THE LANCET

Supplementary appendix

This appendix formed part of the original submission and has been peer reviewed. We post it as supplied by the authors.

Supplement to: 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; published online Feb 28. http://dx.doi.org/10.1016/S0140-6736(12)62129-1.

Supplemental Material

I. Quality Control and Analysis Pipeline

Briefly, SNPs were initially removed for missingness > 5% within each study, followed by removal of individuals with genotype missingness exceeding 2%. Remaining SNPs with missing rates > 2% were then removed as were SNPs with case-control difference in missingness > 2%. Finally, we also removed SNPs with allele frequency difference > 15% compared to HapMap CEU population or for which Hardy-Weinberg equilibrium $p < 10^{-6}$ among controls. Pictures of the first two principal components can be found in Figure S1. Genetic quality control included relatedness testing and principal components analyses. Relatedness testing was done with PLINK (see URLs), reporting pairs with genome identity (pi-hat) > 0.9 as 'identical samples' and with pi-hat > 0.2 as being closely related. After random shuffling, one ID from each pair was excluded from downstream analysis. From groups with multiple related pairs (for example, a family or multiple use of NIMH controls), only one individual was kept. For the family-based samples, we recoded any Mendelian error as missing and checked the parent-offspring relationships via identity-by-state. For all pairs of samples included in the analysis, we estimated the IBD relationship using PLINK. [1] This enabled us to identify relatives and sample duplicates across the entire dataset, so as to prevent the increase in false positives and overdispersion of the combined test statistic that would result from violating the assumptions of independence. Siblings in family based samples were retained.

For meta-analysis of case control samples, the Z-score is generated by regressing phenotype on the dosage of the genotype, including seven multi-

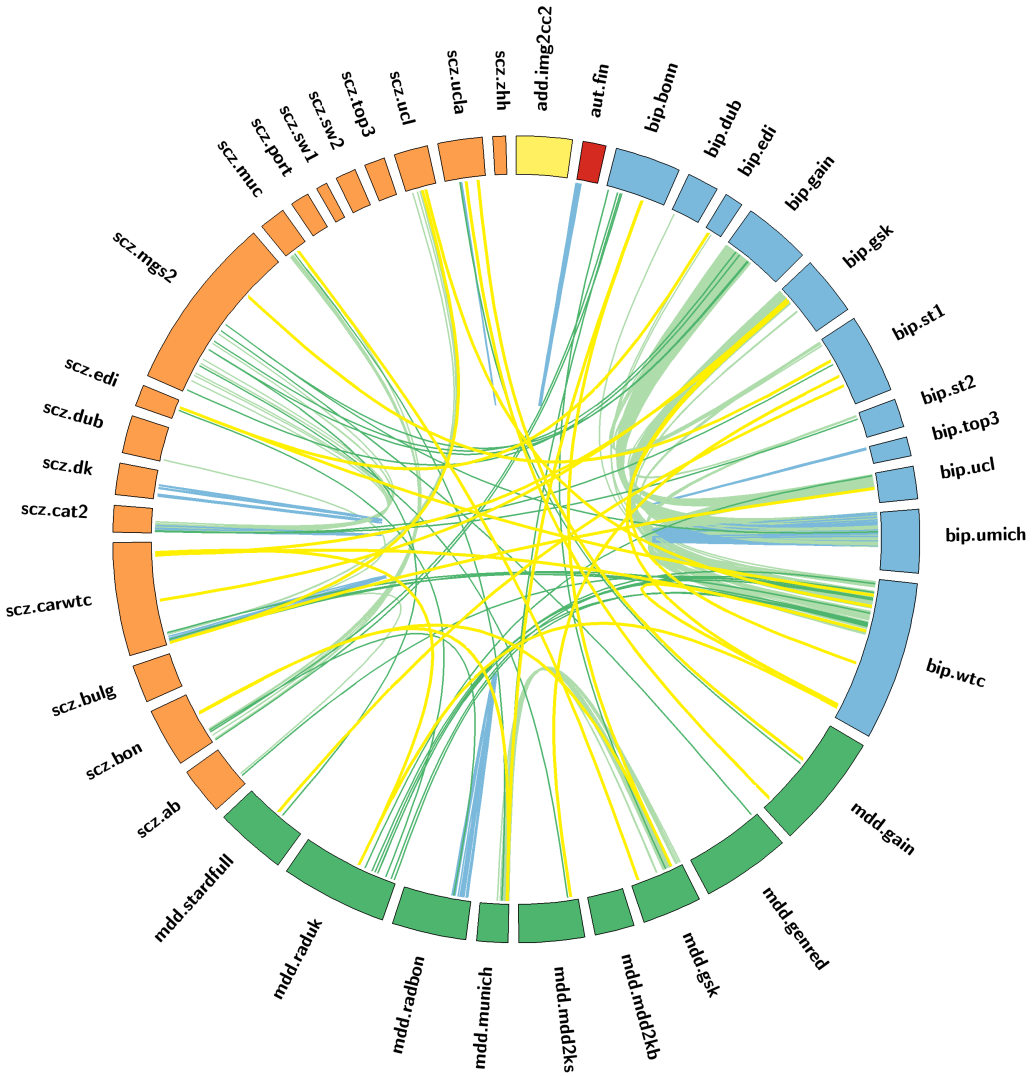
dimensional scaling (MDS) dimensions as covariates. [2] For the family-based samples, we applied a case-pseudocontrol approach by using the non-transmitted alleles at each position as the control for each case. Families with more than one offspring were broken into independent trios with each affected appearing as the offspring of an 'independent' trio with parental genotypes replicated. We ran logistic regression analysis on the cases and pseudo-controls without the MDS as the transmitted and non-transmitted haplotypes come from the same population.

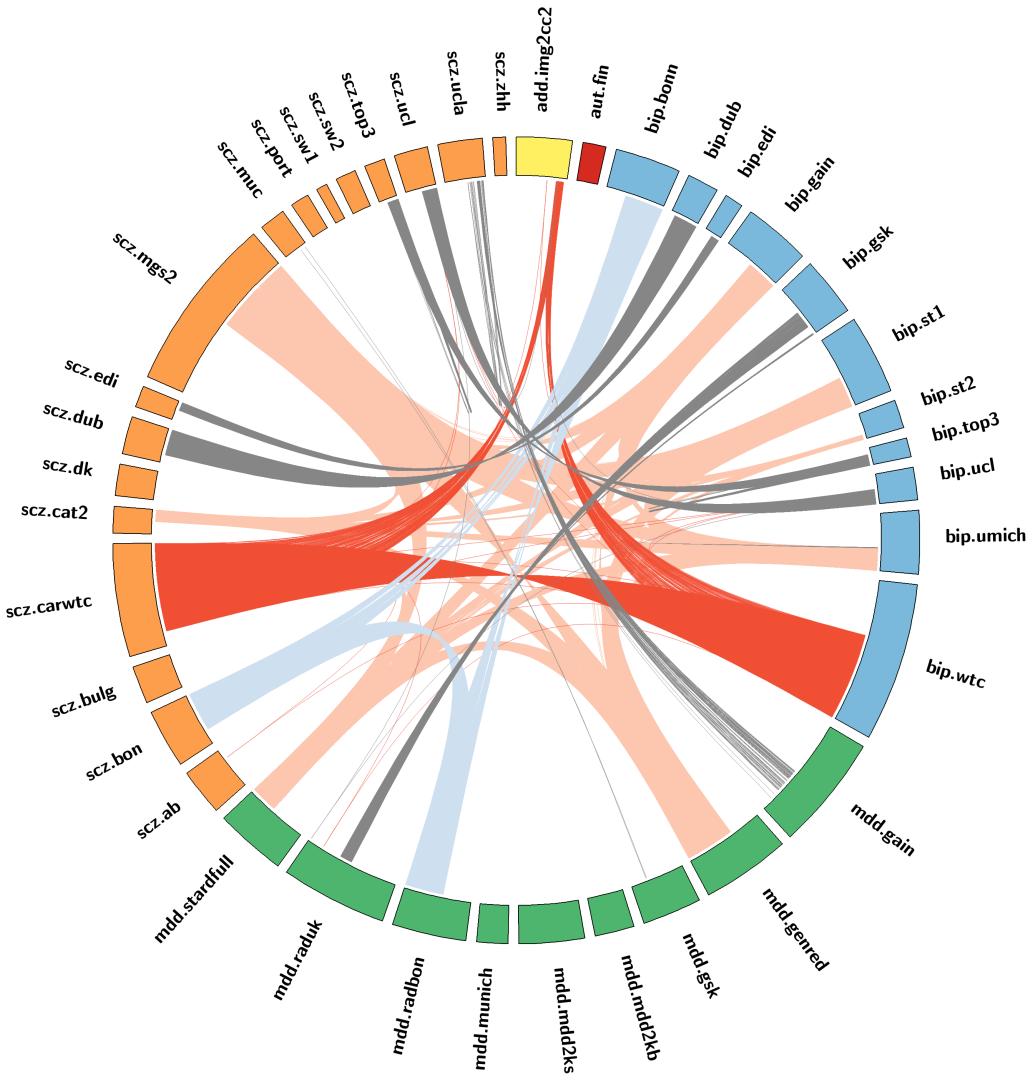
Using Beagle 3.0.4,[3] we performed imputation within each study in batches of 300 randomly drawn individuals in order to keep the same case-control ratio as in the total sample from that study. As a reference panel, we used the CEU+TSI Hapmap Phase 3 data with 410 phased haplotypes encompassing 1,252,901 SNPs.

II. Figure S1: Overlap of cases and controls from contributing datasets

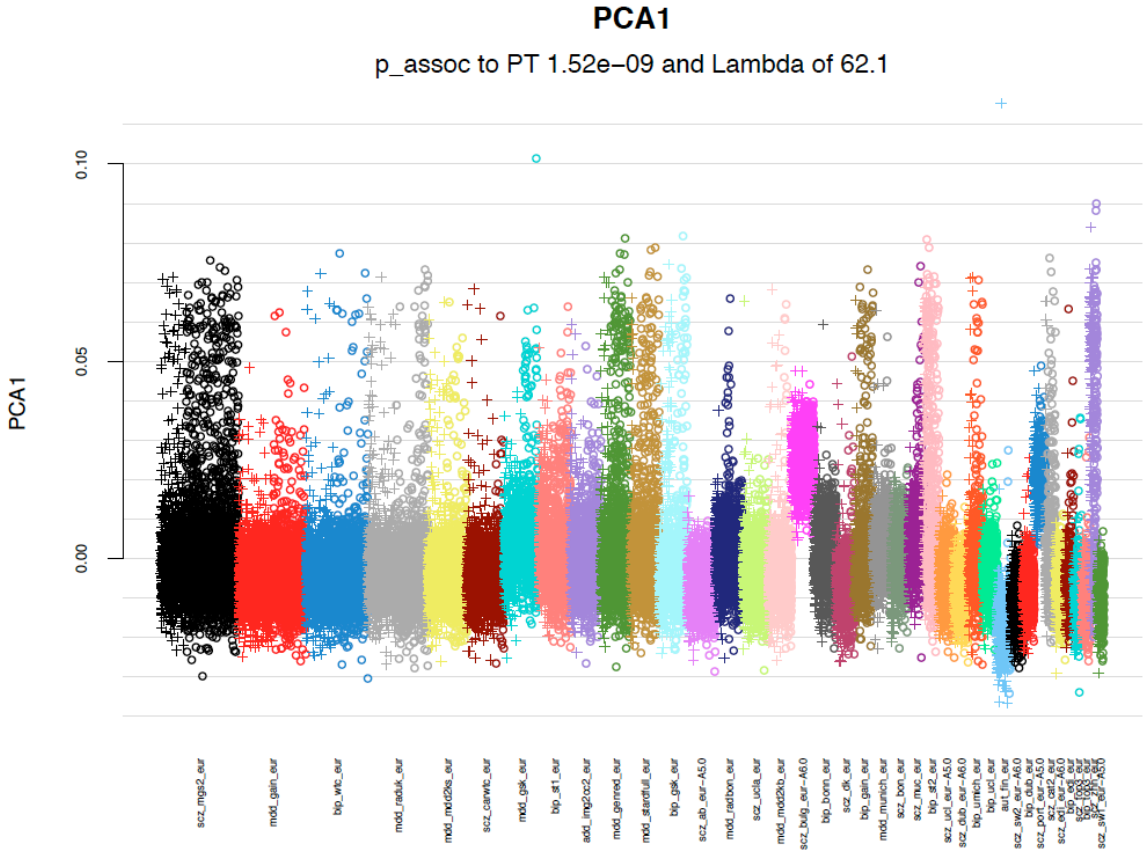
Overlapping or related cases (top) and controls (bottom) are shown as connecting lines between

