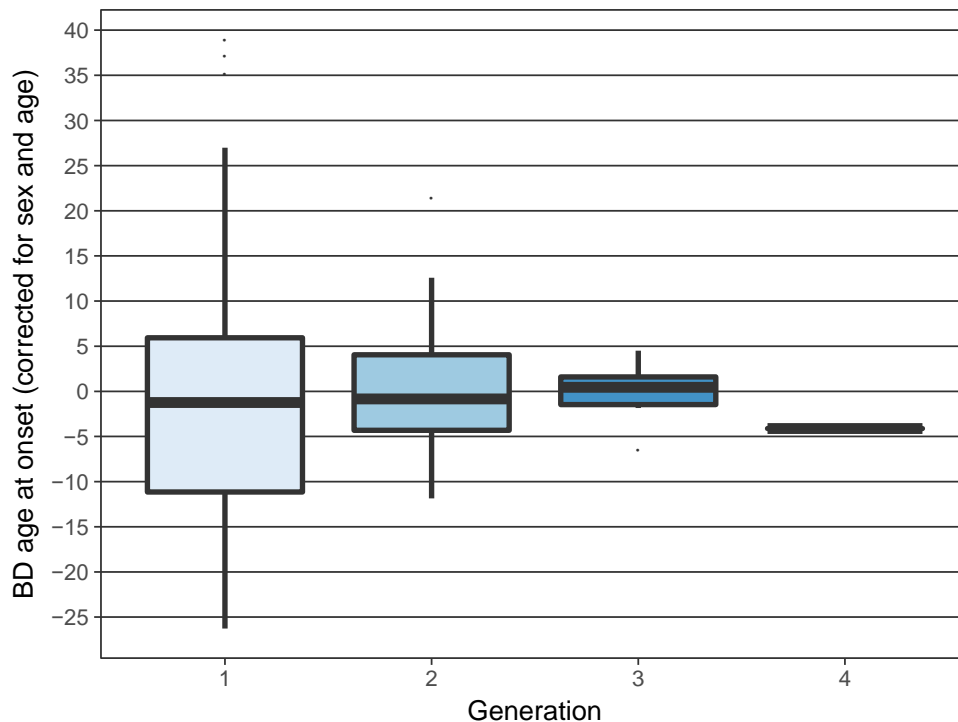
Supplementary Fig. S6B: Association analysis comparing the BD PRS in unaffected married-in family members to $FAM_{unaffected}$.



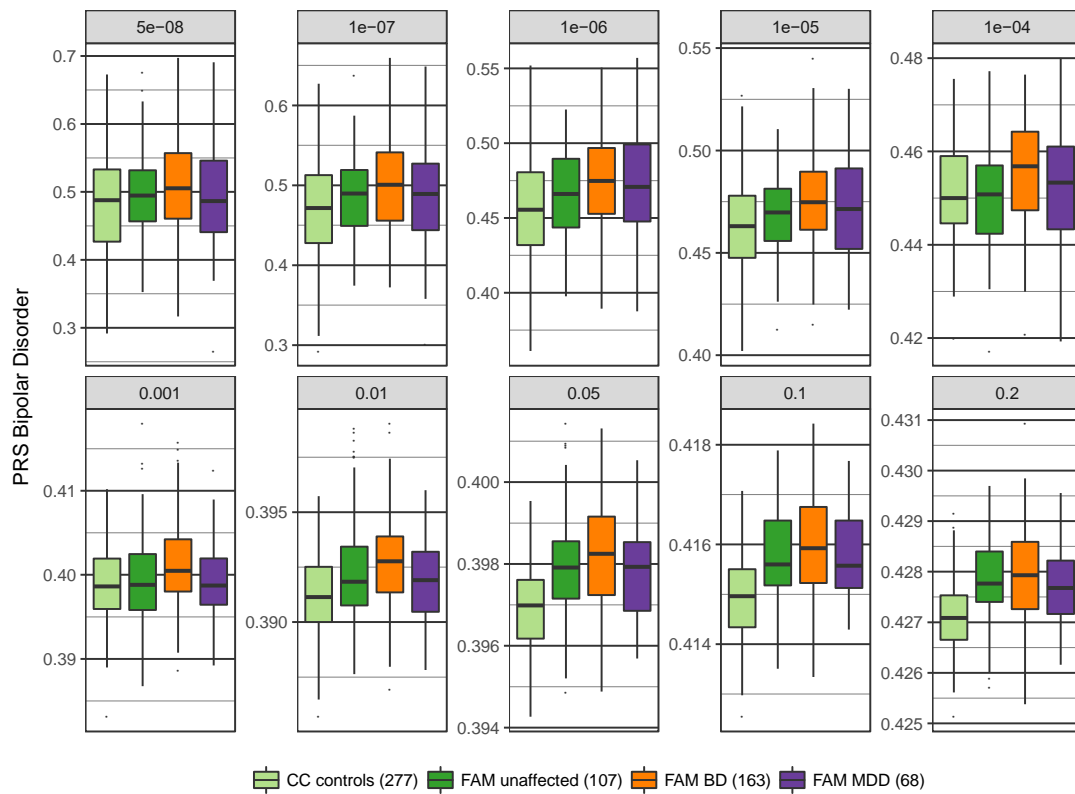
Supplementary Fig. S7: Analysis of anticipation in the FAM sample. Further details regarding the analysis and plots are described in the legend for Fig. 2D. Full association test statistics including p -values are shown in Supplementary Table S8.
Supplementary Fig. S7A: Association of the BD PRS with generation.



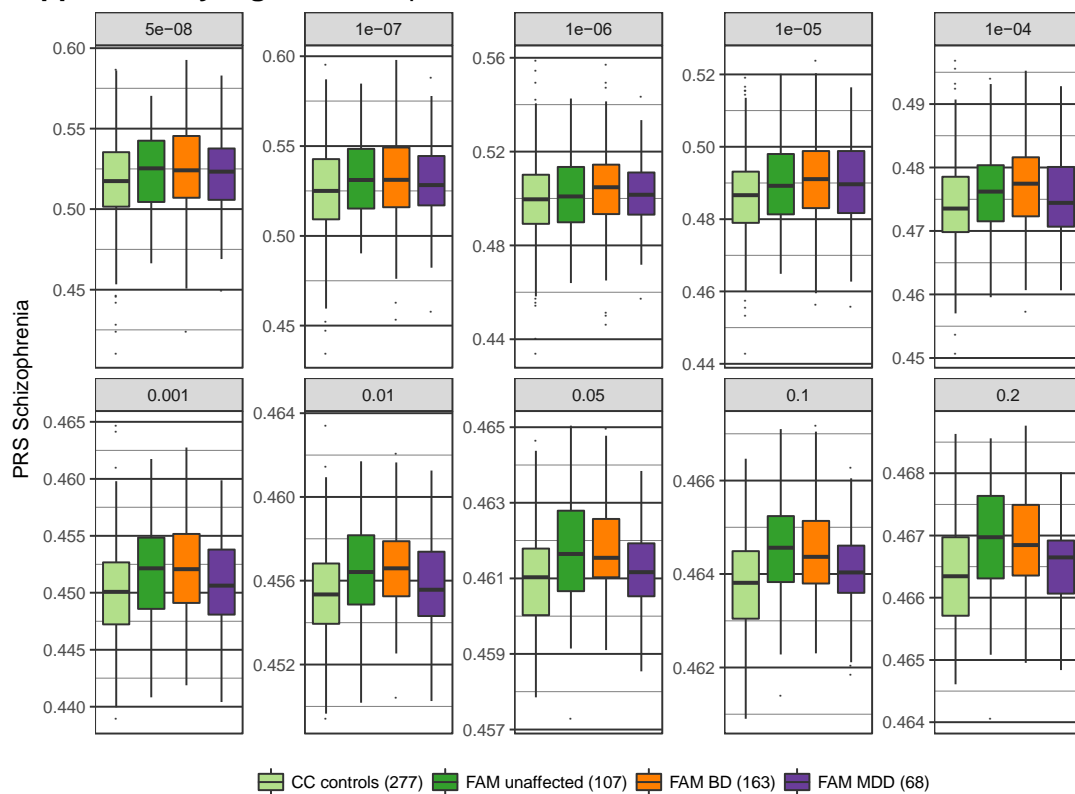
Supplementary Fig. S7B: Association analysis comparing the age at onset for BD across generations. The age at onset did not decrease over generations ($p=0.54$, Supplementary Table S8). Covariates were sex and age. One-sided p -values were calculated, following the hypothesis that the age at onset decreases across generations. The y-axis shows the residuals from the linear model.



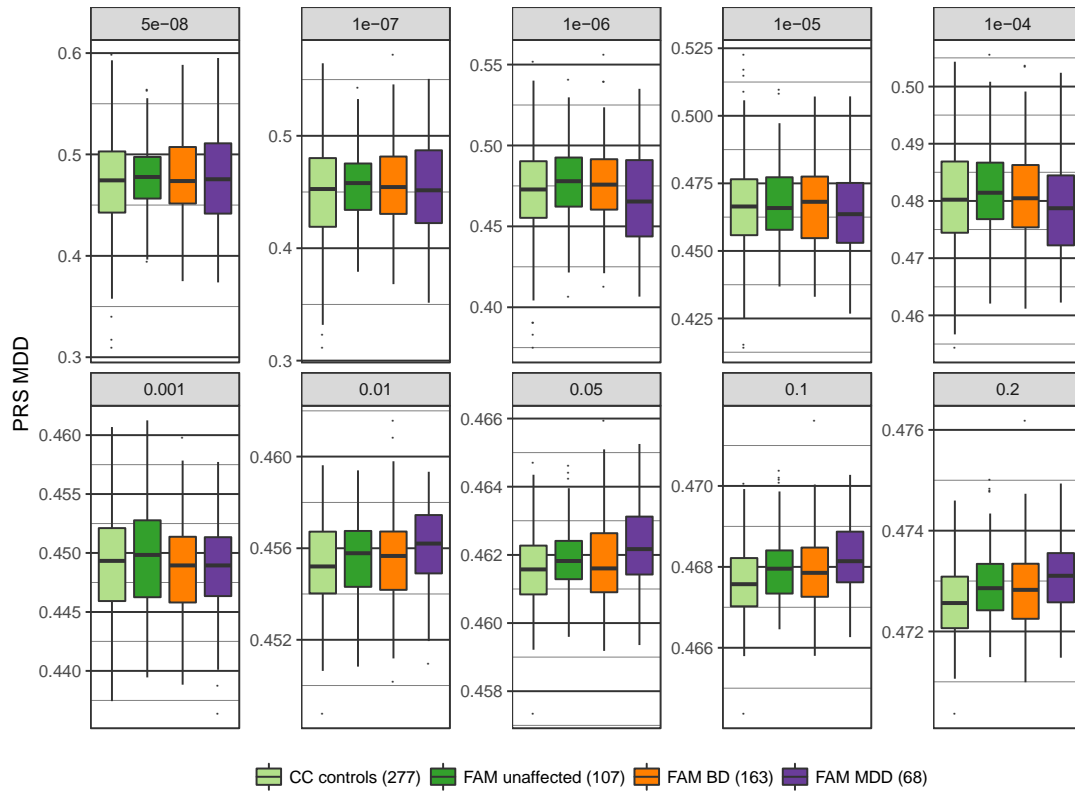
Supplementary Fig. S8: Boxplots of PRS at different p -value thresholds, including FAM_{MDD} cases. The following individuals are not shown in these plots: Family members with a history of substance abuse, married-in family members, and CC_{BD} cases.
Supplementary Fig. S8A: Boxplots of BD PRS.



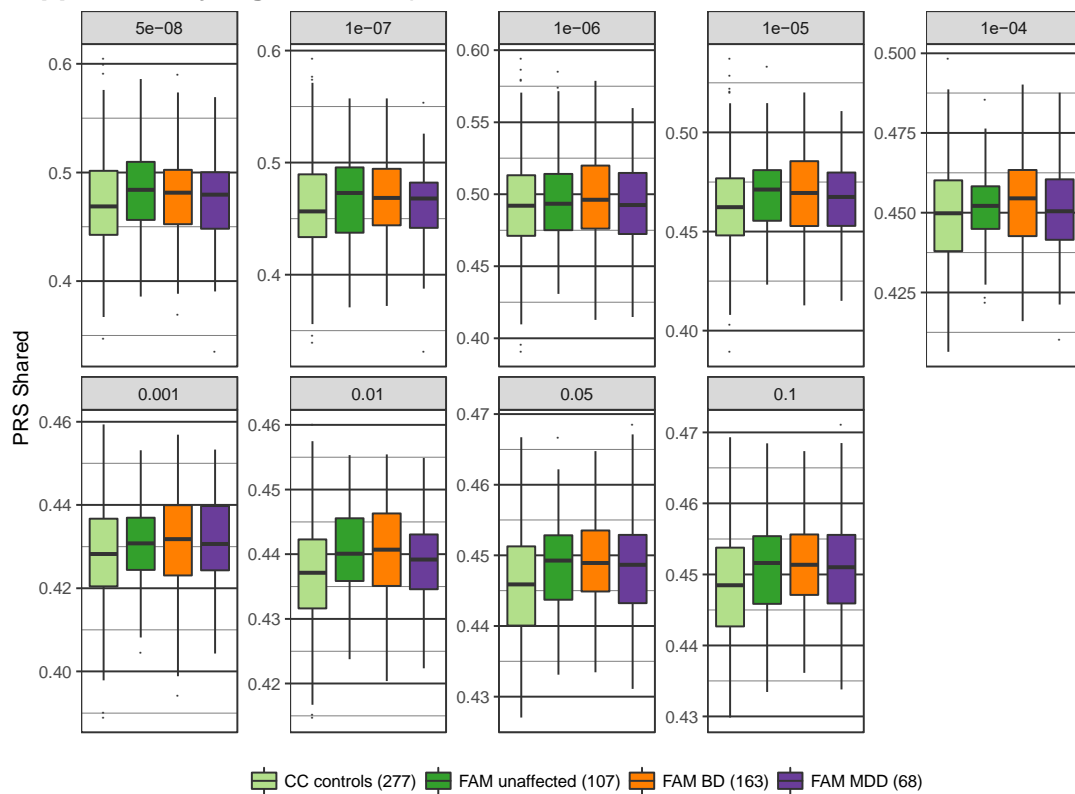
Supplementary Fig. S8B: Boxplots of SCZ PRS.



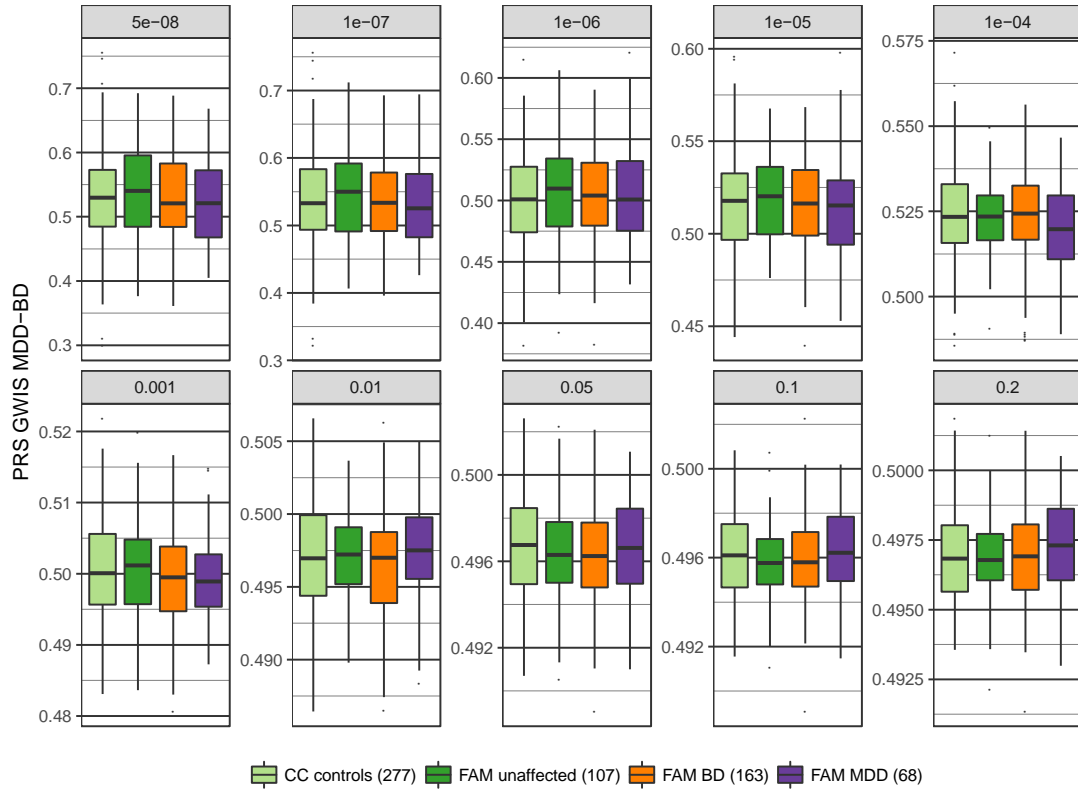
Supplementary Fig. S8C: Boxplots of MDD PRS.



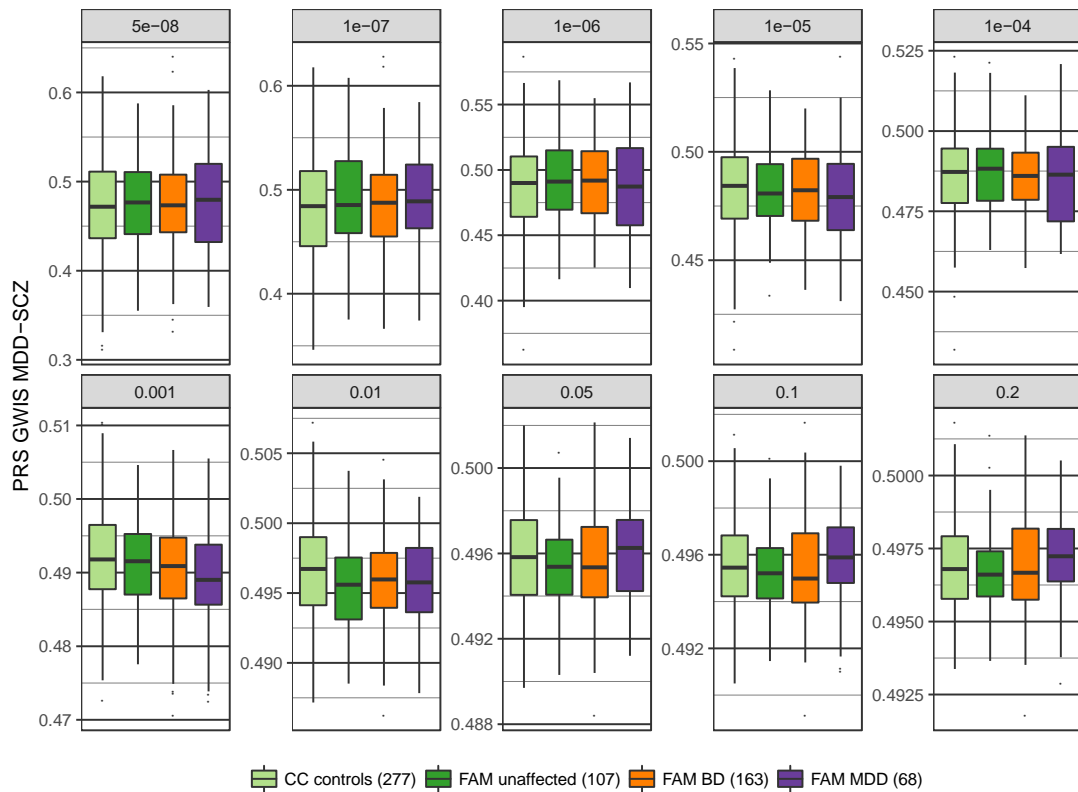
Supplementary Fig. S8D: Boxplots of the BD+SCZ+MDD Shared PRS.



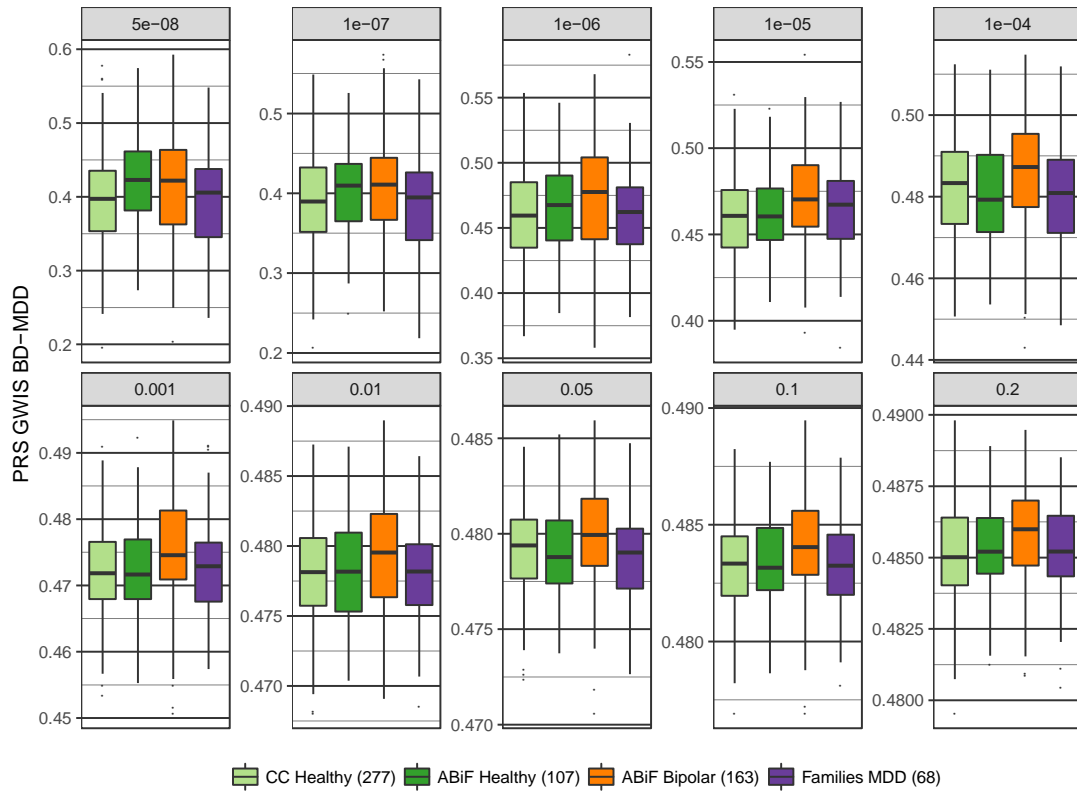
Supplementary Fig. S8E: Boxplots of the MDD-BD GWIS PRS.



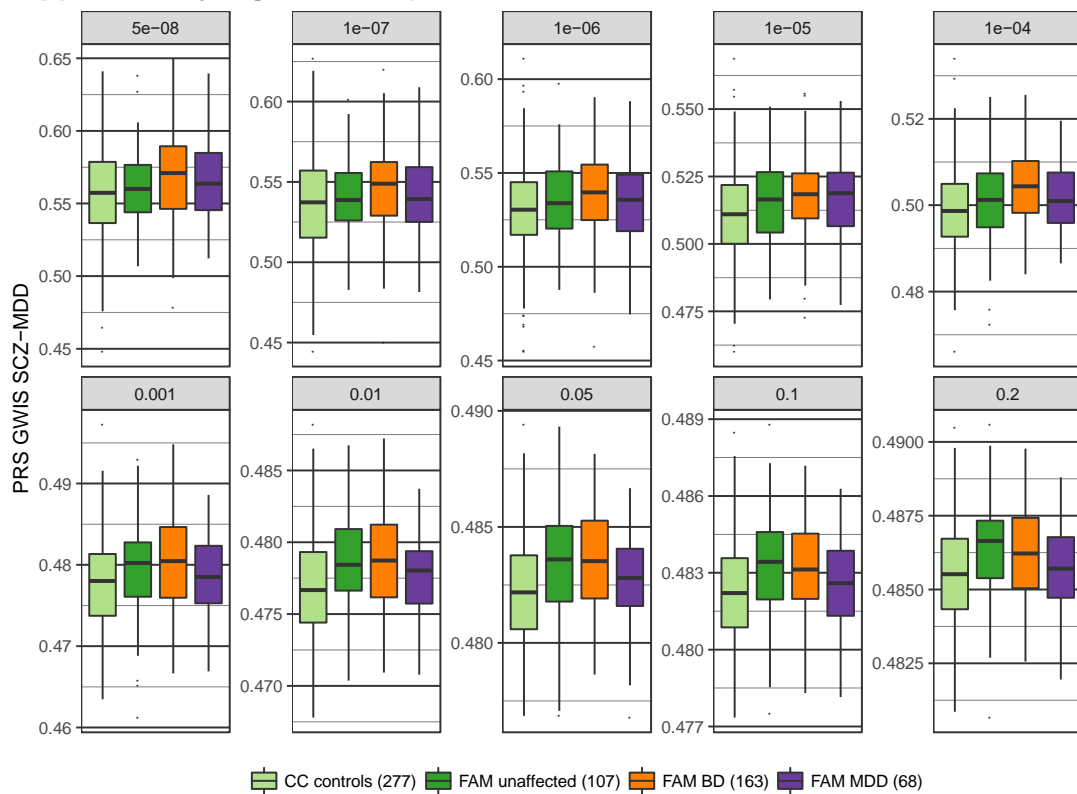
Supplementary Fig. S8F: Boxplots of the MDD-SCZ GWIS PRS.



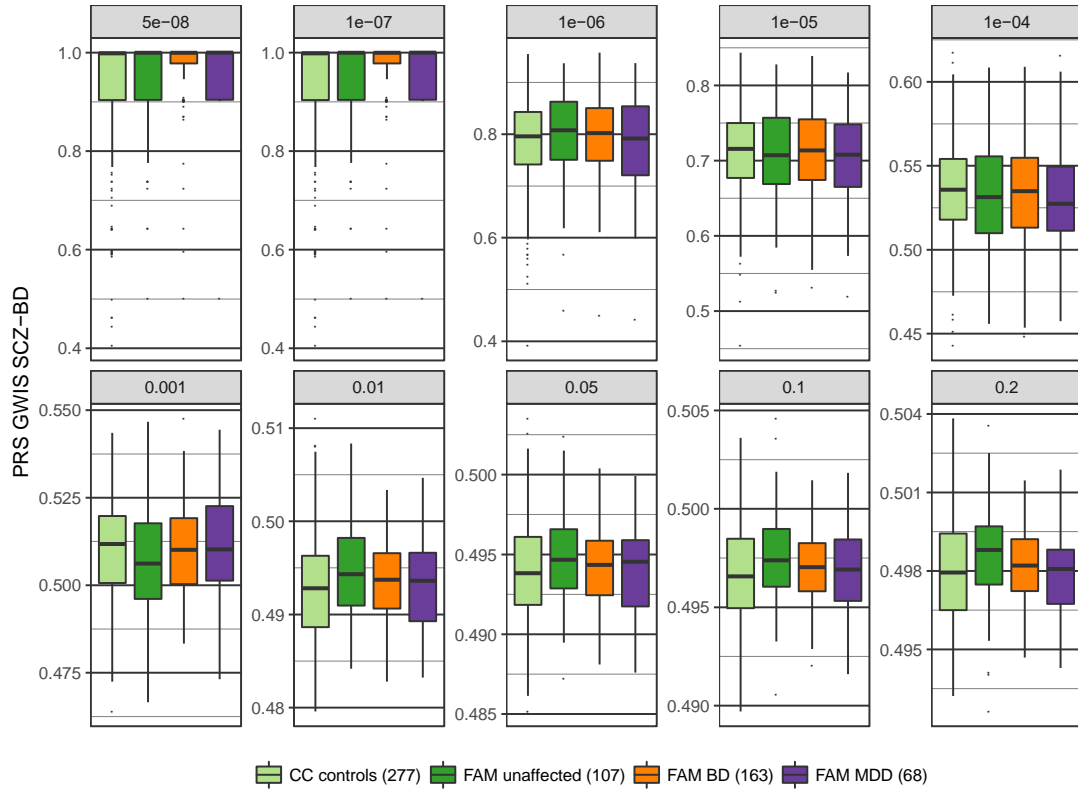
Supplementary Fig. S8G: Boxplots of the BD-MDD GWIS PRS.