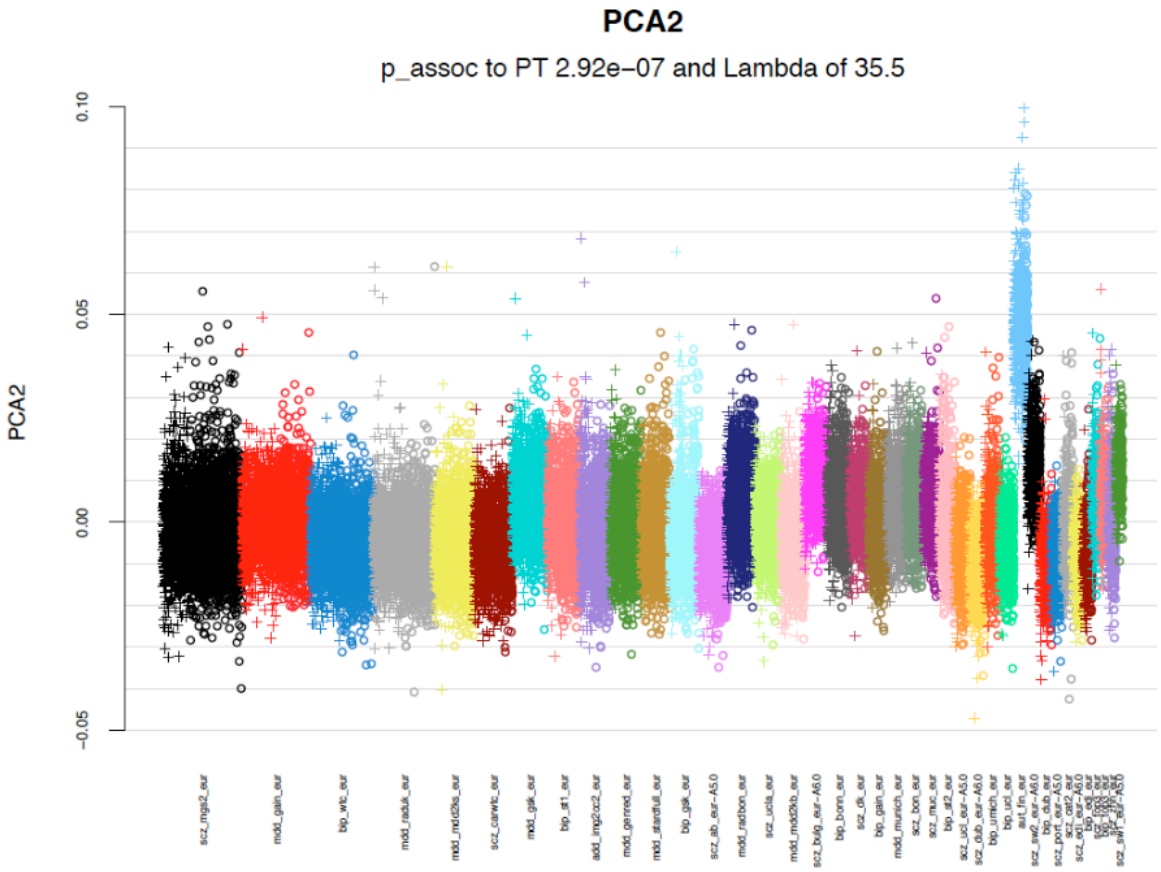
samples.


add.img2cc2
aut.fin
bip.bonn
bip.dub
bip.edi
bip.gain
bip.gsk
bip.st1
bip.st2
bip.top3
bip.ucl
bip.umich
bip.wtc
mdd.gain
mdd.genred
mdd.gsk
mdd.mdd2kb
mdd.mdd2ks
mdd.munich
mdd.radbon
mdd.raduk
mdd.stardfull
scz.ab
scz.bon
scz.bulg
scz.carwtc
scz.cat2
scz.dk
scz.dub
scz.edi
scz.mgs2
scz.muc
scz.port
scz.sw1
scz.sw2
scz.top3
scz.ucl
scz.ucla
scz.zhh


add.img2cc2
aut.fin
bip.bonn
bip.dub
bip.edi
bip.gain
bip.gsk
bip.st1
bip.st2
bip.top3
bip.ucl
bip.umich
bip.wtc
mdd.gain
mdd.genred
mdd.gsk
mdd.mdd2kb
mdd.mdd2ks
mdd.munich
mdd.radbon
mdd.raduk
mdd.stardfull
scz.ab
scz.bon
scz.bulg
scz.carwtc
scz.cat2
scz.dk
scz.dub
scz.edi
scz.mgs2
scz.muc
scz.port
scz.sw1
scz.sw2
scz.top3
scz.ucl
scz.ucla
scz.zkh

III. Supplementary Figure S2: Multidimensional scaling plot of identity-by state distances: Plot of first two MDS components by sample.





IV. Cross-Disorder Modeling Analysis

To characterize the specificity of allelic effects for loci demonstrating association in the primary meta analyses or 5 d.f. meta-analysis, and for loci associated with BPD or SCZ in recent disorder-specific PGC meta-analyses[4, 5], we applied a model selection approach as previously described. [6, 7] In brief, this approach involves a single genomewide omnibus test of association across disorders followed by a model selection approach that asks which configuration of

phenotypes is most likely to be associated with a given variant. The omnibus test consists of a single likelihood ratio test computed from a multinomial logistic regression in which allele frequencies at a given SNP can vary for each case-control disorder group compared to a null model in which allele frequencies are the same across all disorder groups. This is essentially a test of whether there is any association between the SNP and any of the five target disorders, but minimizes the experiment-wise Type I error rate compared to the alternative of testing all possible subsets of disorders. As shown by Lee et al. [6], the omnibus test has equivalent or greater power compared to individual tests of disorder subsets.

The omnibus test is followed by fitting log-linear or multinomial logistic regression models corresponding to a range of causal models that characterize the effect of a given allele on one or more of the target disorders. These models specify risk effect configurations that vary or are constrained to be equal among subsets of the disorders. Then the best-fitting model is identified according to two commonly used information criteria: the Bayesian Information Criterion (BIC) or the Akaike Information Criterion (AIC). The model with the lowest BIC (and/or AIC) is selected as the best-fit model. The use of these information criteria allows comparison of non-nested models.

In the present analysis, we modified this approach as follows. First, because our primary analysis consisted of the single five-disorder meta-analysis, we do not additionally report the results of the omnibus test. Second, for the model-fitting procedure, we relied on prior epidemiologic and genetic evidence of disorder relationships to predefine a set of causal models for comparison rather than

considering all possible models. Third, the best-fit model results we present are based on the BIC criterion. Finally, we do not predict the best model if the information criterion measure differences do not differ significantly between the best-fit model and the second-best-fit model. As suggested in [8], we use the absolute score difference of greater than two as the significant superiority of the best model fitness compared to the others. We acknowledge that alternative best-fit models might be selected by use of the AIC or other criteria.

Specifically, we defined 13 cross-disorder risk models corresponding to the following 13 patterns of allele frequency configurations across attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), bipolar disorder (BP), major depressive disorder (MDD), schizophrenia (SCZ), and control (CTRL) subject groups: 1) *ADHD specific* risk (ADHD ≠ [ASD=BP=MDD=SCZ=CTRL]); 2) *ASD specific* risk (ASD ≠ [ADHD=BP=MDD=SCZ=CTRL]); 3) *BP specific* risk (BP ≠ [ADHD=ASD=MDD=SCZ=CTRL]); 4) *MDD specific* risk (MDD ≠ [ADHD=ASD=BP=SCZ=CTRL]); 5) *SCZ specific* risk (SCZ ≠ [ADHD=ASD=BP=MDD= CTRL]); 6) *shared BP* and *MDD* risk ([BP=MDD] ≠ [ADHD=ASD=SCZ=CTRL]); 7) *shared* BP and SCZ ([BP=SCZ] ≠ [ADHD=ASD=MDD=CTRL]); 8) *shared MDD* and *SCZ* ([MDD =SCZ] ≠ [ADHD = ASD=BP=CTRL]); 9) *shared ADHD* and *BP* ([ADHD= BP] ≠ [ASD= MDD= SCZ=CTRL]); 10) *shared ASD* and *SCZ* ([ASD= SCZ] ≠ [ADHD=BP=MDD=CTRL]); 11) *shared ADHD* and *ASD* ([ADHD =ASD] ≠ [BP=MDD=SCZ=CTRL]); 12) *shared BP, MDD* and *SCZ* ([BP=MDD= SCZ] ≠ [ADHD=ASD=CTRL]); 13) risk shared by all 5 disorders ([ADHD=ASD=BP=MDD=SCZ] ≠ [CTRL]) . Table S3 summarizes the parameters that

were fit for the 13 risk models. We selected the best-fit model among 13 based on the Bayesian information criterion (BIC) [2]. The selected model indicates the pleiotropic disorder model(s) providing the best fit for genotype-phenotype association for a given variant. Tables S4-S5 summarize the modeling analysis results for the top SNPs from the primary meta-analyses and the 5 degree of freedom tests, respectively. Supplementary Figures S4, S5, and S6 show the BIC scores of 13 cross-disorder models of the examined SNPs for the primary meta-analyses and top BP and SCZ PGC GWAS analyses.

TABLE S1. Cross disorder risk models

Category	Model	Risk Effect Parameter Setting					
		ADHD	**ASD**	**BP**	**MDD**	**SCZ**	**Control**
One Disorder Risk Model	ADHD	$\boldsymbol{\alpha_1}$	α_0	α_0	α_0	α_0	α_0
	ASD	α_0	$\boldsymbol{\alpha_1}$	α_0	α_0	α_0	α_0
	BP	α_0	α_0	$\boldsymbol{\alpha_1}$	α_0	α_0	α_0
	MDD	α_0	α_0	α_0	$\boldsymbol{\alpha_1}$	α_0	α_0
	SCZ	α_0	α_0	α_0	α_0	$\boldsymbol{\alpha_1}$	α_0
Two Disorder Risk Model	BP & MDD	α_0	α_0	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	α_0	α_0
	BP & SCZ	α_0	α_0	$\boldsymbol{\alpha_1}$	α_0	$\boldsymbol{\alpha_1}$	α_0
	MDD & SCZ	α_0	α_0	α_0	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	α_0
	ADHD & BP	$\boldsymbol{\alpha_1}$	α_0	$\boldsymbol{\alpha_1}$	α_0	α_0	α_0
	ASD & SCZ	α_0	$\boldsymbol{\alpha_1}$	α_0	α_0	$\boldsymbol{\alpha_1}$	α_0
	ADHD & ASD	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	α_0	α_0	α_0	α_0
Three Disorder Risk Model	BP, MDD & SCZ	α_0	α_0	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	α_0
All Disorder Risk Model	5 Disorder	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	$\boldsymbol{\alpha_1}$	α_0
		$\boldsymbol{\alpha_1}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\delta_1}$	$\boldsymbol{\varepsilon_1}$	α_0

* For each of five disorder groups and one control subject group, risk effect parameters are estimated from multinomial logistic regression. The same parameter setting across different groups constraints the same risk effect estimates for the corresponding disorder groups

V. Polygenic Risk Score Profie Analysis

We used risk score profiling to examine the aggregate polygenic sharing of allelic effects between pairs of disorders. For each of these pairs we defined one disorder in each pair as a "training" dataset and a second disorder as "target" dataset. We selected a filtered set of SNPs from the training results using "clumping". We first made a subset containing SNPs that met the following criteria: not AT or CG genotypes, minor allele frequency > 0.02, and imputation INFO score > 0.9. We then applied the --clump algorithm in PLINK with options: --clump-p1 1 --clump-p2 1 --clump-r2 0.25 --clump-kb 500. With these setting, SNPs with $r^2 < 0.25$ within 500 kb windows were retained, while filtering for the highest significance levels within linkage disequilibrium blocks. Due to the strong signal and high linkage disequilibrium in the MHC, only one SNP was kept from the extended MHC region.

We used PLINK's --score function to calculate the individual scores of the target set. To account for population stratification, the training analysis in the PGC data used the usual logistic regression framework including study indicator and significant MDS scores. In the target set, we estimated the variance explained in disease state by the difference in the Nagelkerke pseudo R^2 of an analysis including the score and covariates such as site and ancestry principal component scores vs. an analysis with the covariates alone. The significance level for each analysis was estimated via classical logistic regression analysis. From the full set of filtered SNPs, we evaluated 10 different association P thresholds (P_T): 0.0001, 0.001, 0.01, 0.05, 0.1, 0.2, 0.3, 0.4, 0.5, and 1.0 (i.e., including all SNPs).

VI. Pathway Analysis Methods

We examined enrichment of specific bio-molecular functions in which the top associated genes from the primary meta-analysis are commonly involved. This analysis used a collection of Gene Ontology (GO) terms and a set-based association test implemented in the INRICH software (http://atgu.mgh.harvard.edu/inrich). [8] First, we downloaded a collection of 14,793 Gene Ontology (GO) terms from the gene ontology consortium web site (http://www.geneontology.org/). [9] This GO gene set defines groups of biologically related genes in terms of their involvement in the same biological processes, cellular components and molecular functions, supported by diverse experimental and computational analysis evidence (www.geneontology.org/GO.evidence.shtml). Of 14,793 total GO terms, we restricted the analysis to terms with at least 5 human genes and not more than 200, leaving 6,600 GO terms (i.e., gene sets). Second, we obtained the gene2go dataset from NCBI (ftp.ncbi.nlm.nih.gov/gene/DATA/gene2go.gz) and mapped the Ensembl gene identifiers to gene symbols. We also downloaded a list of 26,914 human gene transcripts (hg18) from the UCSC Genome Browser database (http://genome.ucsc.edu/). Third, linkage-disequilibrium (LD)-based clumping was conducted to generate LD-independent association regions from the primary meta-analysis results using PLINK .[1] By applying a *p* value threshold of $1x10^{-3}$, 874 LD-independent genomic intervals were defined (clumping kb=1000; gene-border 35kb upstream, 10kb downstream; tag r^2=0.2). Any associated regions that are physically overlapping with each other or spanning the same gene were collapsed into one, so

that association signals from the same genes were not double-counted. Lastly, enrichment tests were conducted as follows: For each GO term, we counted the number of association intervals that intersected with at least one target gene annotated by that GO term. We then calculated for each term, the probability of observing at least the same number of intersecting intervals by chance alone, via a random-genomic shuffling permutation procedure. Specifically, our permutation procedure controlled potential biases due to different SNP and gene density, and gene sizes by matching the genomic characteristics of the randomly selected intervals to those of the originally associated genomic intervals. That is, the total number of SNPs, genes, and size of the random intervals were constrained to approximately match those of the originals within a factor of 0.9 to 1.1. We repeated this permutation procedure 100,000 times to generate a null distribution of the enrichment statistics. We then corrected for multiple testing of 6,600 gene sets by evaluating the distribution of minimum empirical *p*-values under the null hypothesis, given that 6,600 targets were tested. As shown in Tables S7, only a single GO term that was enriched in the top $P < 1 \times 10^{-3}$ association regions withstood experiment-wide correction for multiple testing: GO:0005262, *calcium channel activity* (http://amigo.geneontology.org/cgi-bin/amigo/term_details?term=GO:0005262). This gene set represents a group of 73 genes involved in the facilitated diffusion of calcium ions through a trans-membrane channel in response to changes in membrane potential. Among 73 genes, 2 genes on the X chromosome were excluded in the analysis, and overlapping genes were merged as one gene segment, resulting in 67 gene segments (CACNA2D4 and

CACNA1C combined; CACNG4 and CACNG1 combined; CACNG6, CACNG7, and CACNG8 combined; for details Table S9). Among the 67 gene regions, 20 regions are present in independent association-intervals. The probability of this enrichment (controlling for the total SNP and gene-density in the association-intervals) is *P* = 9.99e-06; the probability of observing an empirical p-value this small, given all the 6,600 targets tested, is *P* =0.03659. The associated twenty gene segments are: *CATSPER4, CACNA1E, CACNA1S, TMEM37, ITPR1, CACNA2D2, CACNA1D, GPM6A TRPM3, GRIN3A, CACNB2, CALHM1, CATSPER1, TPCN2, CACNA2D4-CACNA1C, ITPR2, TRPC4, PSEN1, CATSPER2,* and *GRIN2A* (see Table S8). Note that the two gene regions, *CACNA2D4* and *CACNA1C* intersect with each other, and were considered as one genic region to avoid inflated type I errors of pathway enrichment due to physically clustered genes[11,12].