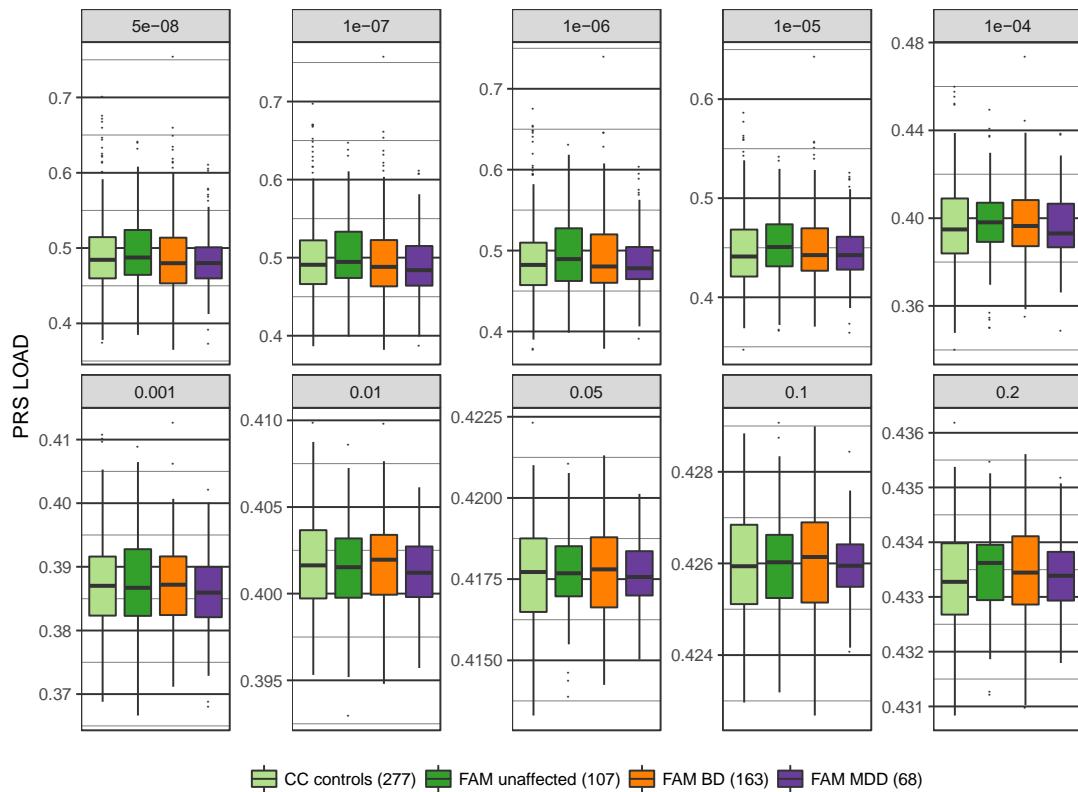
Supplementary Fig. S8H: Boxplots of the SCZ-MDD GWIS PRS.



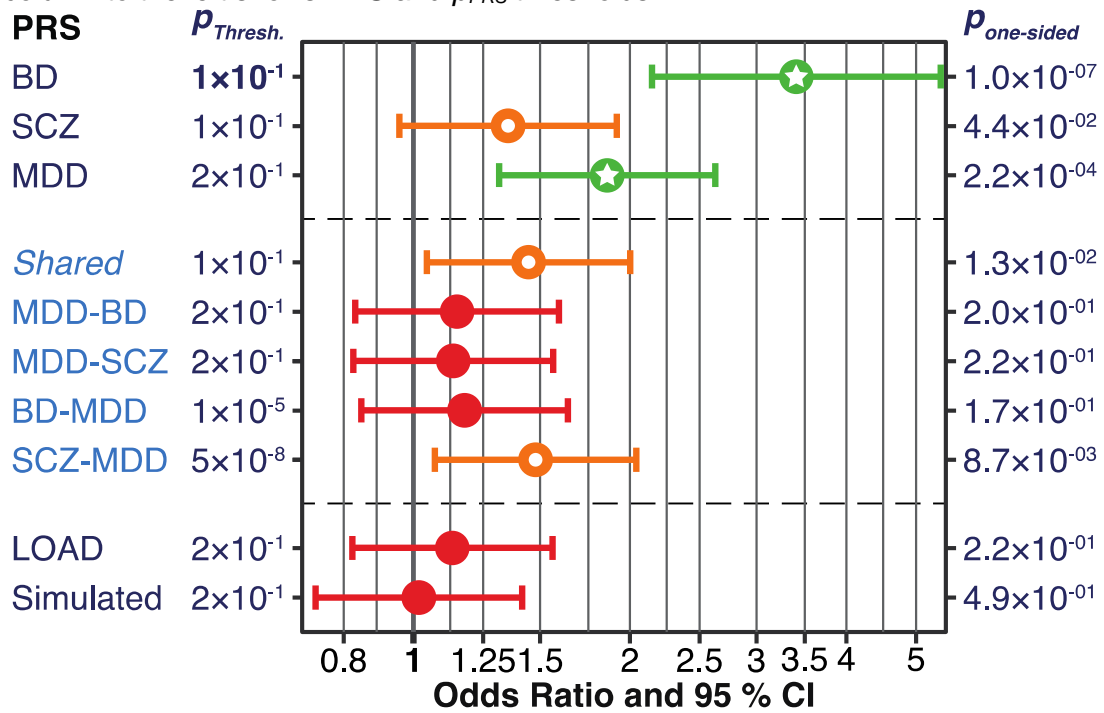
Supplementary Fig. S8I: Boxplots of the SCZ-BD GWIS PRS.



Supplementary Fig. S8J: Boxplots of the LOAD (Alzheimer) PRS.

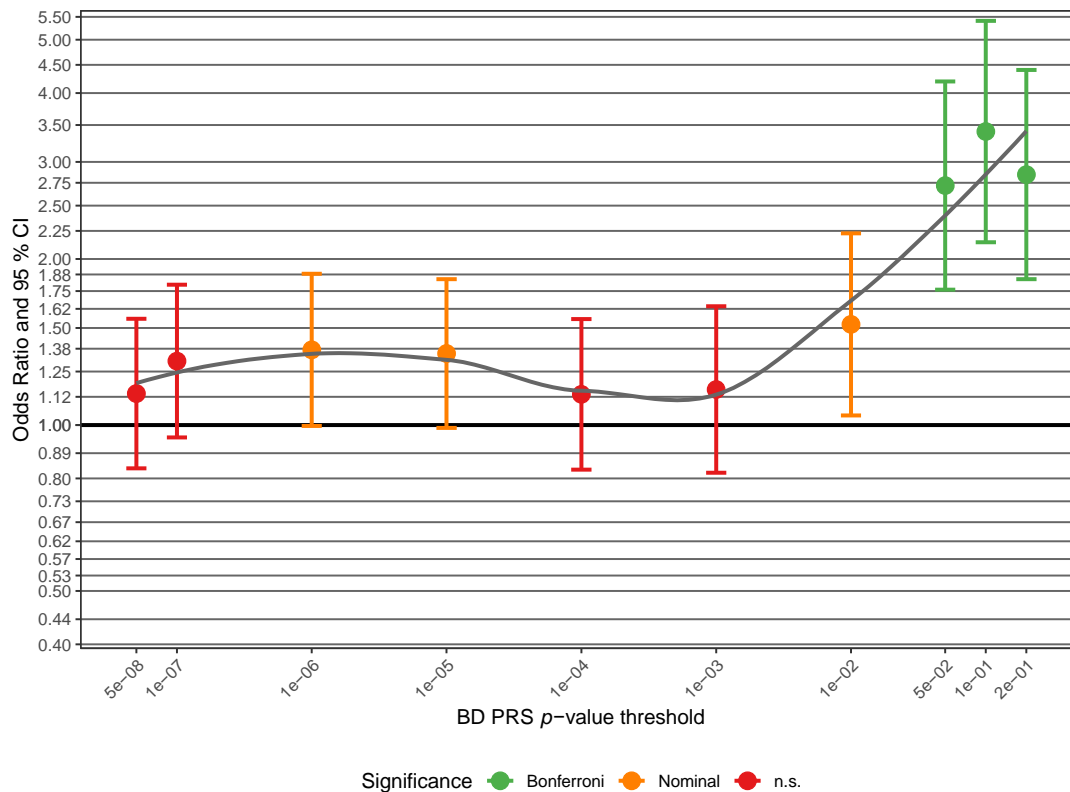


Supplementary Fig. S9: Association analysis comparing PRS in FAM_{MDD} cases and CC_{controls}. Further details of the plots are described in the legend for Fig. 1. Full association test statistics including p -values are shown in Supplementary Table S9.
Supplementary Fig. S9A: Top-associated p -value thresholds for the tested PRS. The column to the left shows PRS and p_{PRS} thresholds.



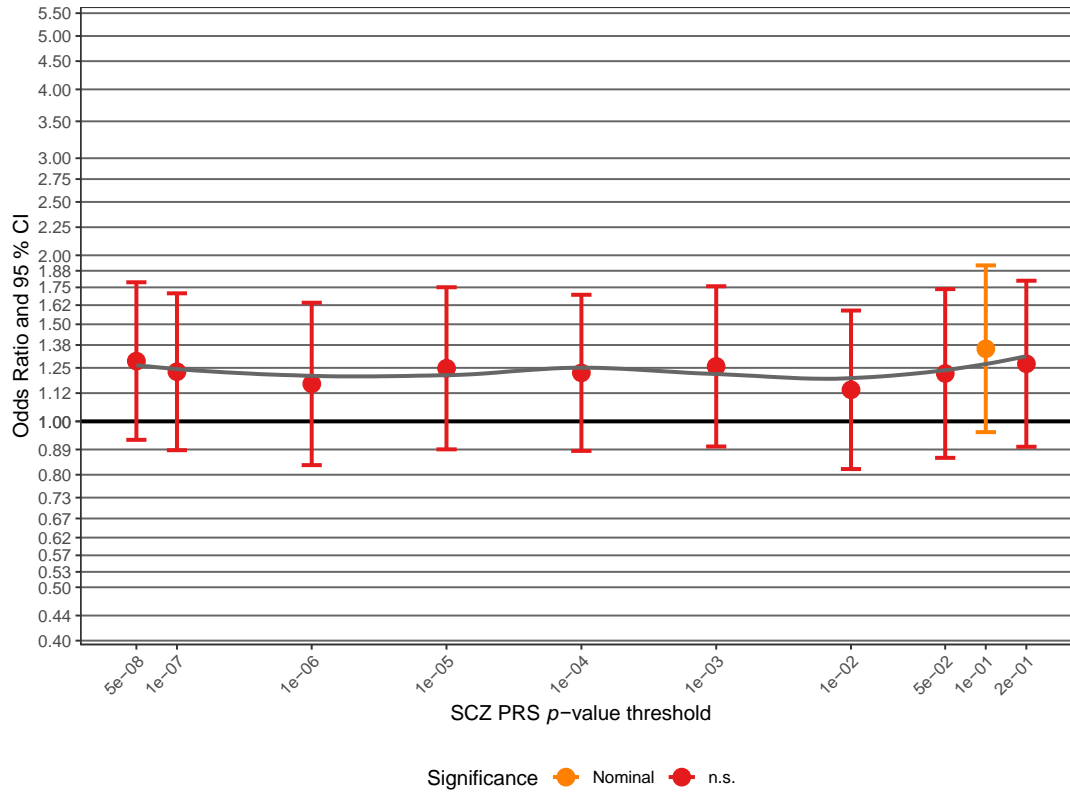
Significance: ★ Bonferroni ○ Nominal ● n.s.

Supplementary Fig. S9B: Association of the BD PRS (all p -value thresholds).

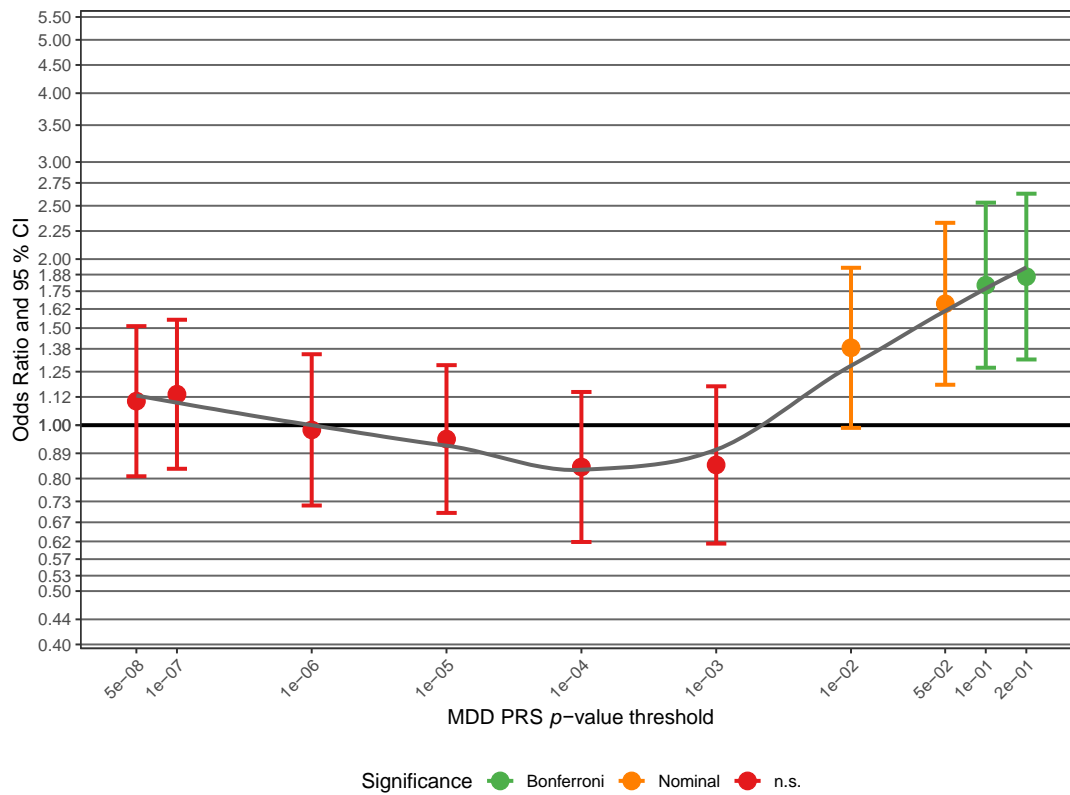


Significance ★ Bonferroni ○ Nominal ● n.s.

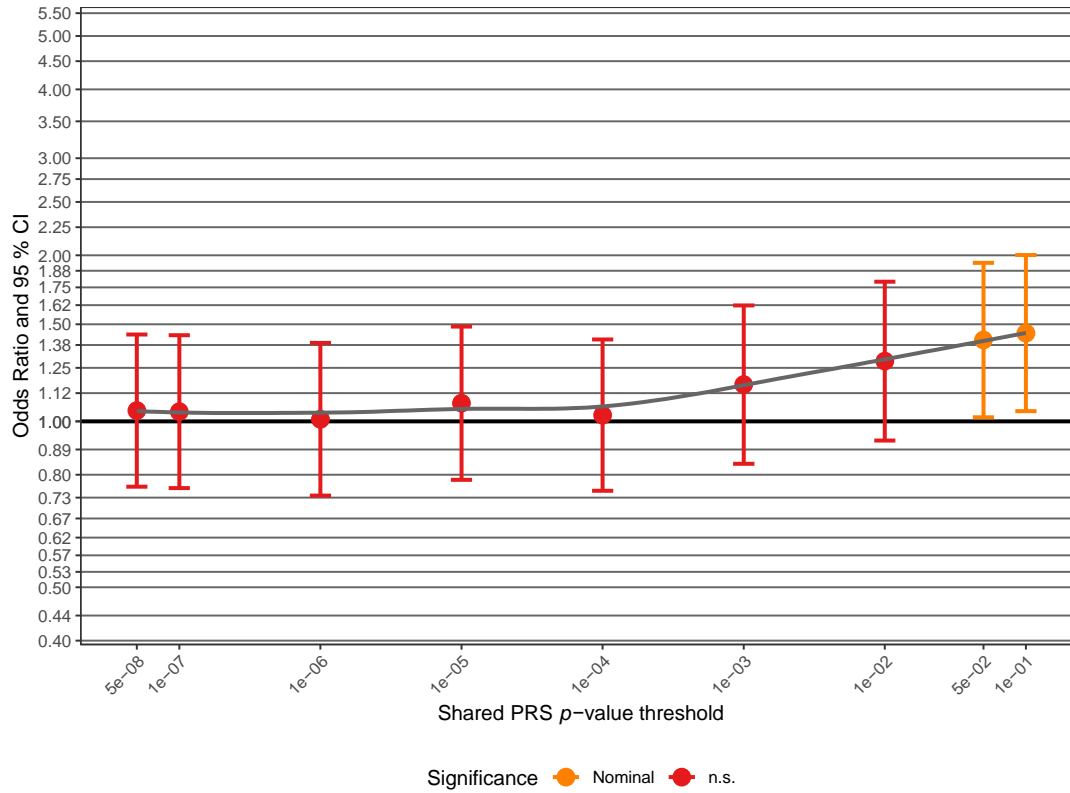
Supplementary Fig. S9C: Association of the SCZ PRS.



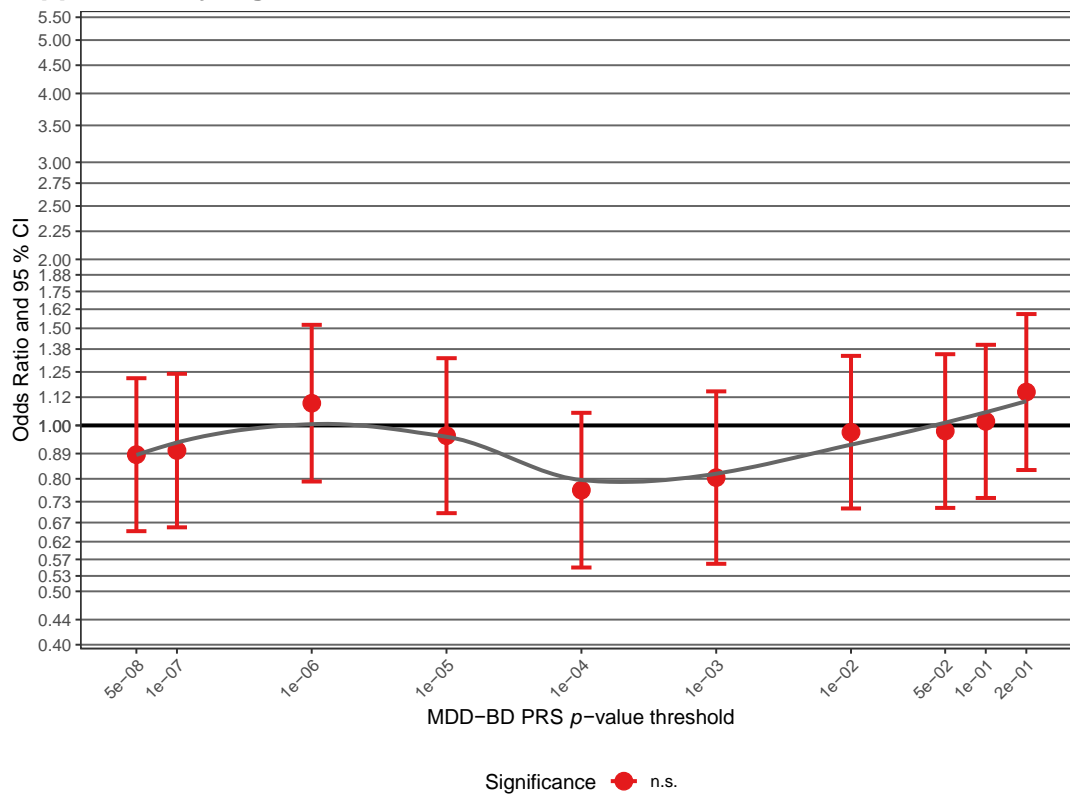
Supplementary Fig. S9D: Association of the MDD PRS.



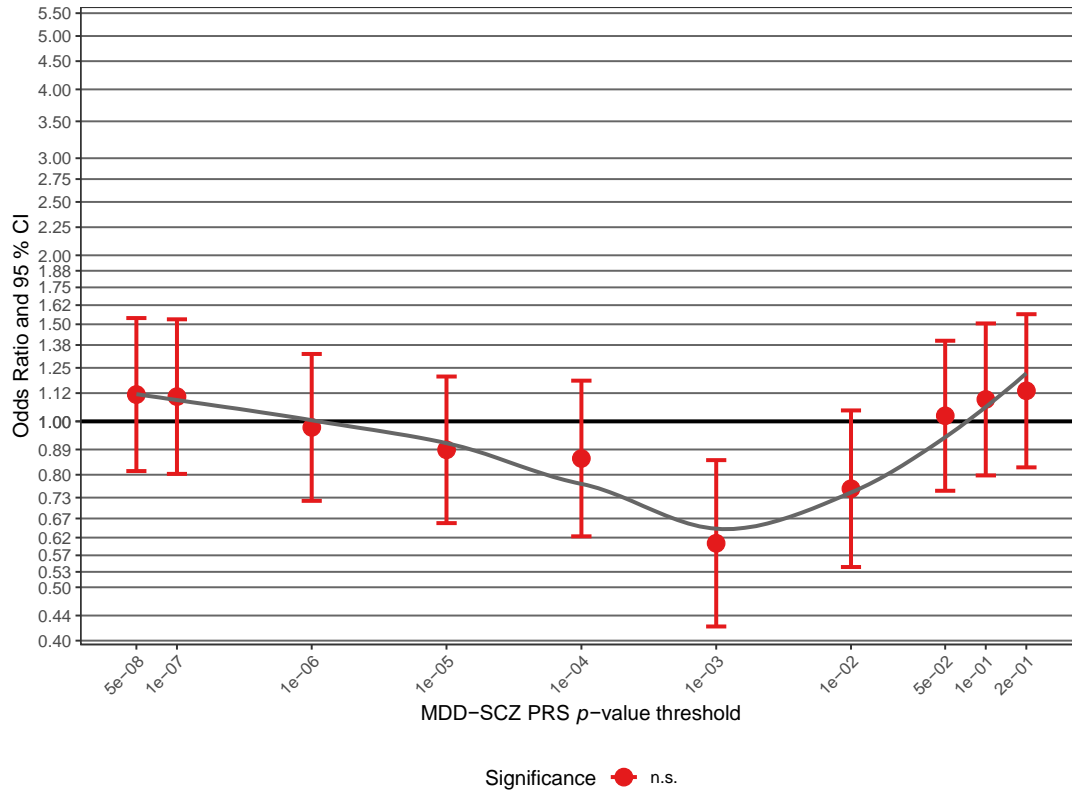
Supplementary Fig. S9E: Association of the *Shared* PRS.



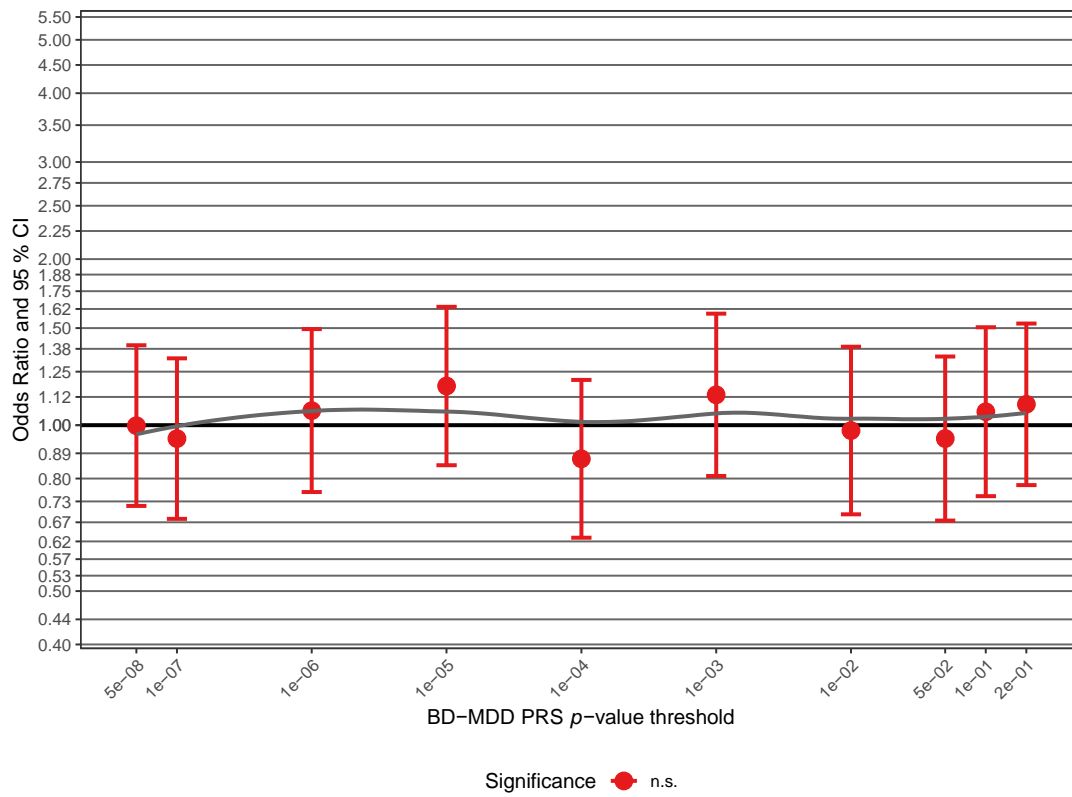
Supplementary Fig. S9F: Association of the MDD-BD GWIS PRS.



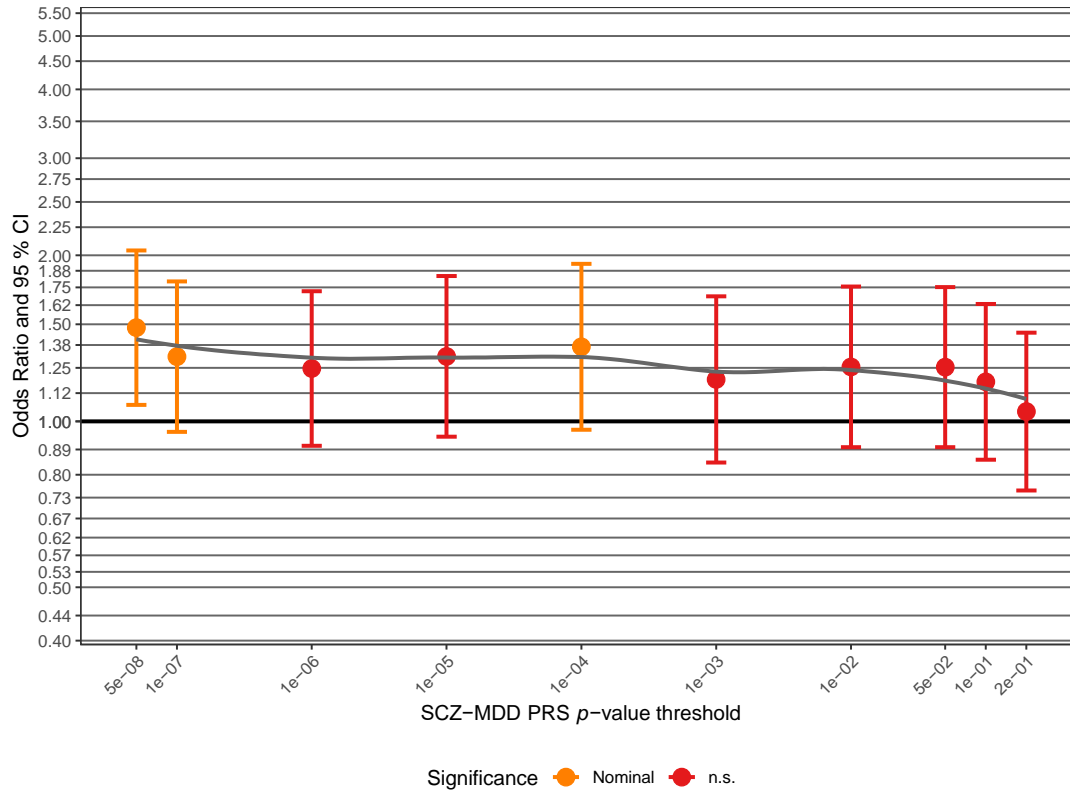
Supplementary Fig. S9G: Association of the MDD-SCZ GWIS PRS.