VII. eQTL Enrichment Analysis

The primary hypothesis we examined here is that SNPs selected from the primary cross-disorder meta-analysis may influence susceptibility to psychiatric illnesses by affecting cis-regulatory gene expression in the human brain. To test the hypothesis, we conducted the eQTL enrichment test described in Nicolae et al.[10], assessing whether our CD-related SNPs are more likely to be cis-regulatory eQTL markers in cortical brain regions. This analysis used six eQTL datasets: three postmortem gene expression studies of brain cortex[11-13], one liver[14], one normal skin[15], and one lymphoblastoid cell line dataset[16]. In brief, for each eQTL dataset, 10,000 randomized SNP sets were generated, each of the same size as the examined CD-analysis derived SNP list (thresholded at p<1e-02, 1e-01, 2e-01, 3e-01, 4e-01, 5e-01), and matched on MAF distribution. Sampling was conducted through a random selection without replacement from all genic SNPs (within 1Mbases from transcription start/end sites) with imputation quality scores exceeding R2 = 0.8 in the primary meta-analysis. These simulations yielded an empirical eQTL enrichment P-value, calculated as the proportion of 10,000 randomized sets in which the number of eQTL SNPs selected in the set is at least the same as the originally observed number in the cross-disorder association SNP list. The same procedures were repeated using LD-pruned SNPs from association regions (i.e., PLINK LD clumping r2=0.8, clumping kb=500). Due to extensive LD, only one association region was retained for the MHC region on chr. 6:25000000..35000000.

The following table describes the eQTL datasets used in this analysis.

eQTL Data Summary	Brain Prefrontal Cortex (Colantuoni et al. [13])	Brain Cortex (Myers et al.[11])	Brain Cortex (Webster et al.[12])	Liver (Schadt et al.[14])	Skin (Ding et al.[15])	LPL (Stranger et al.[16])
Number of Subjects in the original eQTL Study	269	193	364	427	57	270
Total Number of SNPs examined in the original eQTL Study	625,439	366,140	502,627	782,476	438,670	2,200,000
Total Number of mRNA transcripts examined In the original eQTL Study	30,176	14,078	24,357	39,280	54,000	47,294
Number *of cis*-eQTL SNPs included in current analysis	1,628	3,240	1,571	1,268	6,250	7,015

Colantuoni et al. 2011[13] investigated the temporal dynamics and genetic control of mRNA expression in human prefrontal cortex of 269 healthy subjects from fetal to adult developmental stages. RNA expression profiles were produced for 49,152 probes, yielding 30,176 mRNA transcripts after QC procedures. Genotyping was conducted using Illumina BeadChips. After QC, 625,439 SNPs remained. Linear

regression-based QTL analyses were performed for every SNP-mRNA pair, and associations for 1,628 pairs surpassed genomewide significance after Bonferroni corections. The same analyses were repeated for African American (n=147) and Caucasian (n=112) samples separately, yielding 560 and 759 significant eQTL markers, respectively. Our analysis were based on the eQTL analysis results conducted for all samples obtained from the publication supplementary materials.

Webster et al. 2009[12] examined postmortem brain samples from 279 healthy subjects and 486 late-onset Alzheimer patients. Genotyping was conducted using the Affymetrix GeneChip 500K Array (502,627 SNPs). Expression profiles were generated using Illumina Human Refseq-8 BeadChip for 24,357 transcripts. After QC, eQTL analysis was done for 380,157 SNPs and 8,650 transcripts. The authors included an interaction term for diagnosis to capture differential effects based on disease status. Genome-wide significance for each transcript was calculated using sample-swapping-based permutation. Our analysis was based on the 1,571 eQTL markers without a significant interaction for diagnosis (obtained from the publication supplementary materials).

Myers et al. 2007[11] conducted eQTL analyses in three brain cortical regions: frontal, temporal and parietal. This study analyzed 193 neuropathologically normal subjects using the Affymetrix 500K Array for genotyping and the Illumina HumanRefseq-8 Expression Array for gene expression measurements. After QC, 366,140 SNPs were examined for the expression of 14,078 transcripts, resulting in

433 SNP–transcript pairs (99 transcripts) that showed significant cis-association (transcript-specific empirical P-value $p<0.05$). 25 of the pairs survived multiple testing correction using a Sidak approach.

Schadt et al.[14] analyzed 427 human liver samples to identify DNA variations associated with liver gene expression. Gene expression was measured for 39,280 transcripts using Agilent microarray and genotyping was done for 783,476 SNPs using Affymetrix 500K and Illumina 650Y arrays. eQTL association analysis was done using the Kruskal-wallis test to identify cis (<1Mb probe to SNP) and trans associations. Our analysis based on 1,268 cis eQTL markers with association $p < 1e-09$ downloaded from the GTEx (Genotype-Tissue Expression) eQTL Browser website (http://www.ncbi.nlm.nih.gov/gtex/GTEX2/gtex.cgi).

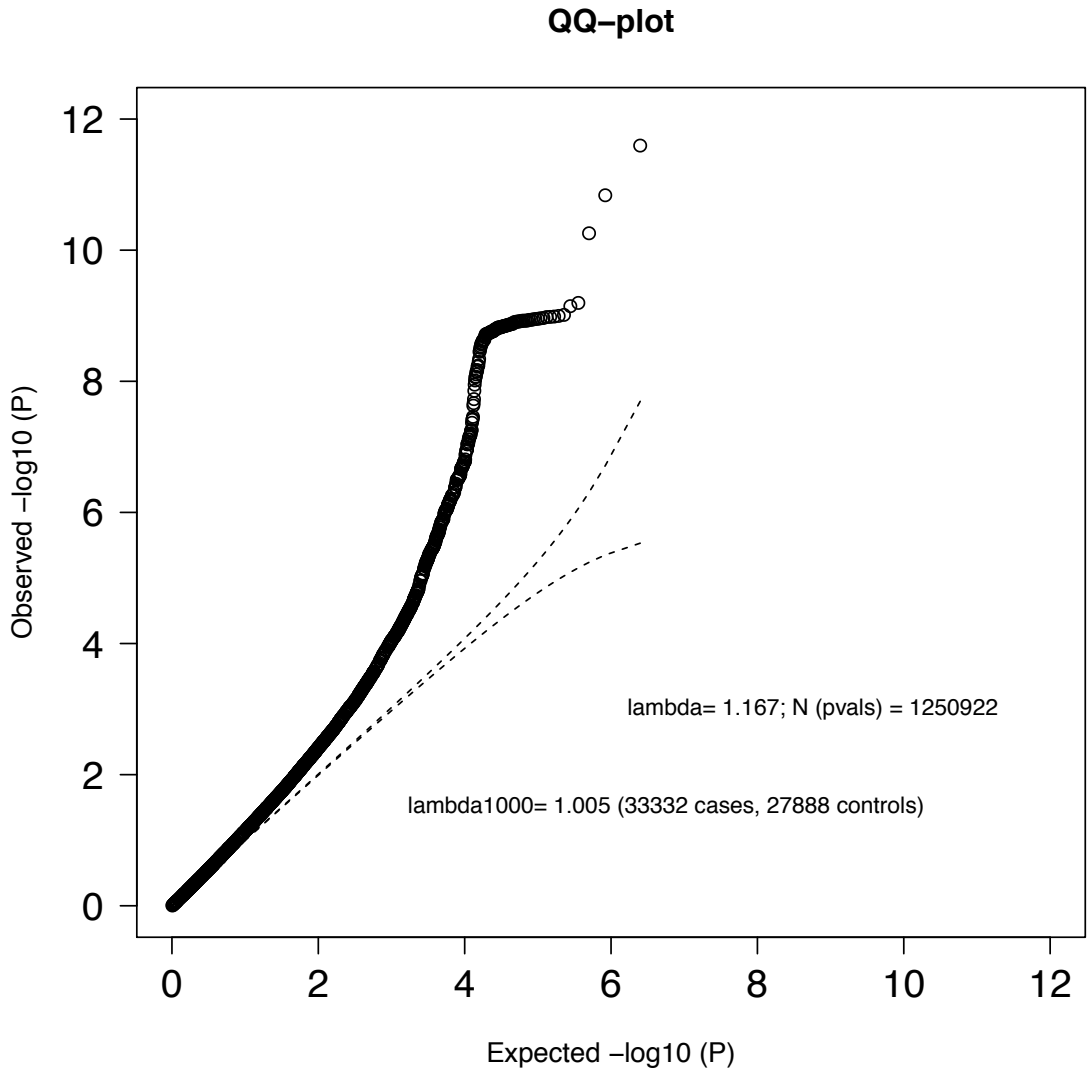
Ding et al.[15] analyzed gene expression data in skin tissue from 53 psoriatic patients and 57 healthy controls. Subjects were genotyped using Perlegen arrarys and profiled for gene expression using Affymetrix U133 arrays. The authors tested SNP-gene eQTL associations separately in normal skin and lesional skin. Our analysis was based on 6,250 cis eQTL markers identified in normal skin downloaded from the authors' webpage (http://www.sph.umich.edu/csg/junding/eQTL/).

Stranger et al.[16] conducted gene expression profiling of lympohoblastoid cell lines derived from 270 HapMap individuals. Illumina's whole genome expression arrays were used for measuring expression values. Linear regression was used for eQTL

association analysis. The significance of the regression *p* values was determined using 10,000 permutations. We downloaded the list of 7,015 cis eQTL markers from the GTEx eQTL browser website (http://www.ncbi.nlm.nih.gov/gtex/GTEX2/gtex.cgi).

Supplemental Results

VIII. Figure S3. Quantile-Quantile plot for the primary 5 disorder meta-analysis

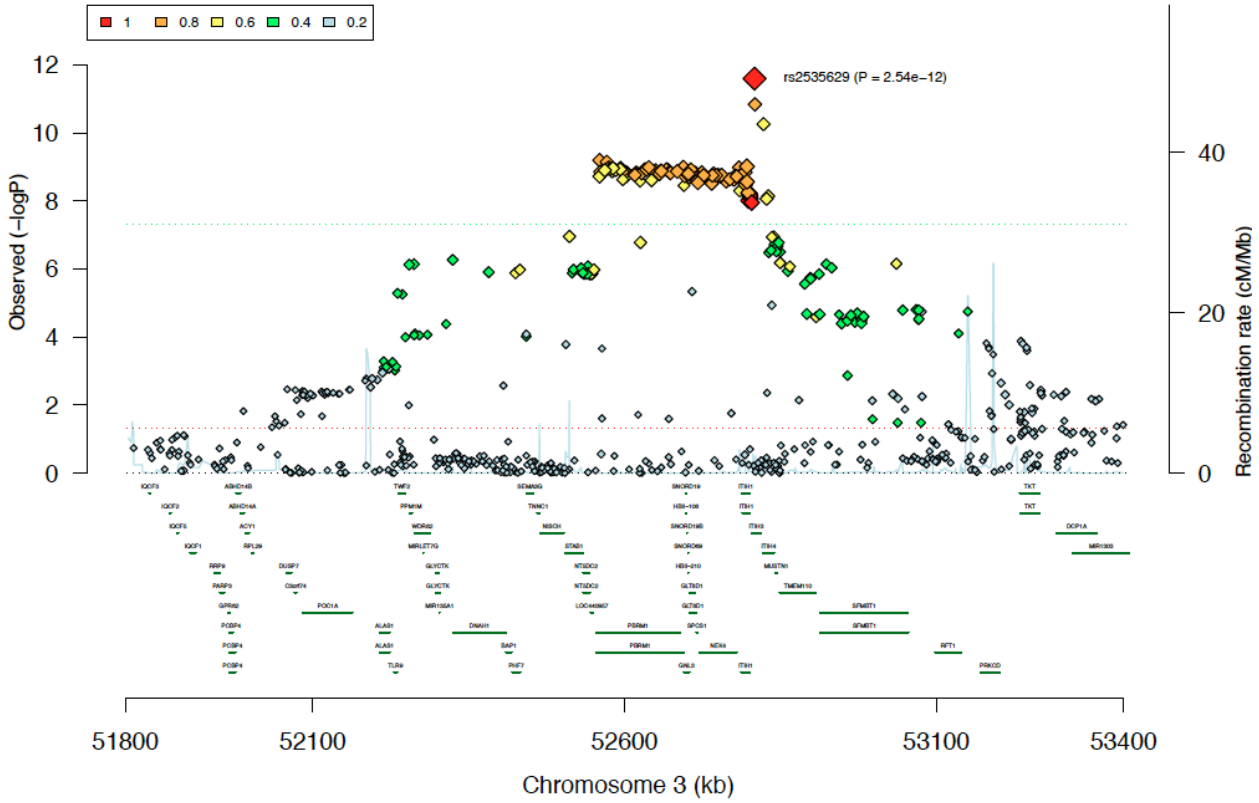


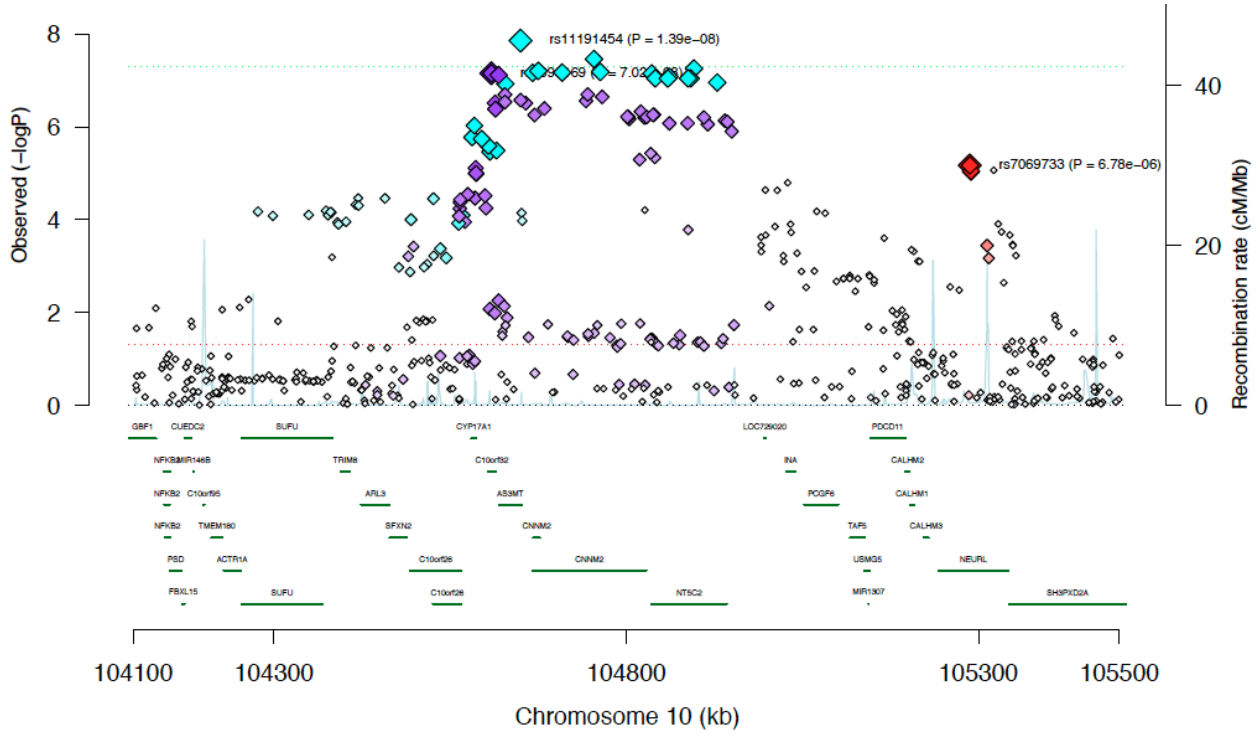
IX. Table S2: Five disorder meta-analysis results for regions with $p < 10^{-5}$

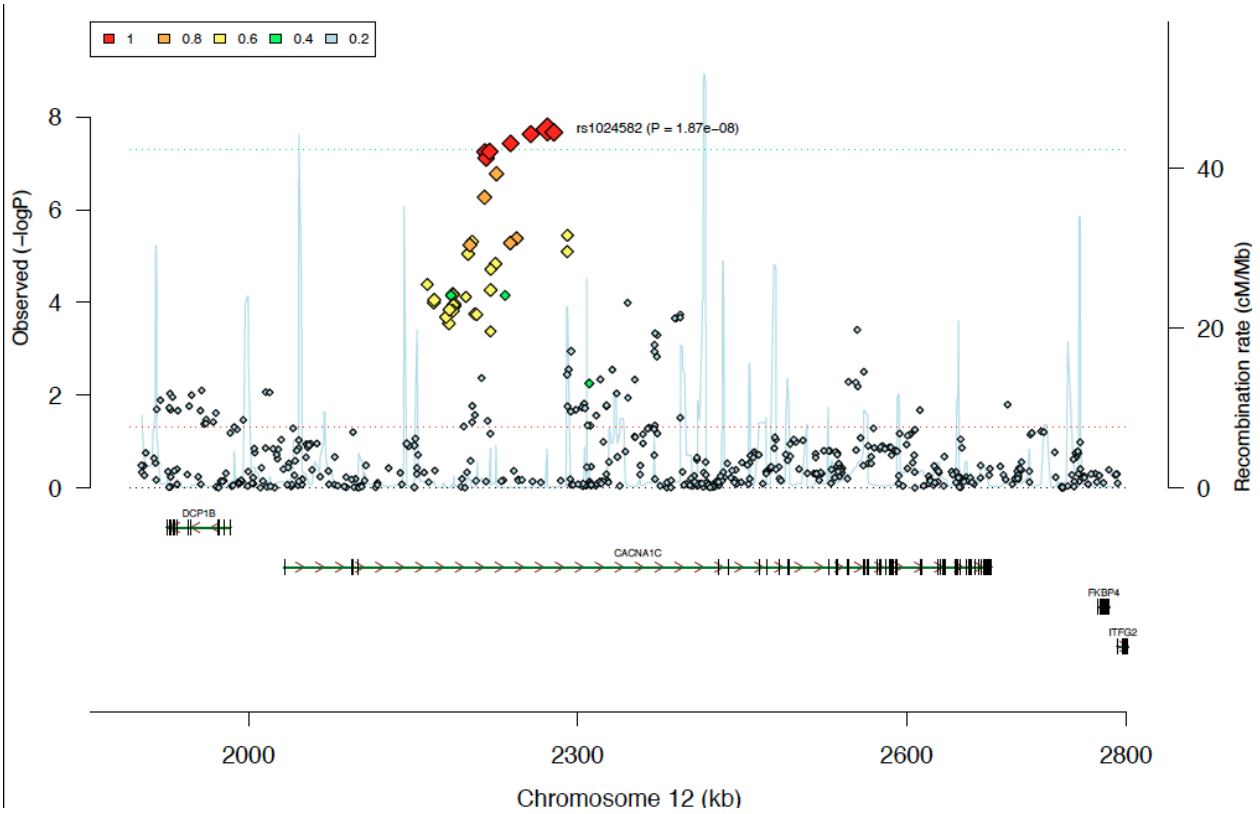
SNP	CHR	BP	Nearest genes	A1A2	INFO	P	OR (95% CI)	Direction	Het P
rs2535629	3	52808259	many	AG	0.942	2.54E-12	1.10 (1.07-1.12)	-----	0.2688
rs11191454	10	104649994	many	AG	1.01	1.39E-08	1.13 (1.08-1.18)	+++++	0.3172
rs1024582	12	2272507	CACNA1C	AG	0.98	1.87E-08	1.07 (1.05-1.10)	+++++	0.005719
rs2799573	10	18641934	CACNB2	TC	0.825	4.29E-08	1.08 (1.05-1.12)	+++++	0.565
rs7096169	10	104608685	many	AG	1	7.02E-08	1.07 (1.04-1.10)	+++++	0.2969
rs2802535	1	98280846	DPYD, MIR137	TC	0.995	1.32E-07	1.08 (1.05-1.12)	+++++	0.07851
rs11152369	18	51217326	TCF4	AC	1	1.73E-07	1.19 (1.12-1.28)	-----	0.55
rs9275524	6	32783087	many	TC	0.993	1.81E-07	1.07 (1.04-1.09)	-----	0.7078
rs9297357	8	106211509	ZFPM2	TC	0.981	4.14E-07	1.07 (1.04-1.10)	+++++	0.7946
rs70018	6	152815726	SYNE1	AG	0.996	4.93E-07	1.06 (1.04-1.09)	+++-+	0.01944
rs2721800	7	24659077	many	CG	0.985	5.54E-07	1.08 (1.05-1.12)	+++++	0.629
rs9951150	18	50972122	-	AG	0.984	5.64E-07	1.06 (1.04-1.09)	-----	0.2062
rs12443954	16	88268997	many	AG	0.898	9.73E-07	1.08 (1.04-1.11)	+++++	0.7906
rs6694545	1	30209855	-	AG	0.923	1.05E-06	1.07 (1.04-1.11)	+++++	0.374
rs12325410	16	9582747	-	TG	0.915	1.16E-06	1.09 (1.05-1.13)	+++++	0.9425
rs13418455	2	180784994	-	TC	0.98	1.17E-06	1.06 (1.04-1.09)	+++++	0.4747
rs12871532	13	107466548	FAM155A	TC	0.973	1.29E-06	1.06 (1.04-1.09)	-----	0.3968
rs6867265	5	130441880	many	AG	0.994	1.52E-06	1.11 (1.06-1.16)	-----	0.4028
rs10860392	12	98022318	ANKS1B	TC	0.995	1.67E-06	1.06 (1.04-1.09)	+++++	0.6042
rs609412	5	146205437	PPP2R2B	AG	0.997	2.00E-06	1.07 (1.04-1.10)	+++++	0.5038
rs249954	16	23547968	many	AG	0.95	2.22E-06	1.07 (1.04-1.10)	-----	0.414
rs2297909	1	199226930	many	AG	0.986	2.27E-06	1.06 (1.04-1.09)	-----	0.9724
rs6602217	10	6986269	-	TC	0.93	2.40E-06	1.12 (1.07-1.17)	-----	0.1408
rs4730430	7	109835200	-	TC	0.988	2.91E-06	1.06 (1.03-1.08)	-----	0.06919

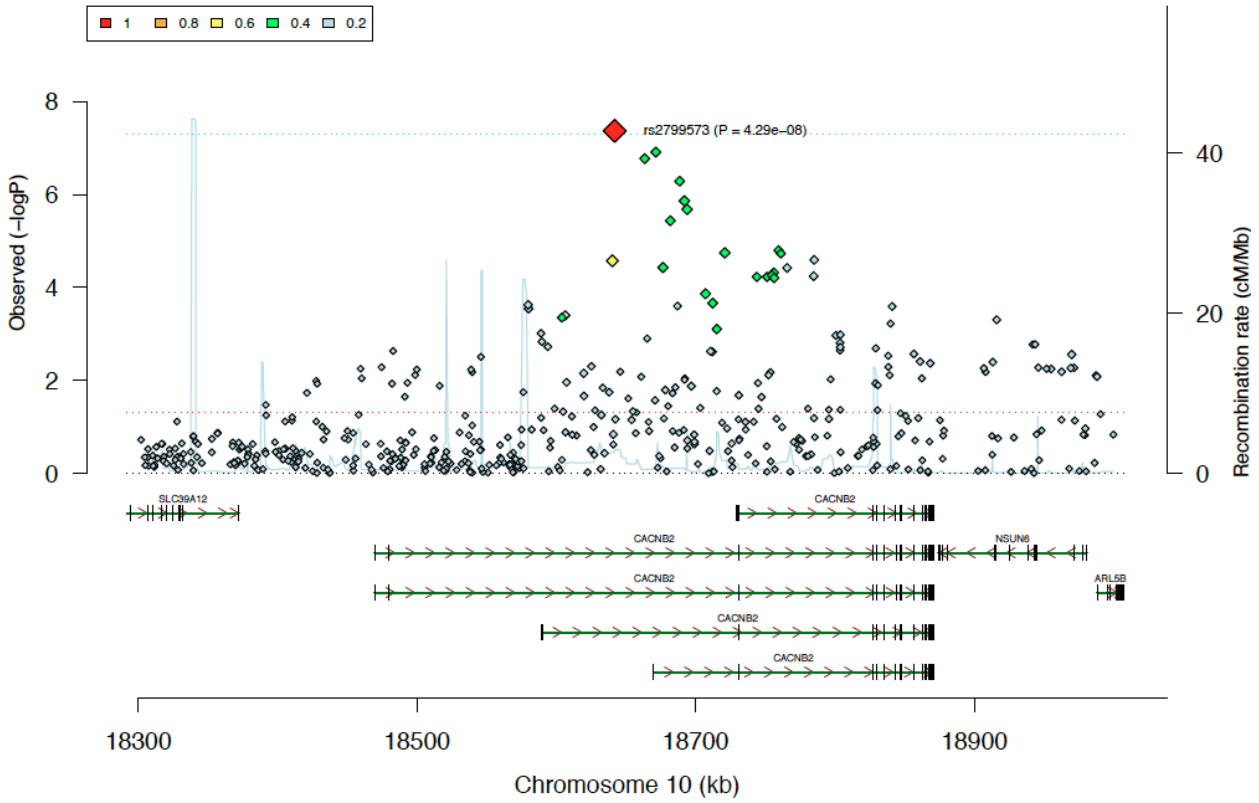
rs2310173	2	102030060	MAP4K4, IL1R2	TG	0.975	2.98E-06	1.06 (1.03-1.08)	+++++	0.7164
rs2609653	8	34356534	-	TC	0.982	3.37E-06	1.13 (1.07-1.19)	----+	0.02925
rs2186903	11	107261761	SLC35F2, RAB39	AG	0.921	3.70E-06	1.07 (1.04-1.11)	-----	0.858
rs7700191	4	102808306	BANK1	AG	0.838	3.72E-06	1.12 (1.06-1.17)	-----	0.2644
rs10873998	1	79036393	ELTD1	TC	0.881	4.04E-06	1.06 (1.03-1.09)	+++++	0.8589
rs10255295	7	137509306	-	AG	0.771	4.51E-06	1.10 (1.06-1.14)	-----	0.204
rs6765687	3	52708146	many	TC	0.769	4.63E-06	1.07 (1.04-1.11)	+++++	0.2759
rs1107592	7	2007958	many	AG	0.95	5.45E-06	1.06 (1.03-1.09)	++-++	0.01765
rs11731175	4	190098245	-	TG	0.991	5.46E-06	1.06 (1.03-1.09)	+++++	0.5333
rs4650608	1	79010603	IFI44L, IFI44	TC	0.998	5.61E-06	1.06 (1.03-1.09)	++++-	0.005063
rs1104918	15	86524716	NTRK3	TG	0.981	5.78E-06	1.07 (1.04-1.10)	-----	0.6807
rs7069733	10	105287760	many	CG	0.835	6.78E-06	1.08 (1.04-1.12)	+++++	0.3501
rs4741652	9	2184227	SMARCA2	TC	0.857	6.91E-06	1.07 (1.04-1.10)	+++++	0.2787
rs8058295	16	9818887	GRIN2A	AC	0.985	7.12E-06	1.06 (1.04-1.09)	+++++	0.7285
rs7254215	19	14574493	many	AG	0.834	7.49E-06	1.09 (1.05-1.13)	-----	0.6357
rs3791556	2	239777909	HDAC4, MGC16025	AG	0.954	7.65E-06	1.08 (1.04-1.11)	-----	0.2316
rs360932	4	153127619	-	AG	0.991	8.36E-06	1.06 (1.03-1.08)	+++++	0.07611
rs13072940	3	36817627	many	AT	0.955	8.60E-06	1.06 (1.03-1.08)	---++	0.007166
rs11191732	10	105321751	many	AG	0.979	8.65E-06	1.07 (1.04-1.10)	+++++	0.2633
rs1533087	20	542780	many	TC	0.834	8.81E-06	1.06 (1.03-1.09)	-----	0.8698
rs11587682	1	148590908	many	TC	0.985	9.48E-06	1.09 (1.05-1.13)	----+	0.01793
rs2941579	2	54715329	many	TC	0.993	9.89E-06	1.08 (1.04-1.11)	+++++	0.7605