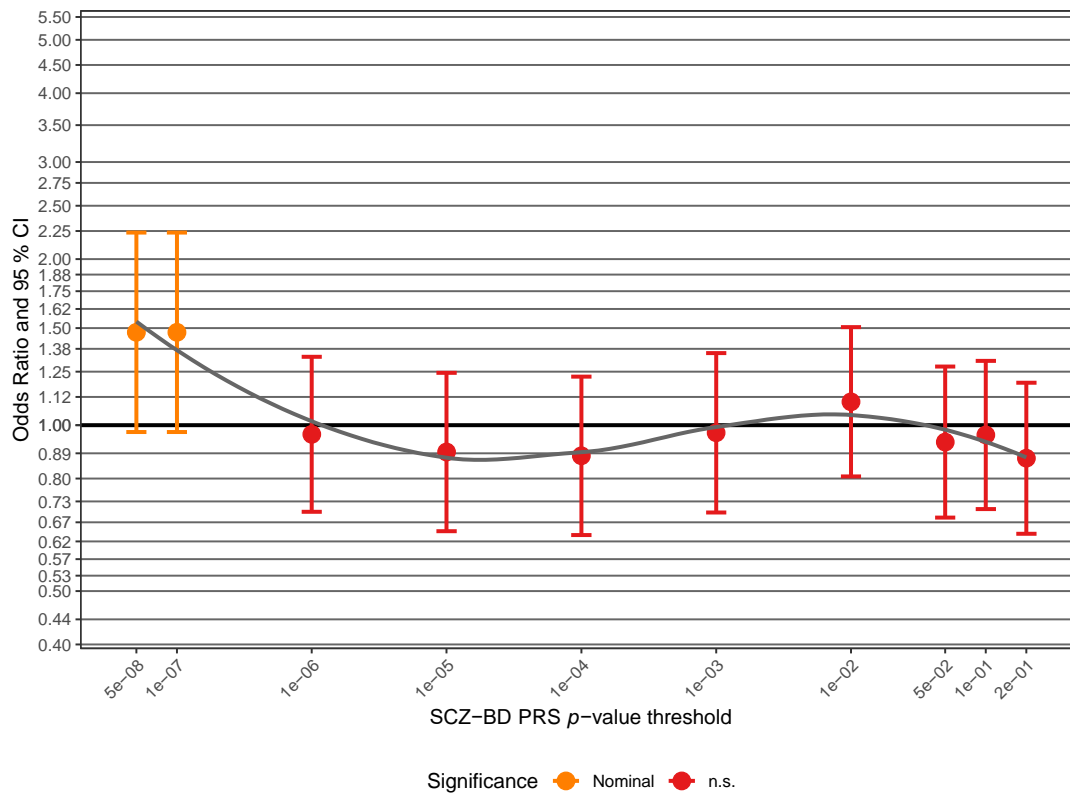
Supplementary Fig. S9H: Association of the BD-MDD GWIS PRS.



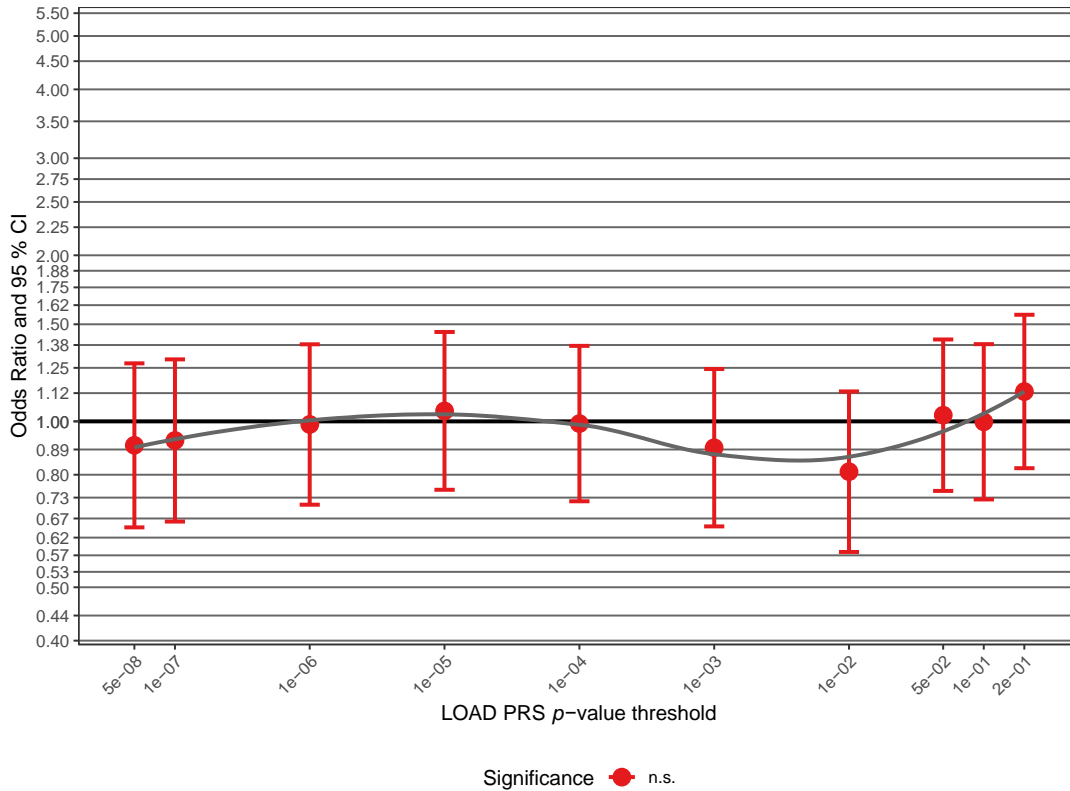
Supplementary Fig. S9I: Association of the SCZ-MDD GWIS PRS.



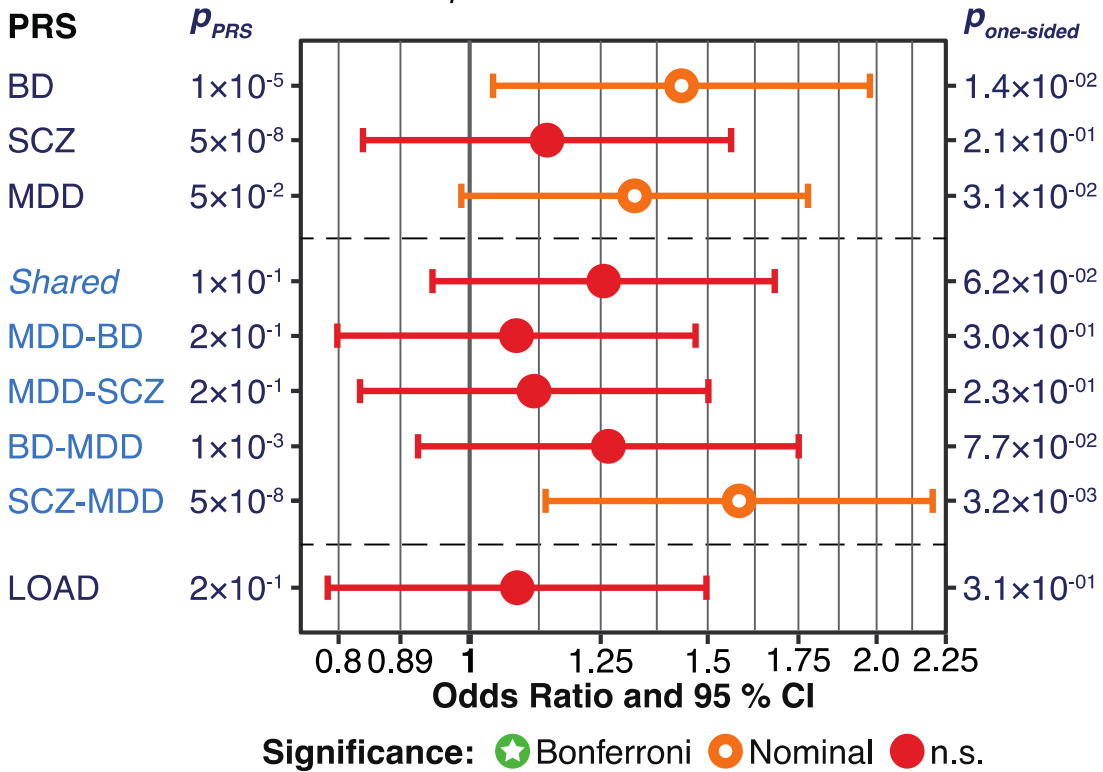
Supplementary Fig. S9J: Association of the SCZ-BD GWIS PRS.



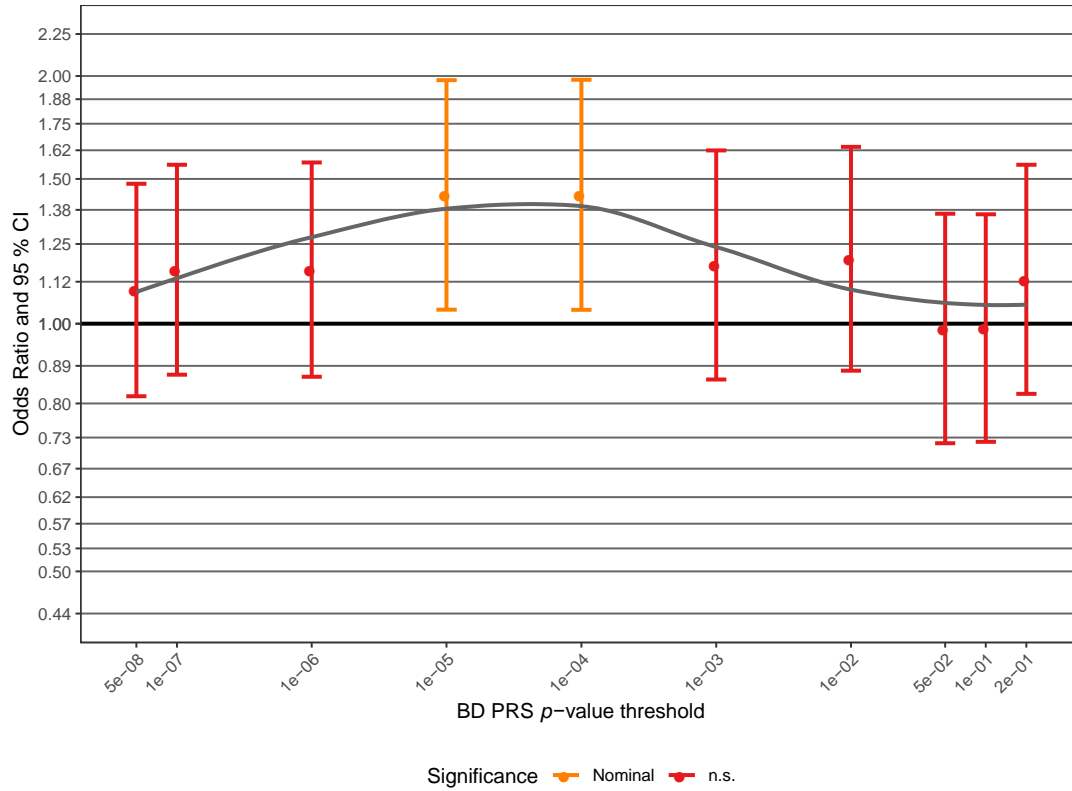
Supplementary Fig. S9K: Association of the LOAD PRS.



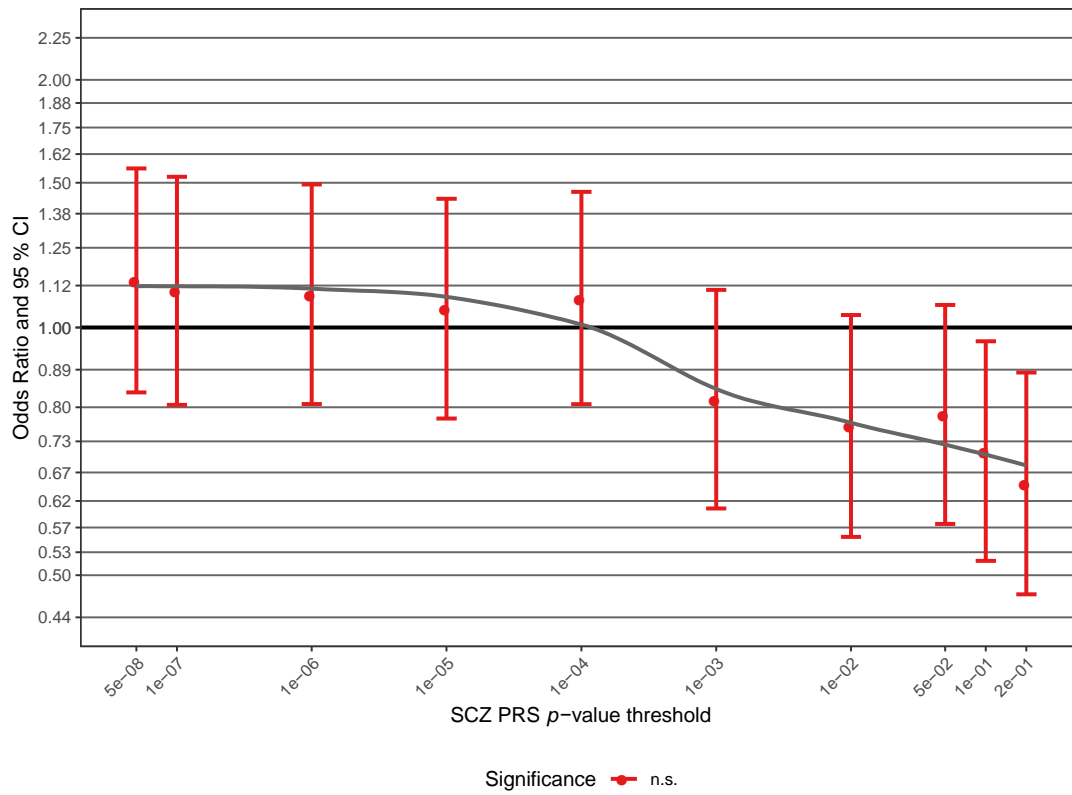
Supplementary Fig. S10: Association analysis comparing PRS in FAM_{MDD} cases and $FAM_{unaffected}$. Further details of the plots are given in the legends for Figs. 1 and 2. Full association test statistics including p -values are shown in Supplementary Table S10.
Supplementary Fig. S10A: Top-associated p_{PRS} thresholds for the tested PRS. The column to the left shows PRS and p_{PRS} thresholds.