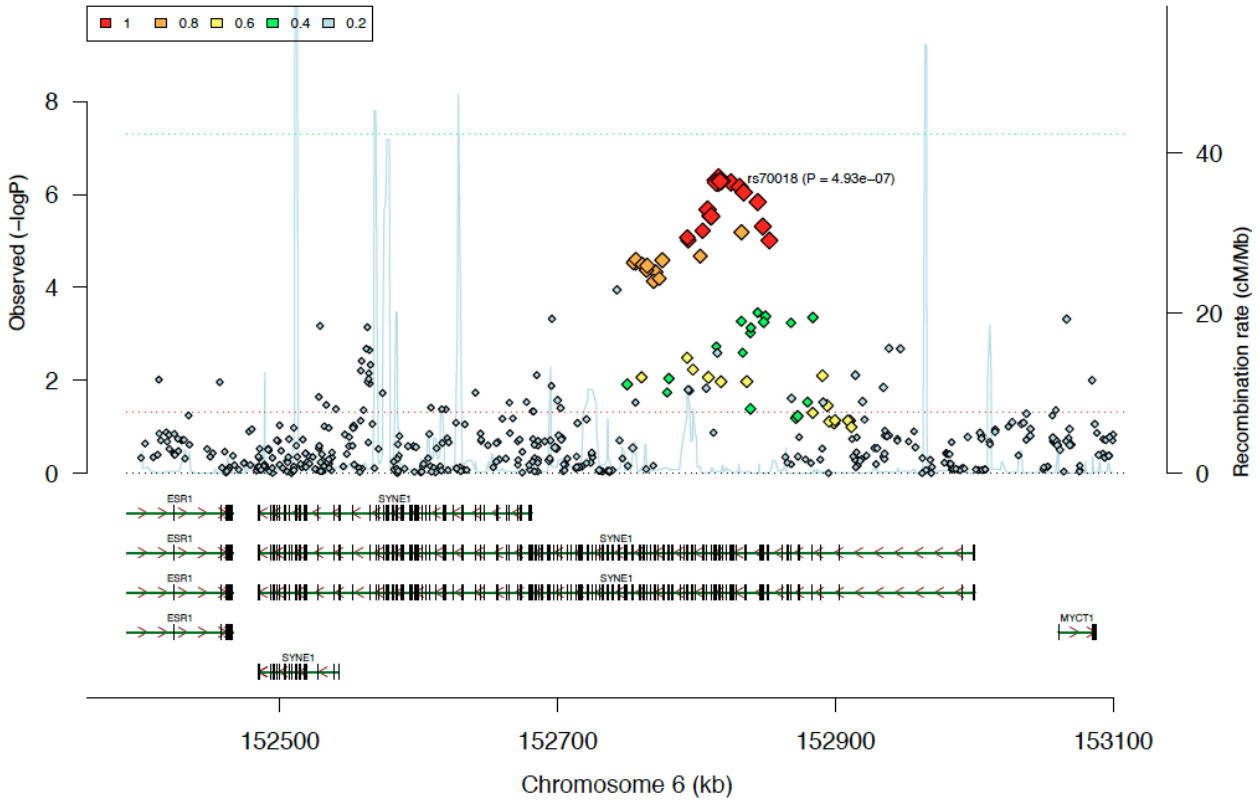
X. Figure S4: Regional Plots for Top Results ($p < 10^{-6}$) from Primary Meta-Analysis

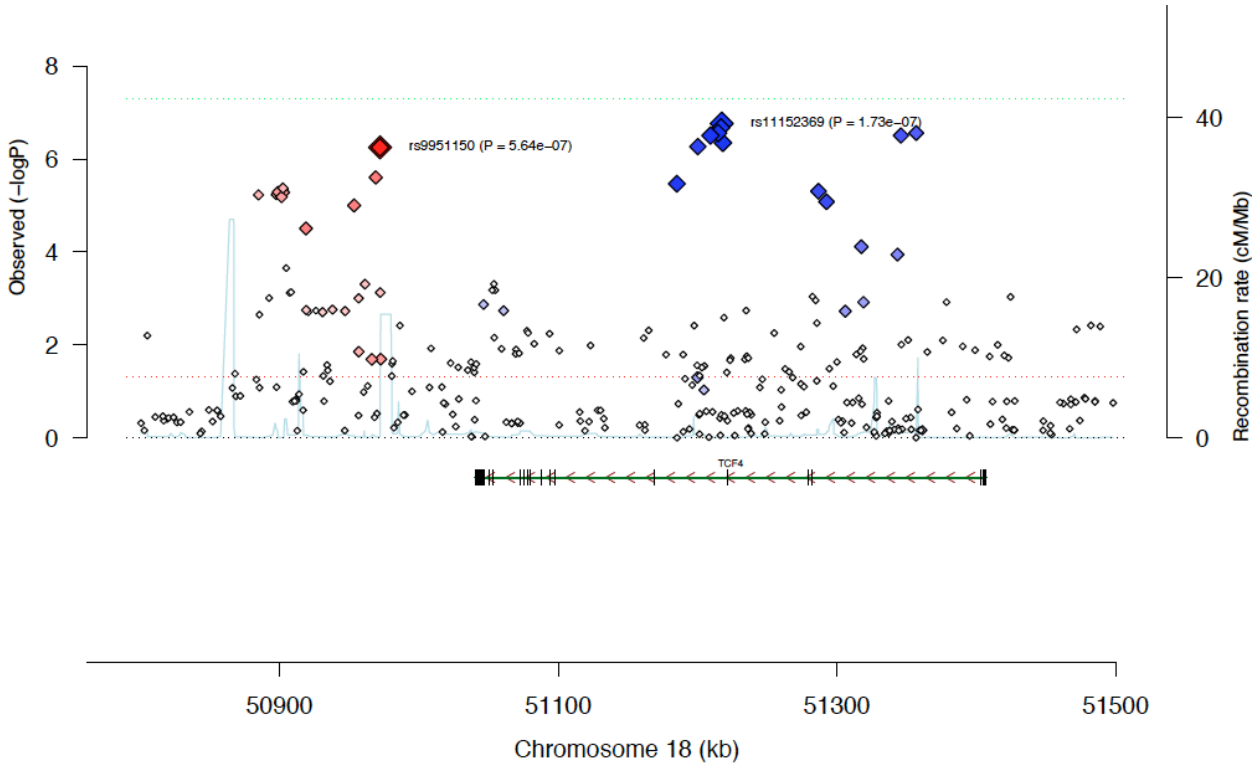


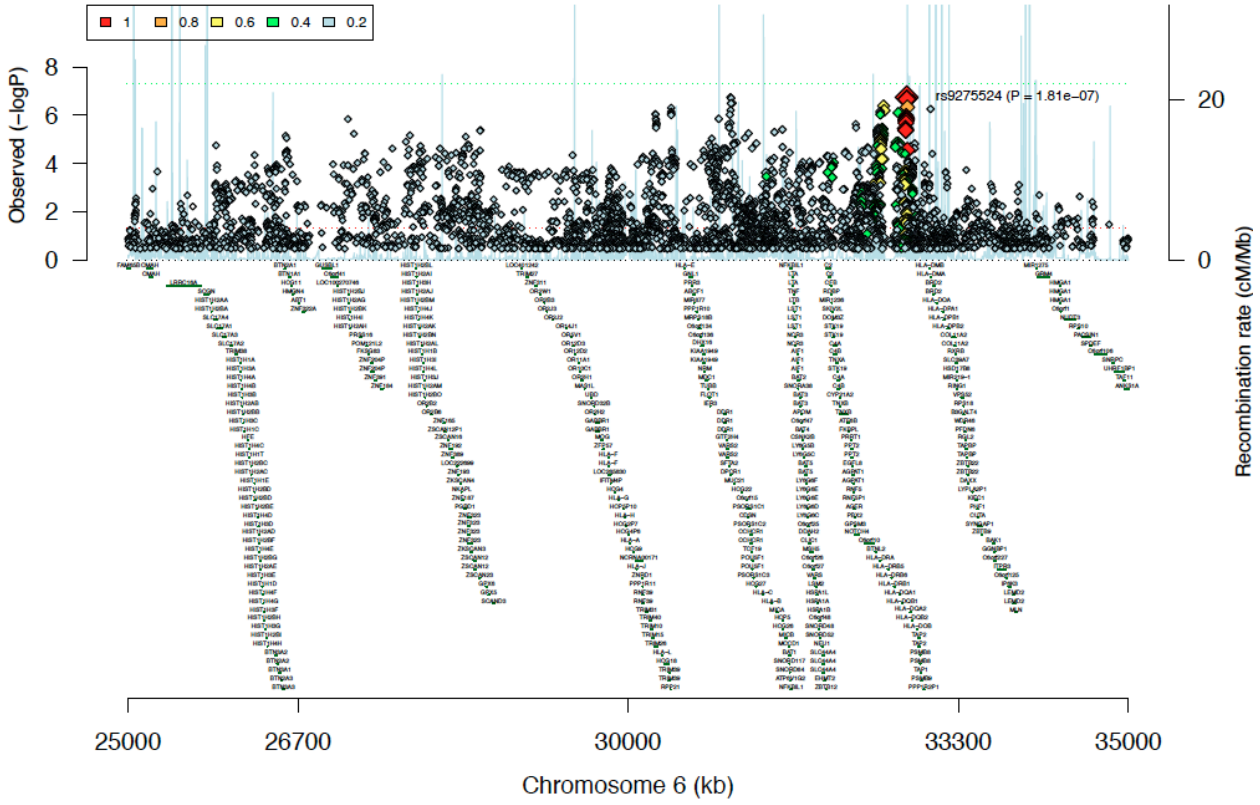

rs11191454 (P = 1.39e−08)
rs7069733 (P = 6.78e−06)
Observed (−logP)
Recombination rate (cM/Mb)
0
2
4
6
8
0
20
40
104100
104300
104800
105300
105500
Chromosome 10 (kb)

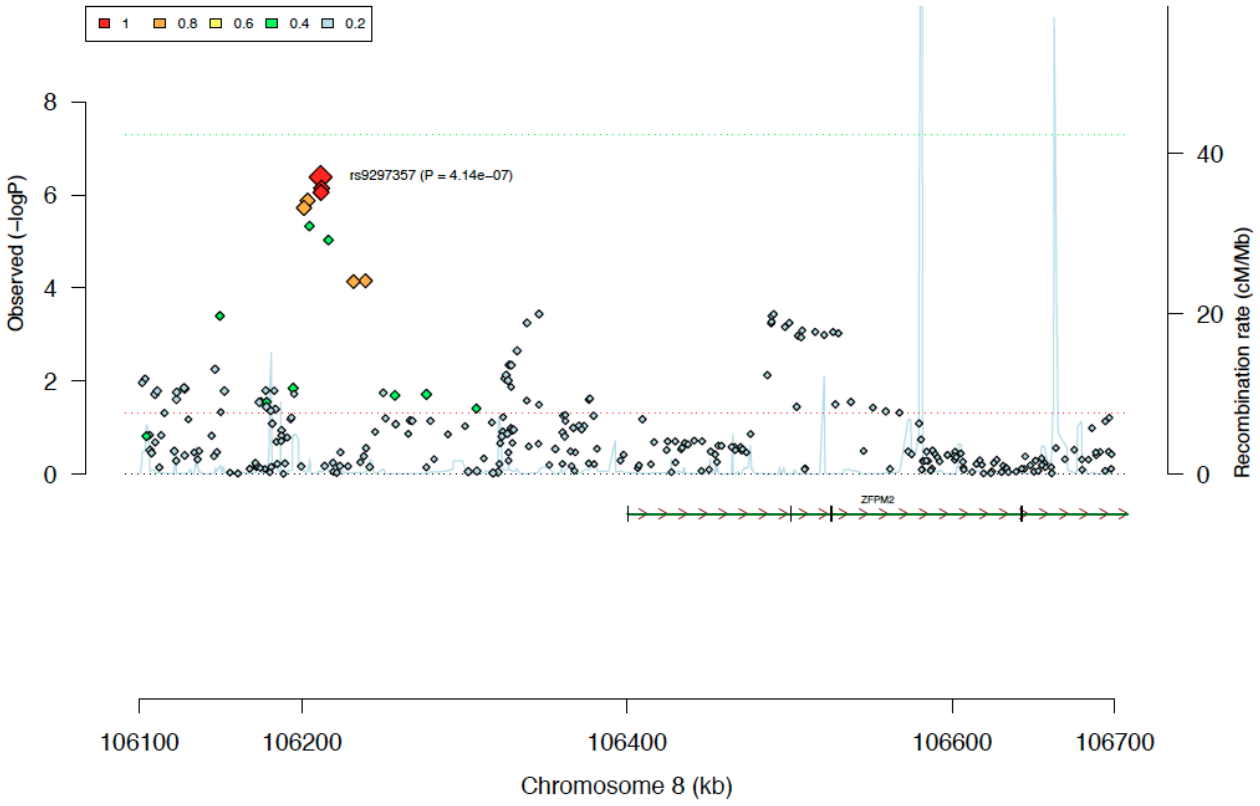

1
0.8
0.6
0.4
0.2
rs1024582 (P = 1.87e−08)
Observed (−logP)
Recombination rate (cM/Mb)
0
2
4
6
8
0
20
40
DCP1B
CACNA1C
FKBP4
ITFG2
2000
2300
2600
2800
Chromosome 12 (kb)