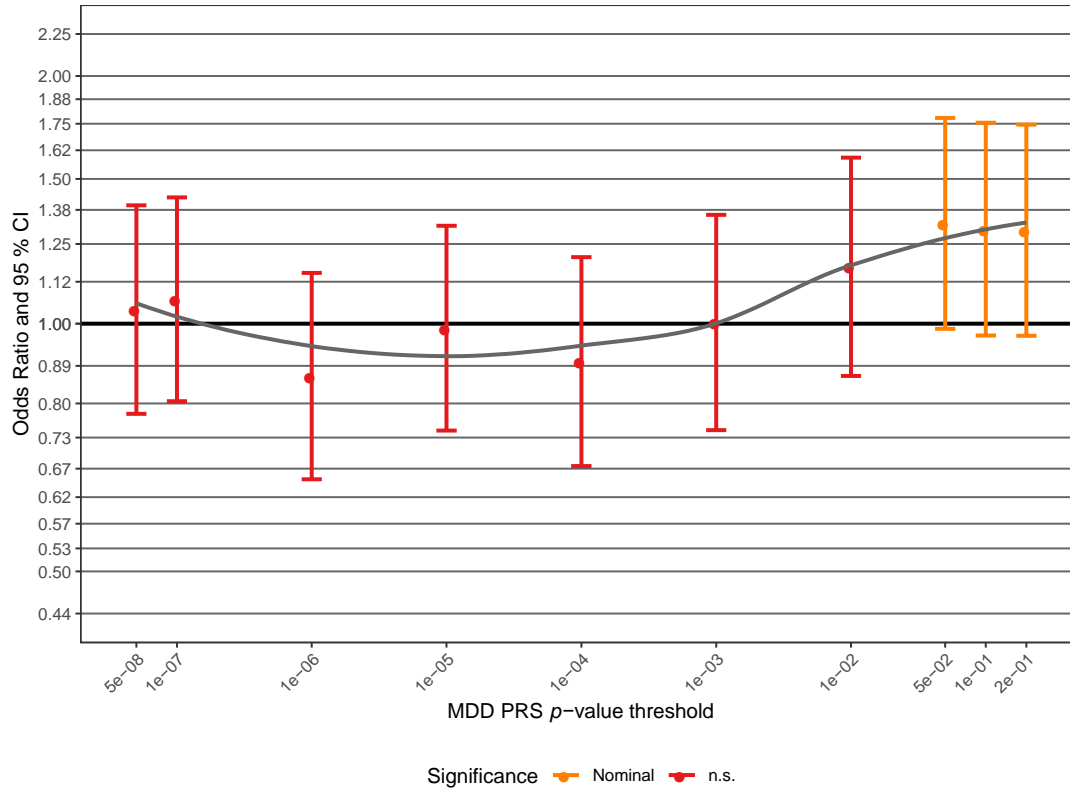
Supplementary Fig. S10B: Association of the BD PRS (all p -value thresholds).



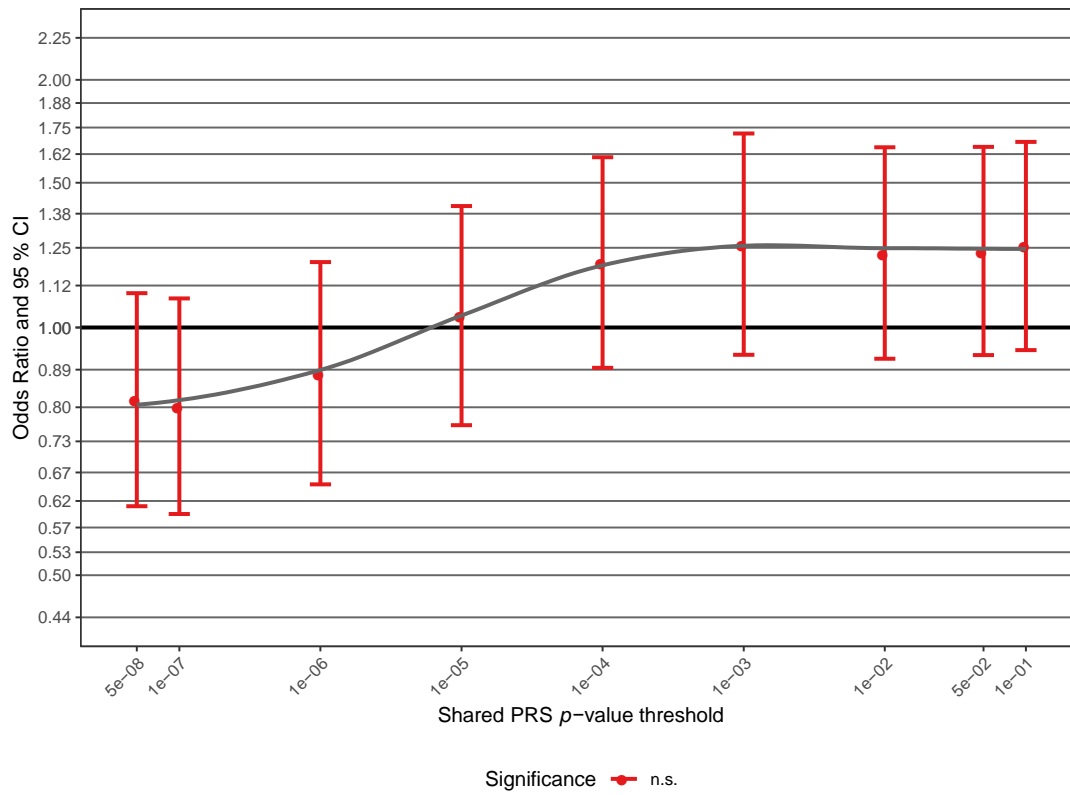
Supplementary Fig. S10C: Association of the SCZ PRS.



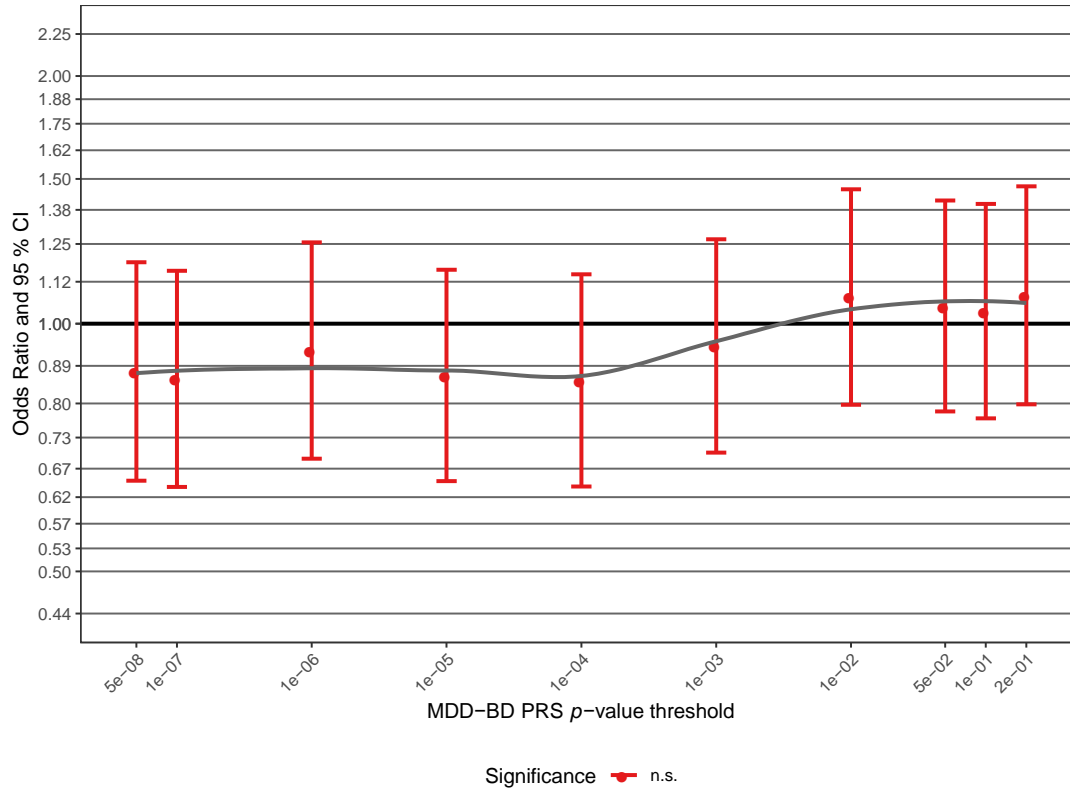
Supplementary Fig. S10D: Association of the MDD PRS.



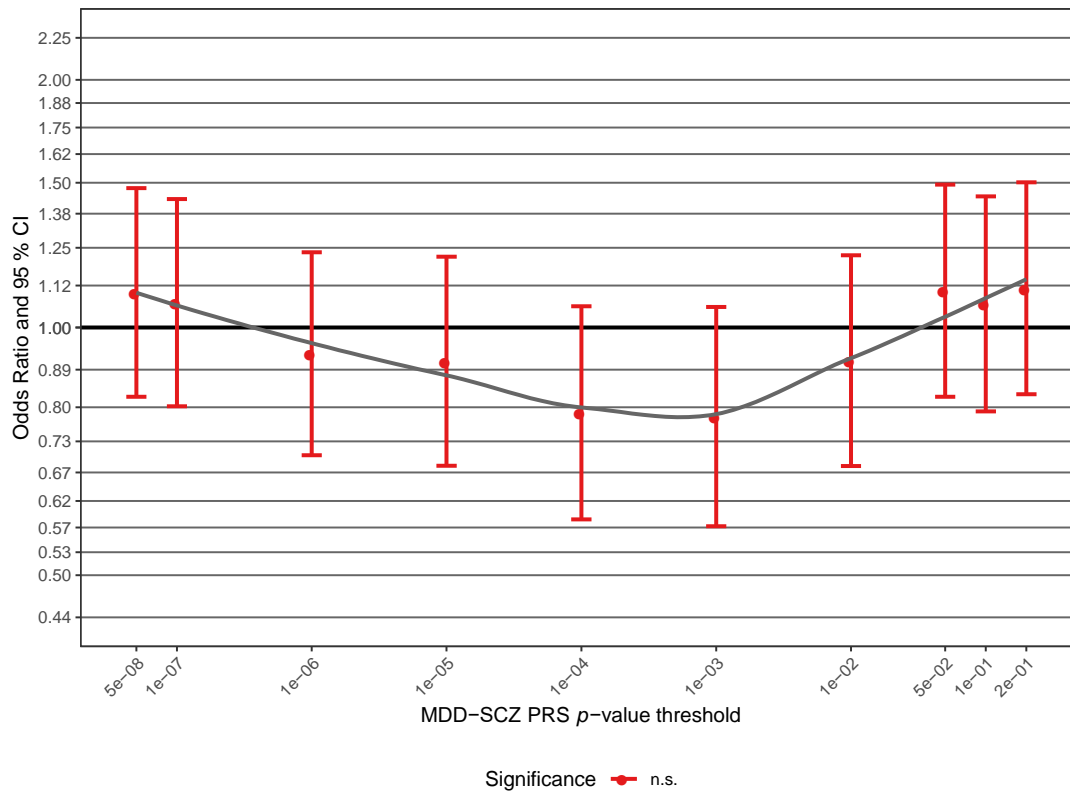
Supplementary Fig. S10E: Association of the Shared PRS.



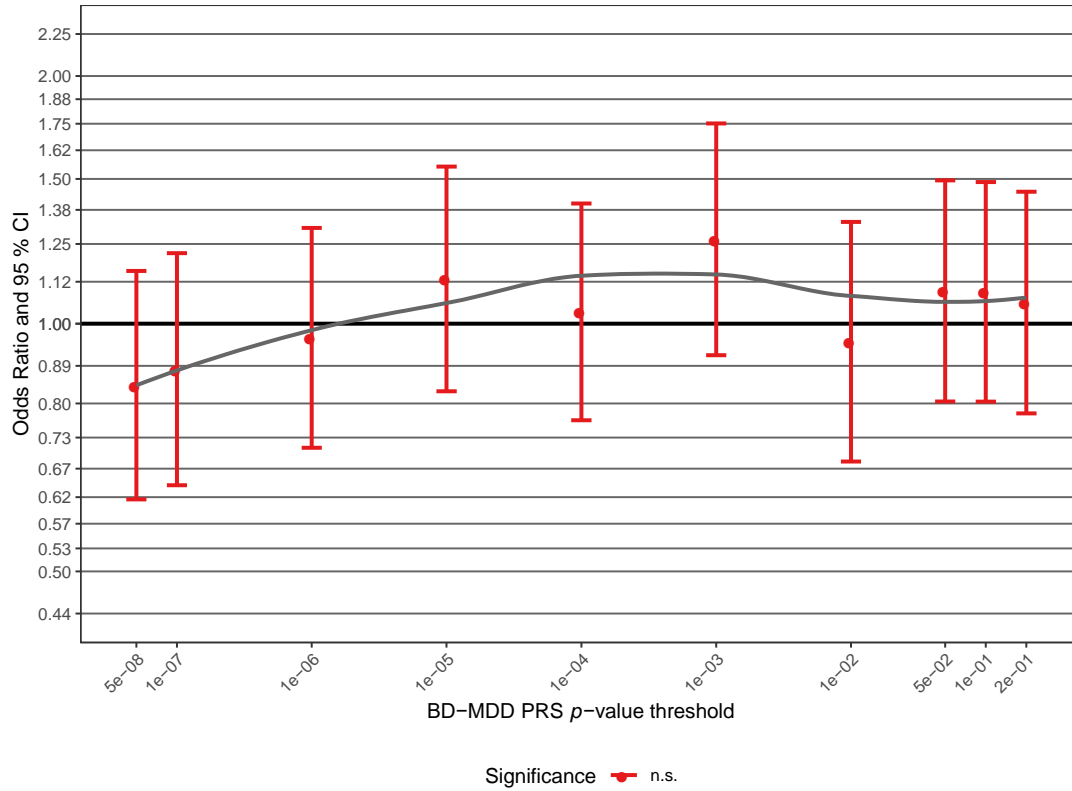
Supplementary Fig. S10F: Association of the MDD-BD GWIS PRS.



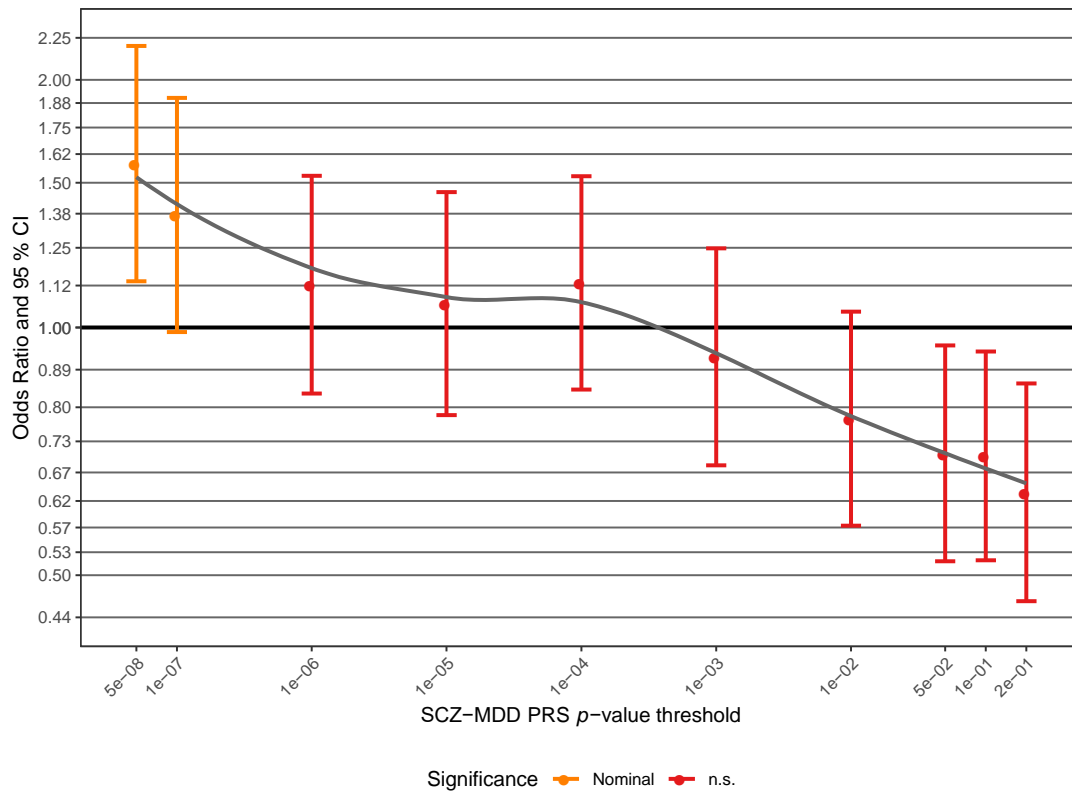
Supplementary Fig. S10G: Association of the MDD-SCZ GWIS PRS.



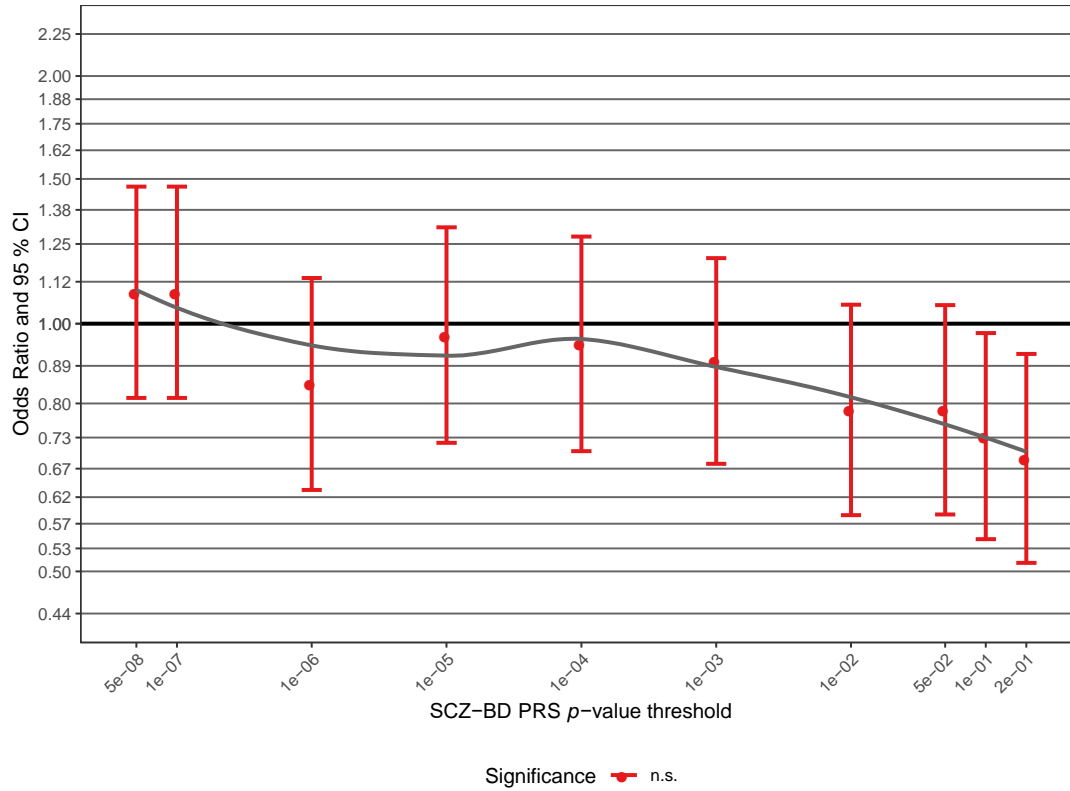
Supplementary Fig. S10H: Association of the BD-MDD GWIS PRS.



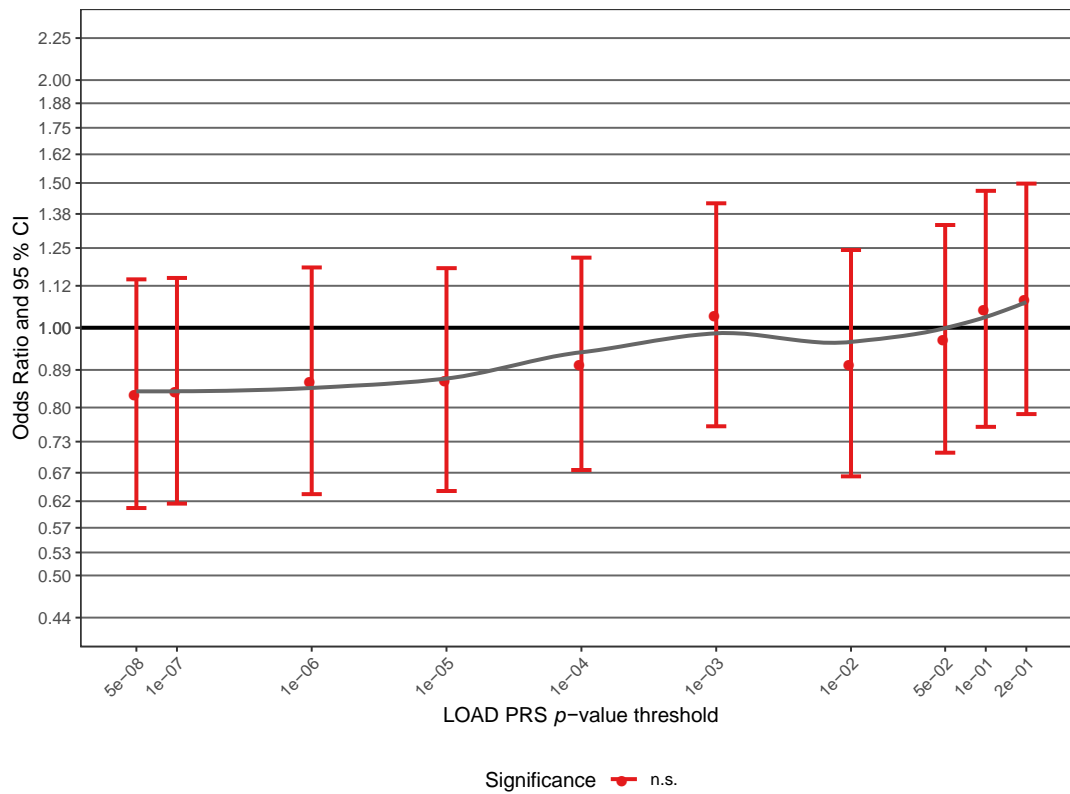
Supplementary Fig. S10I: Association of the SCZ-MDD GWIS PRS.



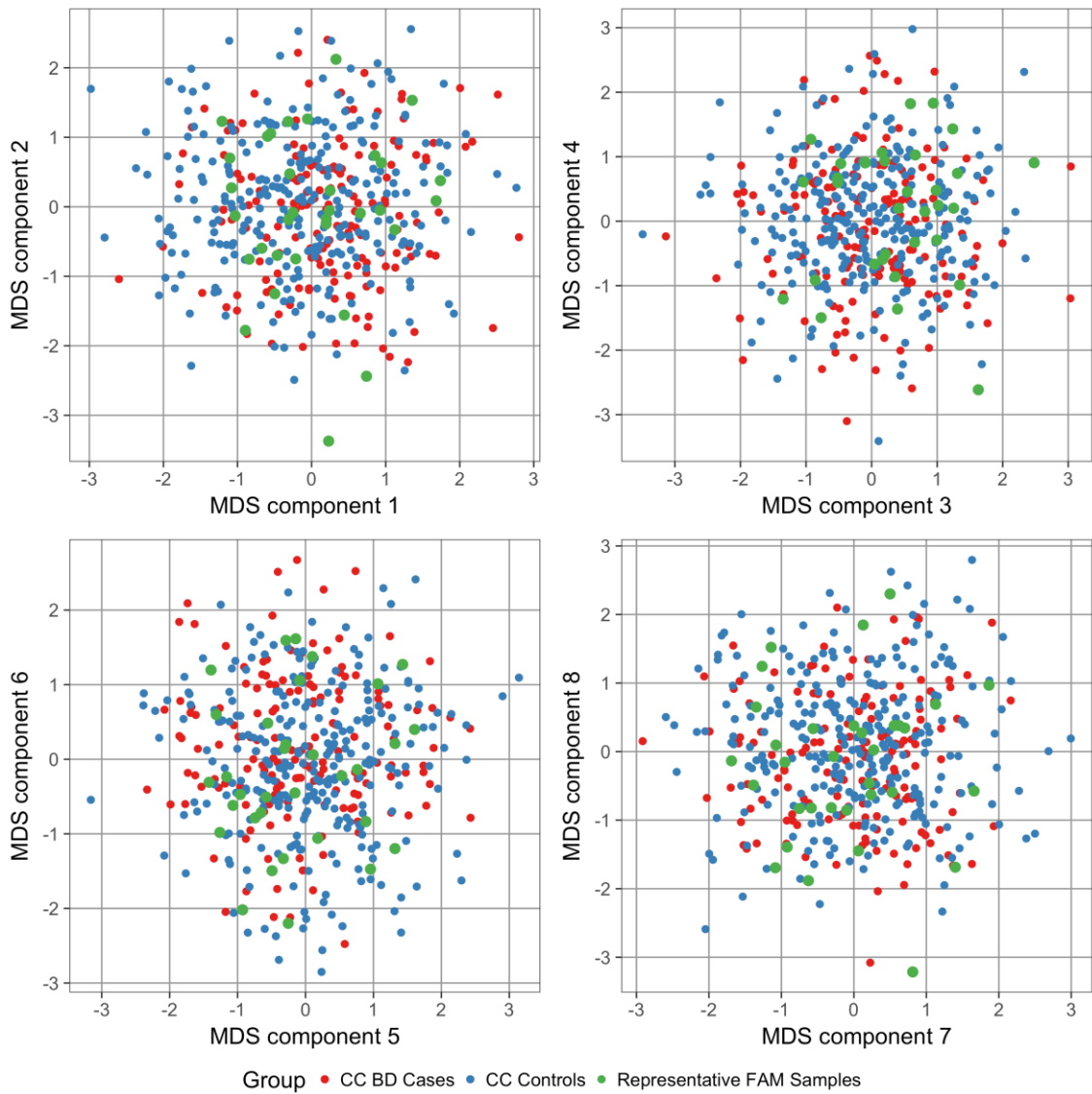
Supplementary Fig. S10J: Association of the SCZ-BD GWIS PRS.



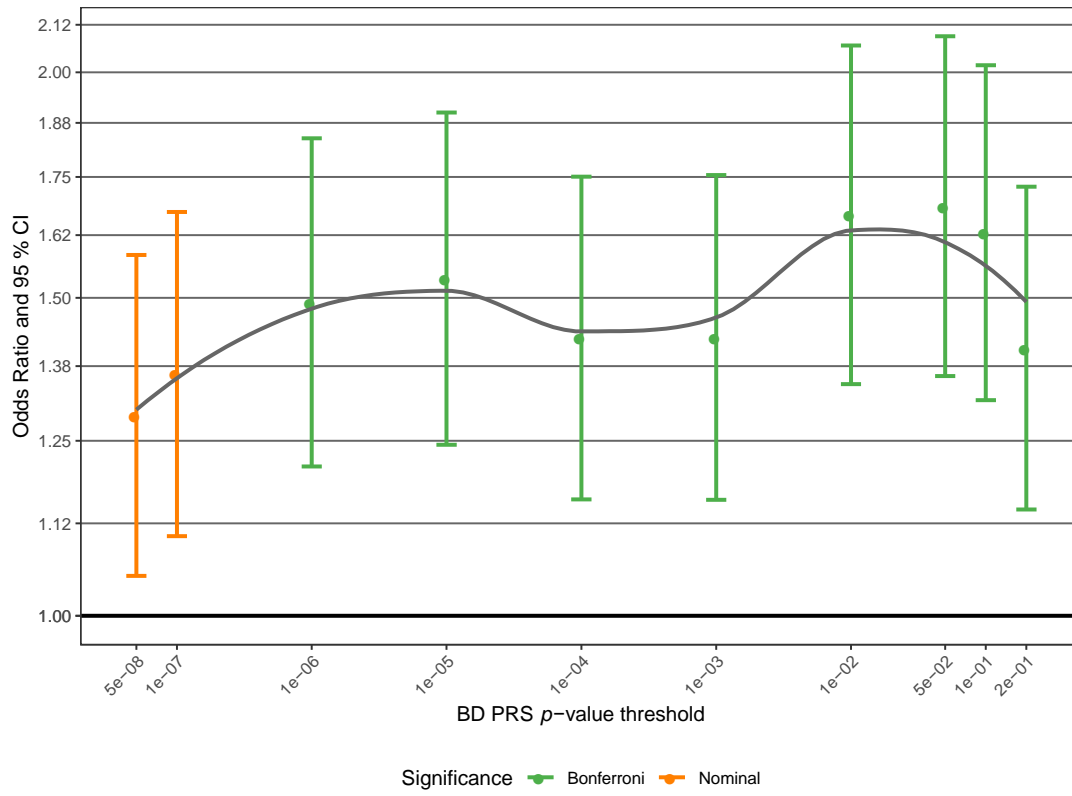
Supplementary Fig. S10K: Association of the LOAD PRS.