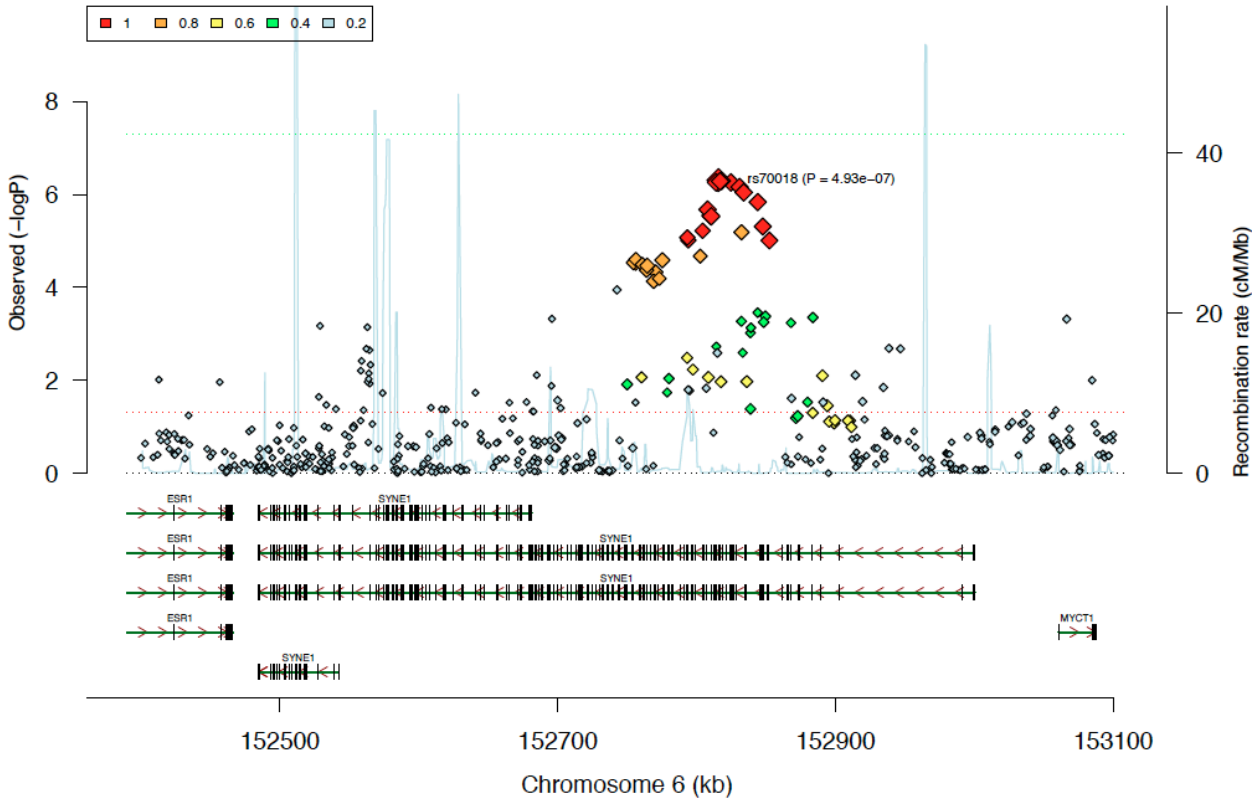

1
0.8
0.6
0.4
0.2
rs2799573 (P = 4.29e−08)
Observed (−logP)
Recombination rate (cM/Mb)
SLC39A12
CACNB2
NSUN6
ARL5B
18300
18500
18700
18900
Chromosome 10 (kb)

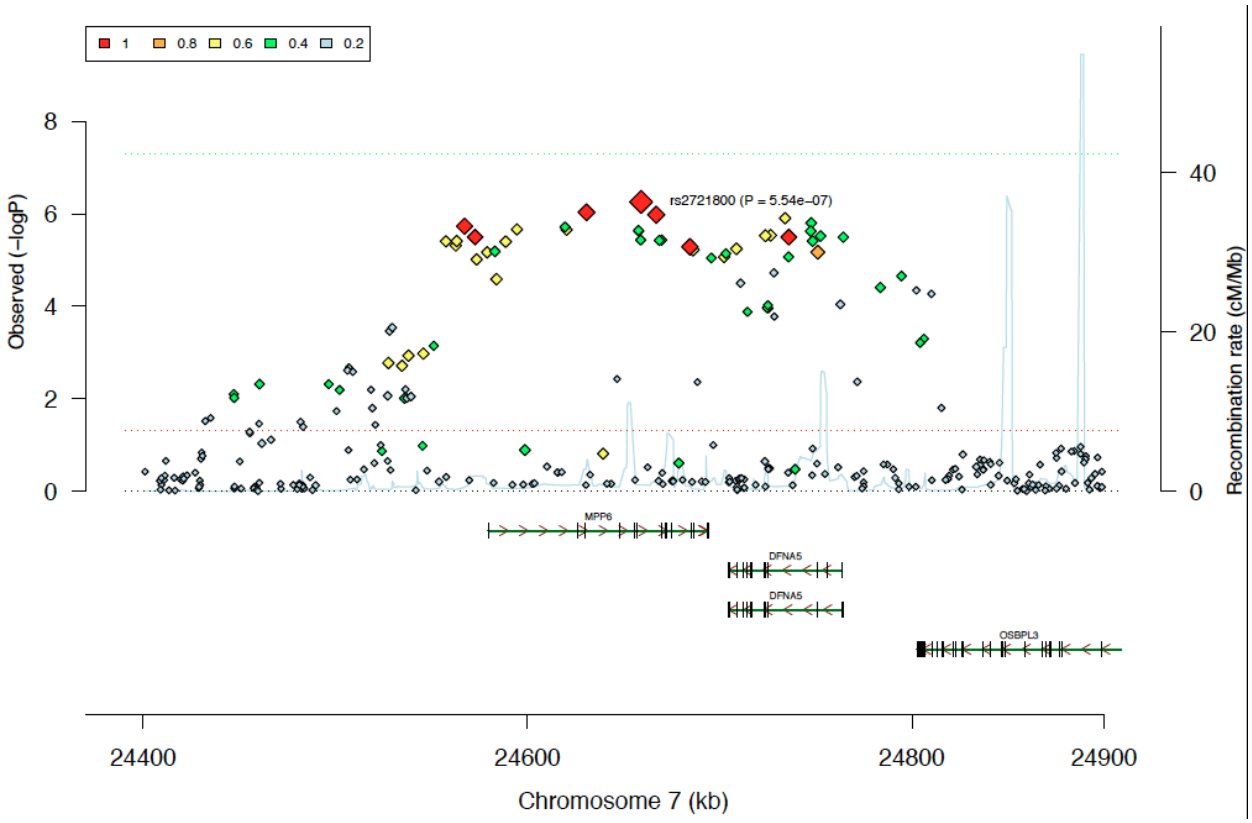

1
0.8
0.6
0.4
0.2
Observed (−logP)
Recombination rate (cM/Mb)
rs70018 (P = 4.93e−07)
ESR1
SYNE1
MYCT1
152500
152700
152900
153100
Chromosome 6 (kb)

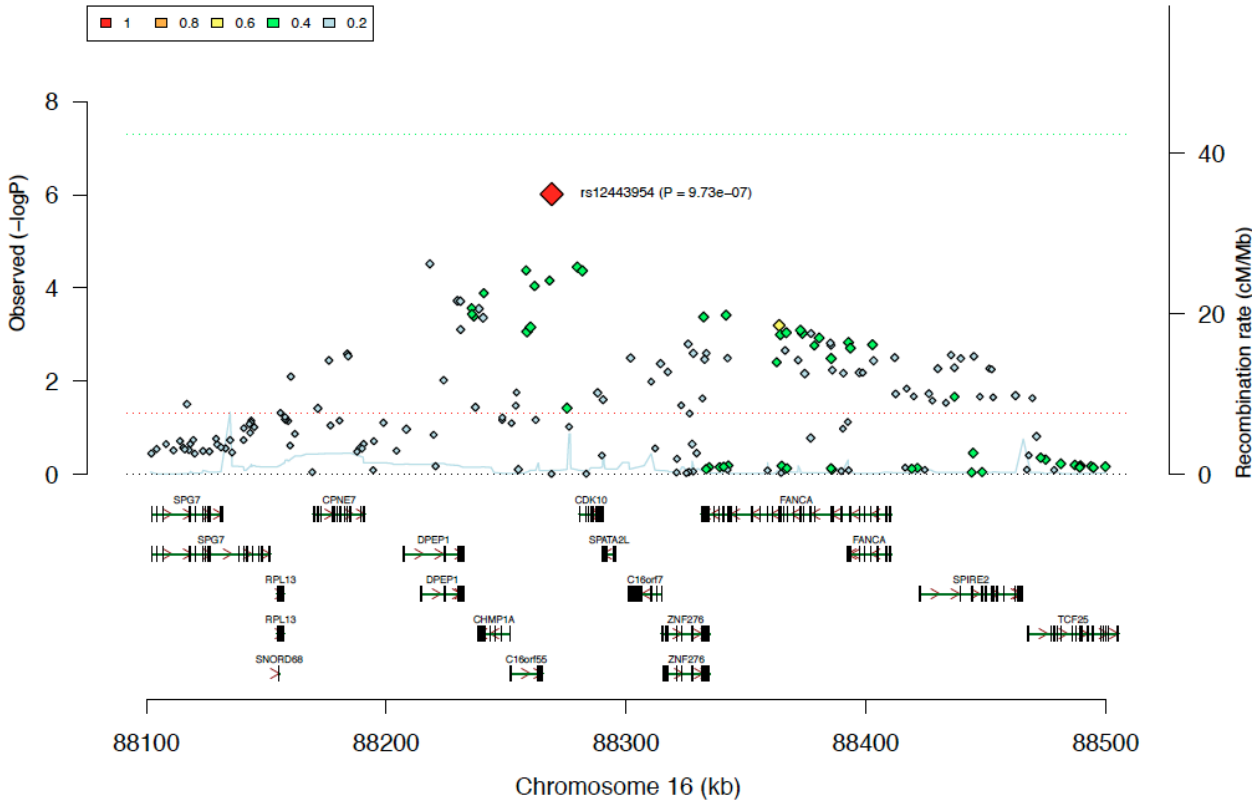

Observed (−logP)
Recombination rate (cM/Mb)
rs9951150 (P = 5.64e−07)
rs11152369 (P = 1.73e−07)
TCF4
50900
51100
51300
51500
Chromosome 18 (kb)


1
0.8
0.6
0.4
0.2
Observed (−logP)
8
6
4
2
0
rs9275524 (P = 1.81e−07)
20
0
Recombination rate (cM/Mb)
25000
26700
30000
33300
35000
Chromosome 6 (kb)


1
0.8
0.6
0.4
0.2
rs9297357 (P = 4.14e−07)
Observed (−logP)
Recombination rate (cM/Mb)
ZFPM2
106100
106200
106400
106600
106700
Chromosome 8 (kb)


1
0.8
0.6
0.4
0.2
Observed (−logP)
Recombination rate (cM/Mb)
rs70018 (P = 4.93e−07)
ESR1
SYNE1
MYCT1
152500
152700
152900
153100
Chromosome 6 (kb)


1
0.8
0.6
0.4
0.2
rs2721800 (P = 5.54e−07)
Observed (−logP)
Recombination rate (cM/Mb)
MPP6
DFNA5
DFNA5
OSBPL3
24400
24600
24800
24900
Chromosome 7 (kb)


1
0.8
0.6
0.4
0.2
Observed (−logP)
Recombination rate (cM/Mb)
rs12443954 (P = 9.73e−07)
SPG7
CPNE7
CDK10
FANCA
DPEP1
SPATA2L
RPL13
C18orf7
SPIRE2
CHMP1A
ZNF276
TCF25
SNORD68
C16orf55
88100
88200
88300
88400
88500
Chromosome 16 (kb)

XI. Table S3: Modeling analysis results for SNPs in regions with a meta analysis $p < 1 \times 10^{-6}$

SNP	CHR	BP	Nearest Gene	Alleles	Freq	P_{meta}	BIC Model	Odds Ratio Estimated from BIC Model				
								ADHD	ASD	BP	MDD	SCZ
rs2535629	3	52808259	*many*	G/A	0.651	2.54×10^{-12}	5 Disorder	1.095	1.095	1.095	1.095	1.095
							Omnibus	1.077	1.107	1.120	1.073	1.099
rs11191454	10	104649994	*many*	A/G	0.910	1.39×10^{-8}	5 Disorder	1.122	1.122	1.122	1.122	1.122
							Omnibus	1.124	1.103	1.137	1.085	1.159
rs1024582	12	2272507	*CACNA1C*	A/G	0.337	1.87×10^{-8}	BP, SCZ	1.000	1.000	1.088	1.000	1.088
							Omnibus	1.046	1.008	1.110	1.028	1.093
rs2799573	10	18641934	*CACNB2*	T/C	0.715	4.29×10^{-8}	5 Disorder	1.091	1.091	1.091	1.091	1.091
							Omnibus	1.170	1.119	1.070	1.090	1.073
rs7096169	10	104608685	*many*	A/G	0.647	7.02×10^{-8}	5 Disorder	1.055	1.055	1.055	1.055	1.055
							Omnibus	1.034	1.038	1.053	1.056	1.072
rs2802535	1	98280846	*DPYD, MIR137*	T/C	0.802	1.32×10^{-7}	ASD, SCZ	1.000	1.127	1.000	1.000	1.127
							Omnibus	1.121	1.164	1.080	1.039	1.161
rs11152369	18	51217326	*TCF4*	C/A	0.030	1.73×10^{-7}	ASD, SCZ	1.000	1.278	1.000	1.000	1.278
							Omnibus	1.120	1.422	1.149	1.096	1.315
rs9275524	6	32783087	*many*	C/T	0.575	1.81×10^{-7}	5 Disorder	1.074	1.074	1.074	1.074	1.074
							Omnibus	1.064	1.125	1.098	1.039	1.069
rs3132581	6	31021437	*many*	G/A	0.860	1.83×10^{-7}	5 Disorder	1.117	1.117	1.117	1.117	1.117
							Omnibus	1.079	1.134	1.074	1.093	1.183
rs9276601	6	32842282	*many*	A/G	0.827	2.06×10^{-7}	5 Disorder	1.101	1.101	1.101	1.101	1.101
							Omnibus	1.076	1.085	1.101	1.086	1.131
rs9297357	8	106211509	*ZFPM2*	T/C	0.715	4.14×10^{-7}	5 Disorder	1.069	1.069	1.069	1.069	1.069
							Omnibus	1.020	1.111	1.068	1.050	1.084
rs70018	6	152815726	*SYNE1*	A/G	0.409	4.93×10^{-7}	BP	1.000	1.000	1.111	1.000	1.000
							Omnibus	1.035	1.109	1.144	1.032	1.056
rs1480380	6	33021224	*many*	C/T	0.908	5.53×10^{-7}	Adult	1.000	1.000	1.153	1.153	1.153
							Omnibus	1.071	1.033	1.132	1.137	1.224

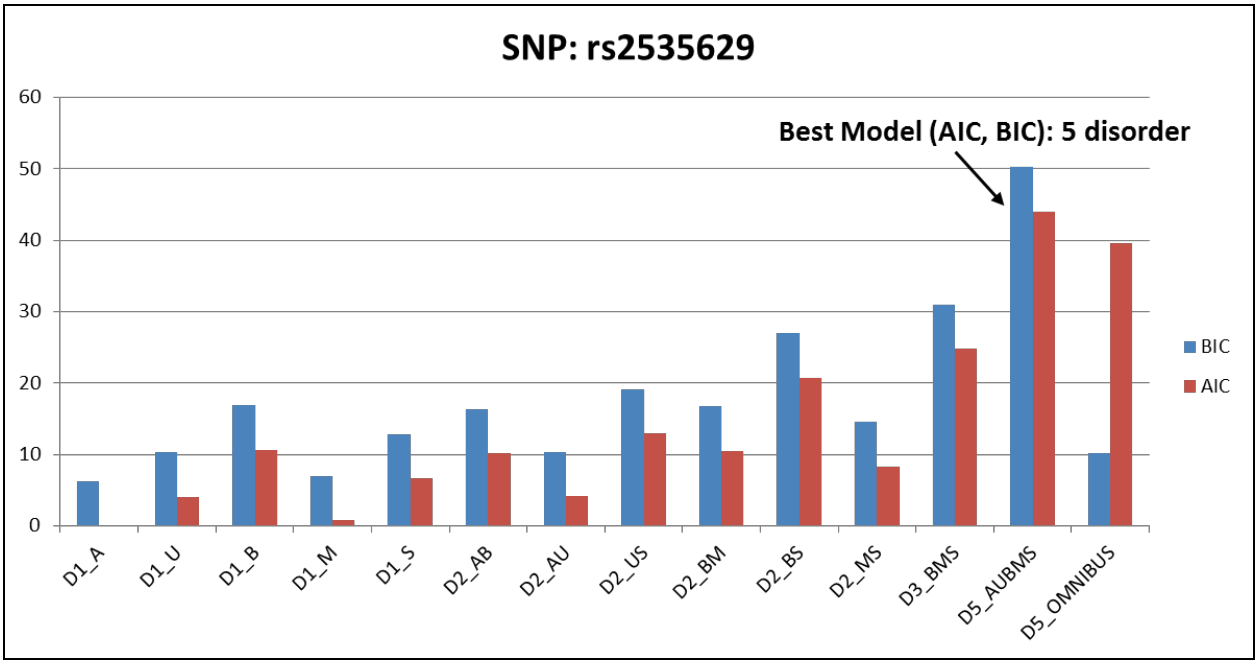
rs2721800	7	24659077	*many*	C/G	0.170	5.54×10^{-7}	BP, SCZ	1.000	1.000	1.086	1.000	1.086
							Omnibus	1.079	1.092	1.136	0.994	1.078
rs9951150	18	50972122	-	G/A	0.448	5.64×10^{-7}	5 Disorder	1.078	1.078	1.078	1.078	1.078
							Omnibus	1.082	1.055	1.090	1.088	1.071
rs12443954	16	88268997	*many*	A/G	0.751	9.73×10^{-7}	5 Disorder	1.078	1.078	1.078	1.078	1.078
							Omnibus	1.074	1.075	1.100	1.043	1.099

XII. Table S4: Association analysis results for top SNPs from the 5 degree of freedom test with p < 1x10-5. This analysis allowed for equal effects from each disease (independent of sample size) and also allowed the detection of opposite allelic effects in different diseases.

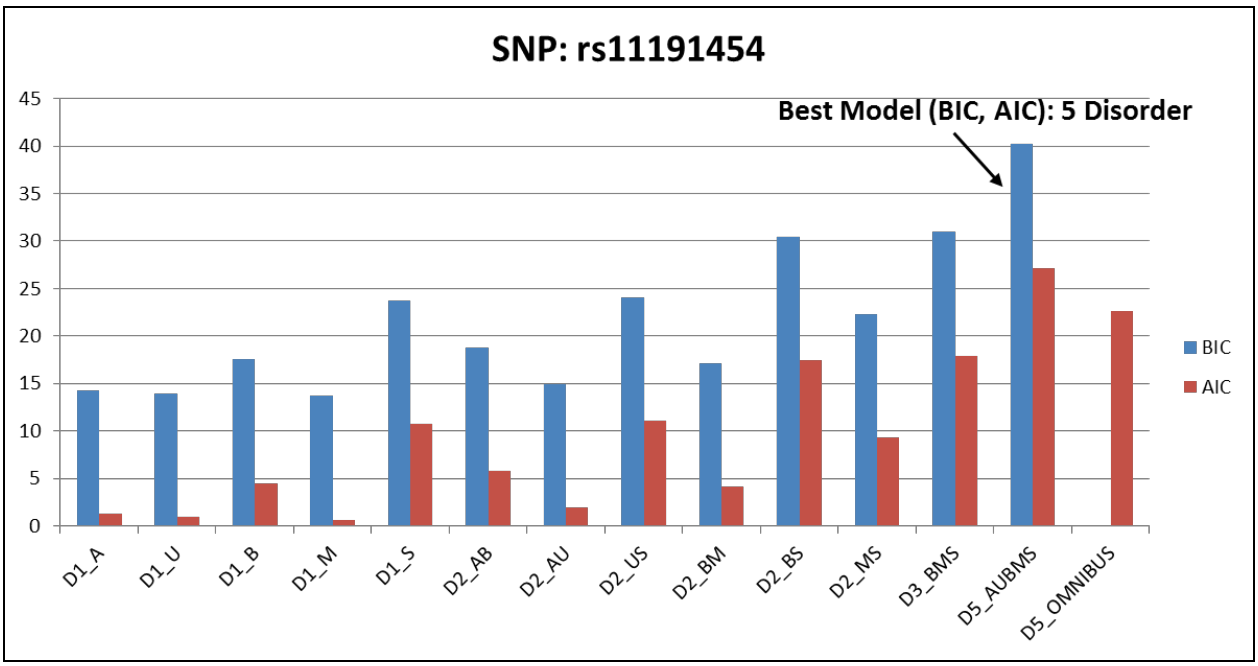
SNP	CHR	BP	P	OR	SE	A1A2	FRQ	INFO	Direction	Gene
rs2535629	3	52808259	1.97E-10	1.095	0.013	GA	0.651	0.942	+++++	many
rs10994359	10	61892113	2.81E-09	1.081	0.025	CT	0.064	0.989	+-+-+	ANK3
rs1006737	12	2215556	4.97E-09	1.071	0.013	AG	0.013	1.01	+-+++	CACNA1C
rs8321	6	30140501	7.96E-09	1.081	0.02	AC	0.02	1.01	--+++	MHC region
rs11191580	10	104896201	4.11E-07	1.123	0.021	TC	0.021	1.01	+++++	many
rs9371601	6	152832266	4.66E-07	1.058	0.013	TG	0.013	0.994	-++++	SYNE1
rs2252865	1	8345263	5.09E-07	1.03	0.013	TC	0.013	0.998	-++-+	SLC45A1,RERE,ENO1
rs1198588	1	98325420	5.69E-07	1.079	0.015	TA	0.788	0.985	-++++	DPYD,MIR137
rs1715	19	57186590	5.70E-07	1.41	0.063	TC	0.063	0.301	++-++	many
rs11191438	10	104627854	1.01E-06	1.065	0.012	GC	0.586	1	+++++	many
rs7972947	12	2040694	1.04E-06	1.032	0.016	CA	0.784	0.803	-+-++	DCP1B,CACNA1C
rs4650608	1	79010603	1.22E-06	1.06	0.013	TC	0.013	0.998	+-+++	IFI44L,IFI44
rs6990255	8	34246490	2.06E-06	1.146	0.03	TC	0.03	0.984	+-+++	-
rs9834970	3	36831034	2.20E-06	1.051	0.012	CT	0.491	0.972	--+++	many
rs7004633	8	89829427	2.73E-06	1.049	0.016	GA	0.184	0.948	++-++	MMP16
rs447	7	83610351	3.63E-06	1.025	0.015	CT	0.788	1	+-+++	SEMA3A
rs2799573	10	18641934	4.10E-06	1.084	0.015	TC	0.015	0.825	+++++	CACNB2
rs7799006	7	2244752	4.51E-06	1.059	0.013	CT	0.635	0.924	+++-+	many
rs363598	21	29935030	4.55E-06	1.006	0.014	CT	0.256	0.99	--+-+	GRIK1
rs2675968	2	233444488	4.77E-06	1.016	0.013	TC	0.013	0.992	--+-+	many
rs703970	10	80623142	5.12E-06	1.01	0.012	CA	0.579	0.956	-++-+	ZMIZ1
rs7948661	11	117846859	5.19E-06	1.05	0.026	CT	0.058	0.942	+--++	many
rs11827962	11	22269142	6.19E-06	1.044	0.053	CT	0.984	0.847	---++	ANO5,SLC17A6
rs159788	20	4300104	6.56E-06	1.012	0.019	GA	0.88	0.965	+-++-	-

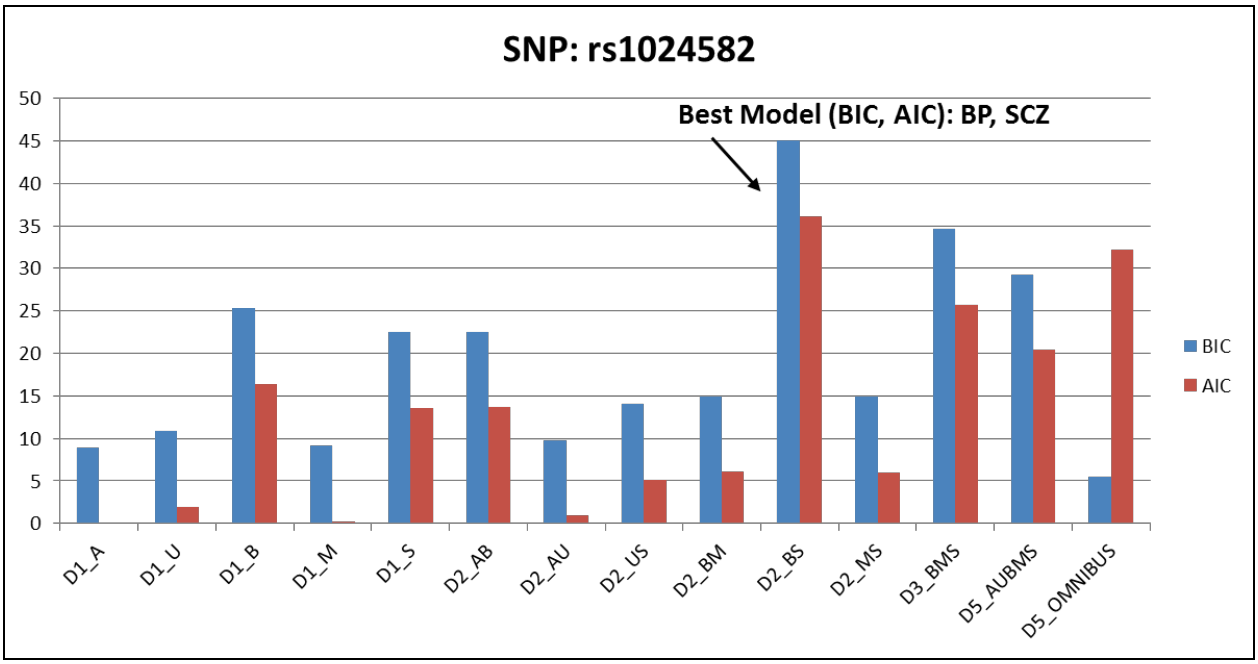
rs7565792	2	48503209	6.65E-06	1.03	0.013	CT	0.631	0.909	-++++	many
rs12966547	18	50903015	7.00E-06	1.057	0.012	GA	0.588	0.995	+-+++	-
rs11587682	1	148590908	7.26E-06	1.086	0.019	CT	0.877	0.985	+-+++	many
rs7597593	2	185241825	7.52E-06	1.055	0.012	TC	0.012	0.979	+-+++	ZNF804A
rs6435387	2	149573496	7.56E-06	1.129	0.033	GA	0.962	0.941	+-+-+	KIF5C,LYPD6B
rs17597926	18	51356936	7.68E-06	1.202	0.036	AG	0.036	0.957	+++++	TCF4
rs7849973	9	22809576	7.74E-06	1.05	0.013	GC	0.342	0.966	+-+++	-
rs10250997	7	135797076	7.96E-06	1.03	0.02	CA	0.89	0.945	+-+-+	-
rs961196	14	90204414	9.67E-06	1.033	0.017	CT	0.812	0.815	-++-+	TTC7B