Supplementary Fig. S11: Population substructure analysis. Details regarding the generation of MDS components and the population substructure analysis are described above in the Supplementary Methods. The axes have been scaled to show standard deviations.



Supplementary Fig. S12: Association analysis comparing BD PRS in unrelated CC_{BD} cases and $CC_{controls}$. Details of the plot are described in the legend for Fig. 1. Covariate used: Sex. Full association test statistics including p -values are shown in Supplementary Table S11.



IGAP Supplementary Methods and Acknowledgments

IGAP Methods for the LOAD GWAS

International Genomics of Alzheimer's Project (IGAP) is a large two-stage study based upon genome-wide association studies (GWAS) on individuals of European ancestry. In stage 1, IGAP used genotyped and imputed data on 7,055,881 single nucleotide polymorphisms (SNPs) to meta-analyze four previously-published GWAS datasets consisting of 17,008 Alzheimer's disease cases and 37,154 controls (The European Alzheimer's disease Initiative – EADI, The Alzheimer Disease Genetics Consortium – ADGC, The Cohorts for Heart and Aging Research in Genomic Epidemiology consortium – CHARGE, The Genetic and Environmental Risk in AD consortium – GERAD). In stage 2, 11,632 SNPs were genotyped and tested for association in an independent set of 8,572 Alzheimer's disease cases and 11,312 controls. Finally, a meta-analysis was performed combining results from stages 1 & 2.

The present study used GWAS summary statistics from stage 1 for the calculation of PRS.

IGAP Acknowledgments

We thank the International Genomics of Alzheimer's Project (IGAP) for providing summary results data for these analyses. The investigators within IGAP contributed to the design and implementation of IGAP and/or provided data but did not participate in analysis or writing of this report. IGAP was made possible by the generous participation of the control subjects, the patients, and their families. The i-Select chips was funded by the French National Foundation on Alzheimer's disease and related disorders. EADI was supported by the LABEX (laboratory of excellence program investment for the future) DISTALZ grant, Inserm, Institut Pasteur de Lille, Université de Lille 2 and the Lille University Hospital. GERAD was supported by the Medical Research Council (Grant n° 503480), Alzheimer's Research UK (Grant n° 503176), the Wellcome Trust (Grant n° 082604/2/07/Z) and German Federal Ministry of Education and Research (BMBF): Competence Network Dementia (CND) grant n° 01GI0102, 01GI0711, 01GI0420. CHARGE was partly supported by the NIH/NIA grant R01 AG033193 and the NIA AG081220 and AGES contract N01-AG-12100, the NHLBI grant R01 HL105756, the Icelandic Heart Association, and the Erasmus Medical Center and Erasmus University. ADGC was supported by the NIH/NIA grants: U01 AG032984, U24 AG021886, U01 AG016976, and the Alzheimer's Association grant ADGC-10-196728.

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Thomas D Als 15,16,19
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Marie Bækvad-Hansen 19,35
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Richard Belliveau 12
Sarah E Bergen 38
Carsten Bøcker Pedersen 19,28,29
Erlend Bøen 39
Marco Boks 40
James Boocock 41
Monika Budde 42
William Bunney 43
Margit Burmeister 44
Jonas Bybjerg-Grauholm 19,35
William Byerley 45
Miquel Casas 46,47,48,49
Felecia Cerrato 12
Pablo Cervantes 50
Kimberly Chambert 12
Alexander W Charney 2
Danfeng Chen 12
Claire Churchhouse 12,14
Toni-Kim Clarke 51
William Coryell 52
David W Craig 53
Cristiana Cruceanu 50,54
Piotr M Czerski 55
Anders M Dale 56,57,58,59
Simone de Jong 4,5
Franziska Degenhardt 8,9
Jurgen Del-Favero 60
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Srdjan Djurovic 62,63
Amanda L Dobbyn 1,2
Ashley Dumont 12
Torbjørn Elvsåshagen 64,65
Valentina Escott-Price 27
Chun Chieh Fan 59
Sascha B Fischer 6,10
Matthew Flickinger 66
Tatiana M Foroud 67
Liz Forty 27
Josef Frank 68
Christine Fraser 27
Nelson B Freimer 69
Louise Frisén 70,71,72
Katrín Gade 42,73
Diane Gage 12
Julie Garnham 74
Claudia Giambartolomei 41
Marianne Giørtz Pedersen 19,28,29
Jaqueline Goldstein 12
Scott D Gordon 75
Katherine Gordon-Smith 76
Elaine K Green 77
Melissa J Green 78
Tiffany A Greenwood 58
Jakob Grove 15,16,19,79
Weihua Guan 80
José Guzman Parra 81
Marian L Hamshere 27
Martin Hautzinger 82
Urs Heilbronner 42
Stefan Herms 6,8,9,10
Maria Hipolito 83
Per Hoffmann 6,8,9,10
Dominic Holland 56,84
Laura Huckins 1,2
Stéphane Jamain 85,86
Jessica S Johnson 1,2
Anders Juréus 38
Radhika Kandaswamy 4
Robert Karlsson 38
James L Kennedy 87,88,89,90
Sarah Kittel-Schneider 91
James A Knowles 92,93
Manolis Kogevinas 94
Anna C Koller 8,9

Ralph Kupka 95,96,97
 Catharina Lavebratt 70
 Jacob Lawrence 98
 William B Lawson 83
 Markus Leber 99
 Phil H Lee 12,14,100
 Shawn E Levy 101
 Jun Z Li 102
 Chunyu Liu 103
 Susanne Lucae 104
 Anna Maaser 8,9
 Donald J MacIntyre 105,106
 Pamela B Mahon 61,107
 Wolfgang Maier 108
 Lina Martinsson 71
 Steve McCarroll 12,109
 Peter McGuffin 4
 Melvin G McInnis 110
 James D McKay 111
 Helena Medeiros 93
 Sarah E Medland 75
 Fan Meng 30,110
 Lili Milani 112
 Grant W Montgomery 25
 Derek W Morris 113,114
 Thomas W Mühleisen 6,115
 Niamh Mullins 4
 Hoang Nguyen 1,2
 Caroline M Nievergelt 58,116
 Annelie Nordin Adolfsson 117
 Evaristus A Nwulia 83
 Claire O'Donovan 74
 Loes M Olde Loohuis 69
 Anil P S Ori 69
 Lilijana Oruc 118
 Urban Ösby 119
 Roy H Perlis 120,121
 Amy Perry 76
 Andrea Pfennig 37
 James B Potash 61
 Shaun M Purcell 2,107
 Eline J Regeer 122
 Andreas Reif 91
 Céline S Reinbold 6,10
 John P Rice 123
 Alexander L Richards 27
 Fabio Rivas 81
 Margarita Rivera 4,124
 Panos Roussos 1,2,125
 Douglas M Ruderfer 126
 Euijung Ryu 127
 Cristina Sánchez-Mora 46,47,49
 Alan F Schatzberg 128
 William A Scheftner 129
 Nicholas J Schork 130
 Cynthia Shannon Weickert 78,131
 Tatyana Shehktman 58
 Paul D Shilling 58
 Engilbert Sigurdsson 132
 Claire Slaney 74
 Olav B Smeland 56,133,134
 Janet L Sobell 135
 Christine Søholm Hansen 19,35
 Anne T Spijker 136
 David St Clair 137
 Michael Steffens 138
 John S Strauss 89,139
 Fabian Streit 68
 Jana Strohmaier 68
 Szabolcs Szelinger 140
 Robert C Thompson 110
 Thorgerir E Thorgerirsson 23
 Jens Treutlein 68
 Helmut Vedder 141
 Weiqing Wang 1,2
 Stanley J Watson 110
 Thomas W Weickert 78,131
 Stephanie H Witt 68
 Simon Xi 142
 Wei Xu 143,144
 Allan H Young 145
 Peter Zandi 146
 Peng Zhang 147
 Sebastian Zollner 110
 Rolf Adolfsson 117
 Ingrid Agartz 17,39,148
 Martin Alda 74,149
 Lena Backlund 71
 Bernhard T Baune 150
 Frank Bellivier 151,152,153,154
 Wade H Berrettini 155
 Joanna M Biernacka 127
 Douglas H R Blackwood 51
 Michael Boehnke 66
 Anders D Børghlum 15,16,19
 Aiden Corvin 114
 Nicholas Craddock 27
 Mark J Daly 12,14
 Udo Dannlowski 156
 Tõnu Esko 3,109,112,157
 Bruno Etain 151,153,154,158
 Mark Frye 159
 Janice M Fullerton 131,160
 Elliot S Gershon 32,161
 Michael Gill 114
 Fernando Goes 61
 Maria Grigoriu-Serbanescu 162
 Joanna Hauser 55
 David M Hougaard 19,35
 Christina M Hultman 38

Ian Jones 27
Lisa A Jones 76
René S Kahn 2,40
George Kirov 27
Mikael Landén 38,163
Marion Leboyer 86,151,164
Cathryn M Lewis 4,5,165
Qingqin S Li 166
Jolanta Lissowska 167
Nicholas G Martin 75,168
Fermin Mayoral 81
Susan L McElroy 169
Andrew M McIntosh 51,170
Francis J McMahon 171
Ingrid Melle 172,173
Andres Metspalu 112,174
Philip B Mitchell 78
Gunnar Morken 175,176
Ole Mors 19,177
Preben Bo Mortensen 15,19,28,29
Bertram Müller-Myhsok 54,178,179
Richard M Myers 101
Benjamin M Neale 3,12,14
Vishwajit Nimgaonkar 180
Merete Nordentoft 19,181
Markus M Nöthen 8,9
Michael C O'Donovan 27
Ketil J Oedegaard 182,183
Michael J Owen 27
Sara A Paciga 184
Carlos Pato 93,185
Michele T Pato 93

Danielle Posthuma 22,186
Josep Antoni Ramos-Quiroga 46,47,48,49
Marta Ribasés 46,47,49
Marcella Rietschel 68
Guy A Rouleau 187,188
Martin Schalling 70
Peter R Schofield 131,160
Thomas G Schulze 42,61,68,73,171
Alessandro Serretti 189
Jordan W Smoller 12,190,191
Hreinn Stefansson 23
Kari Stefansson 23,192
Eystein Stordal 193,194
Patrick F Sullivan 38,195,196
Gustavo Turecki 197
Arne E Vaaler 198
Eduard Vieta 199
John B Vincent 139
Thomas Werge 19,200,201
John I Nurnberger 202
Naomi R Wray 24,25
Arianna Di Florio 27,196
Howard J Edenberg 203
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Lynsey S Hall 10, 52
Christine Søholm Hansen 13, 18
Thomas F Hansen 53, 54, 55
Stefan Herms 35, 47
Ian B Hickie 56
Per Hoffmann 35, 47
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David M Howard 10, 28
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Isaac S Kohane 64, 65, 66
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Warren W. Kretschmar 67
Zoltán Kutalik 68, 69
Yihan Li 67
Penelope A Lind 29
Donald J MacIntyre 70, 71
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Robert M Maier 2
Wolfgang Maier 72
Jonathan Marchini 73
Hamdi Mbarek 9
Patrick McGrath 74
Peter McGuffin 28
Sarah E Medland 29
Divya Mehta 2, 75
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Roseann E Peterson 17, 85
Erik Pettersson 22
Wouter J Peyrot 19
Giorgio Pistis 27
Danielle Posthuma 86, 87
Jorge A Quiroz 88

Per Qvist 8, 13, 24
 John P Rice 89
 Brien P. Riley 17
 Margarita Rivera 28, 90
 Saira Saeed Mirza 36
 Robert Schoevers 91
 Eva C Schulte 92, 93
 Ling Shen 62
 Jianxin Shi 94
 Stanley I Shyn 95
 Engilbert Sigurdsson 96
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 Hreinn Stefansson 99
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 Alexander Teumer 102
 Wesley Thompson 13, 54, 103, 104
 Pippa A Thomson 105
 Thorgeir E Thorgeirsson 99
 Matthew Traylor 106
 Jens Treutlein 45
 Vassily Trubetskoy 4
 André G Uitterlinden 107
 Daniel Umbricht 108
 Sandra Van der Auwera 109
 Albert M van Hemert 110
 Alexander Viktorin 22
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 Yunpeng Wang 13, 54, 104
 Bradley T. Webb 111
 Shantel Marie Weinsheimer 13, 54
 Jürgen Wellmann 101
 Gonneke Willemsen 9
 Stephanie H Witt 45
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 Hualin S Xi 112
 Jian Yang 2, 113
 Futao Zhang 1
 Volker Arolt 114
 Bernhard T Baune 115, 116, 117
 Klaus Berger 101
 Dorret I Boomsma 9
 Sven Cichon 35, 47, 118, 119
 Udo Dannlowski 114
 EJC de Geus 9, 120
 J Raymond DePaulo 50
 Enrico Domenici 121
 Katharina Domschke 122, 123
 Tõnu Esko 5, 78
 Hans J Grabe 109
 Steven P Hamilton 124
 Caroline Hayward 125
 Andrew C Heath 89
 Kenneth S Kendler 17
 Stefan Kloiber 59, 126, 127
 Glyn Lewis 128
 Qingqin S Li 129
 Susanne Lucae 59
 Pamela AF Madden 89
 Patrik K Magnusson 22
 Nicholas G Martin 29
 Andrew M McIntosh 10, 34
 Andres Metspalu 78, 130
 Ole Mors 13, 131
 Preben Bo Mortensen 11, 12, 13, 24
 Bertram Müller-Myhsok 15, 132, 133
 Merete Nordentoft 13, 134
 Markus M Nöthen 35
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 Roy H Perlis 38, 136
 David J Porteous 105
 James B Potash 137
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 Thomas G Schulze 45, 93, 138, 139, 140
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 Kari Stefansson 99, 141
 Henning Tiemeier 36, 142, 143
 Rudolf Uher 144
 Henry Völzke 102
 Myrna M Weissman 74, 145
 Thomas Werge 13, 54, 146
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