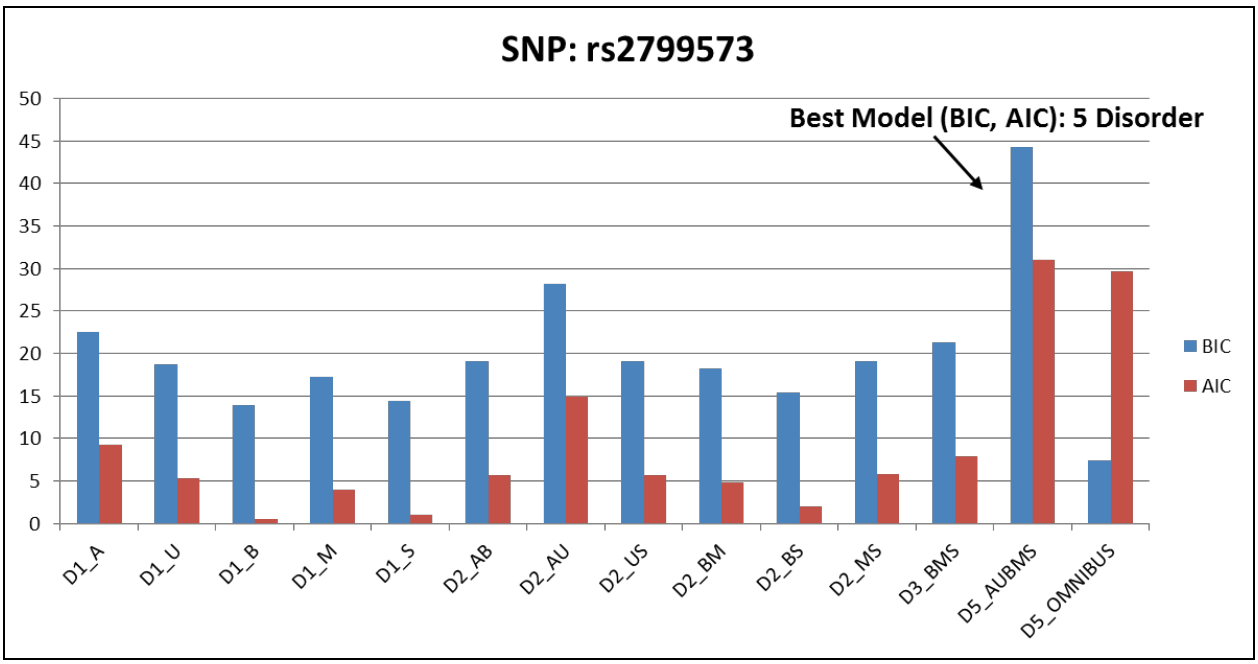
XIII. Figure S5: Comparisons of Model Fit by BIC/AIC criteria for 16 SNPs in regions with a meta analysis $p < 1 \times 10^{-6}$. The blue bar and the red bar represent score differences across 21 models with respect to Bayesian information criterion (BIC) and Akaike information criterion (AIC) measures, respectively. The X-axis represents the 21 disease causal models, while the Y-axis represents the relative difference in the information criterion score of each model compared to that of the highest score model (i.e., worst-fit model). The best-fit model is marked by a circle in each plot. Note that the best-fit model could not be determined when the score difference between the best and the second-best model is ≤ 2 [17]

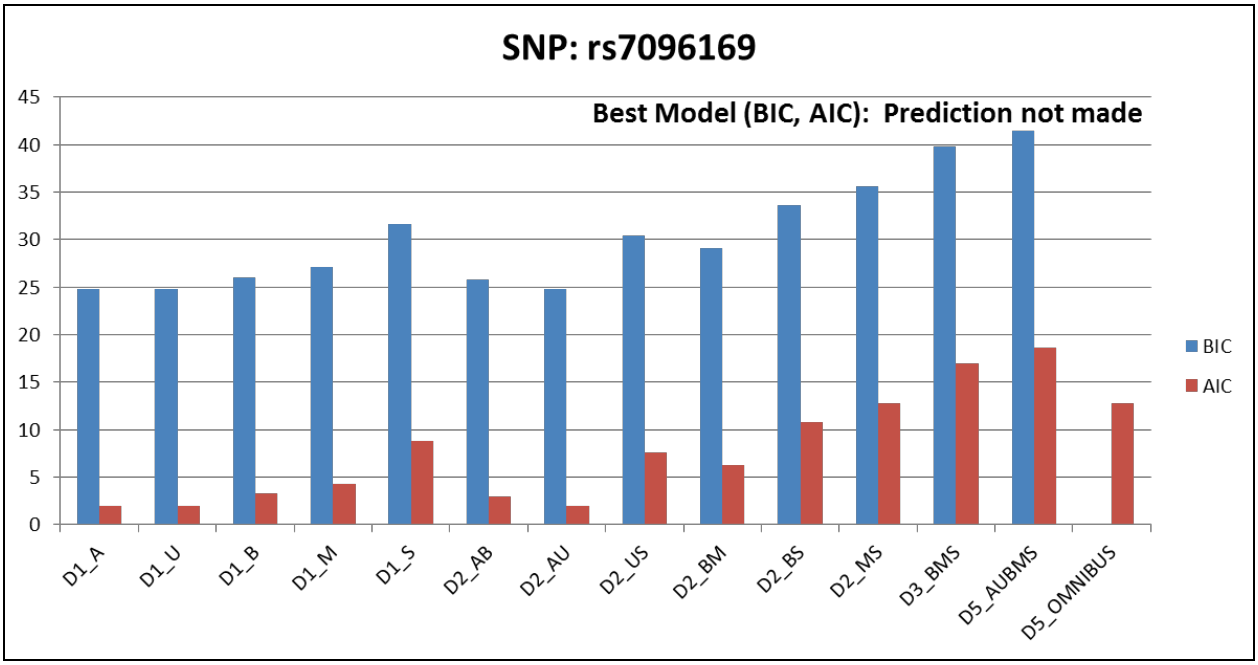


Legend: D1_A : ADHD specific risk model; D1_U: Autism specific risk model; D1_B: Bipolar disorder (BP) specific model; D1_M: major depressive disorder (MDD) specific risk model; D1_S: schizophrenia (SCZ) specific risk model; D2_AB: ADHD & BP risk model; D2_AU: ADHD & Autism risk model; D2_US: Autism & SCZ model; D2_BM: BP & MDD risk model; D2_BS: BP & SCZ risk model; D2_MS: MDD & SCZ risk model; D3_BMS: BP & MDD & SCZ risk model; D5_AUBMS: ADHD, Autism, BP, MDD & SCZ risk model; D5_OMNIBUS: General disorder risk model

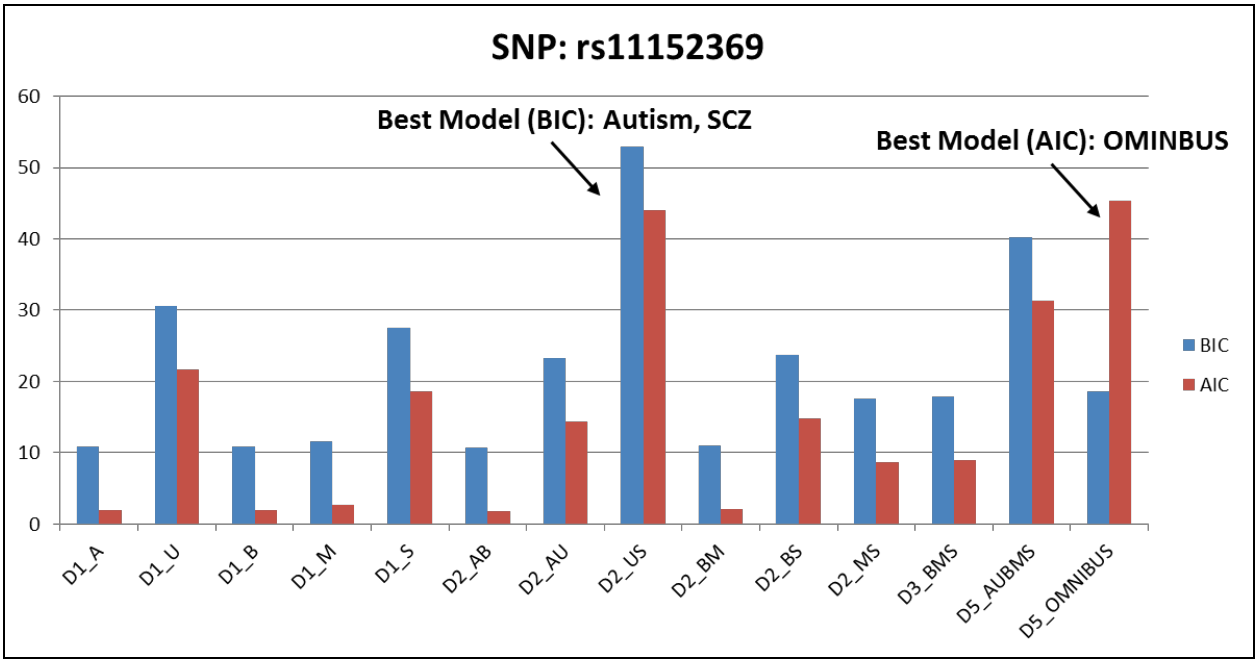

SNP: rs11191454
Best Model (BIC, AIC): 5 Disorder
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

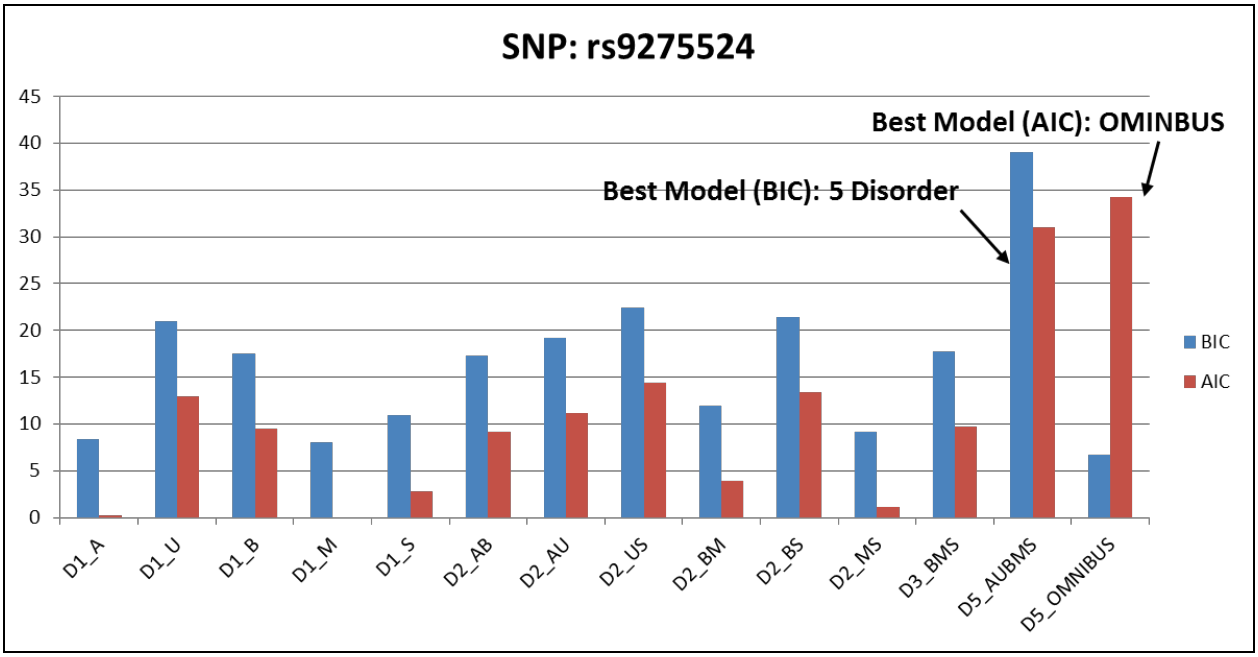

SNP: rs1024582
Best Model (BIC, AIC): BP, SCZ
50
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

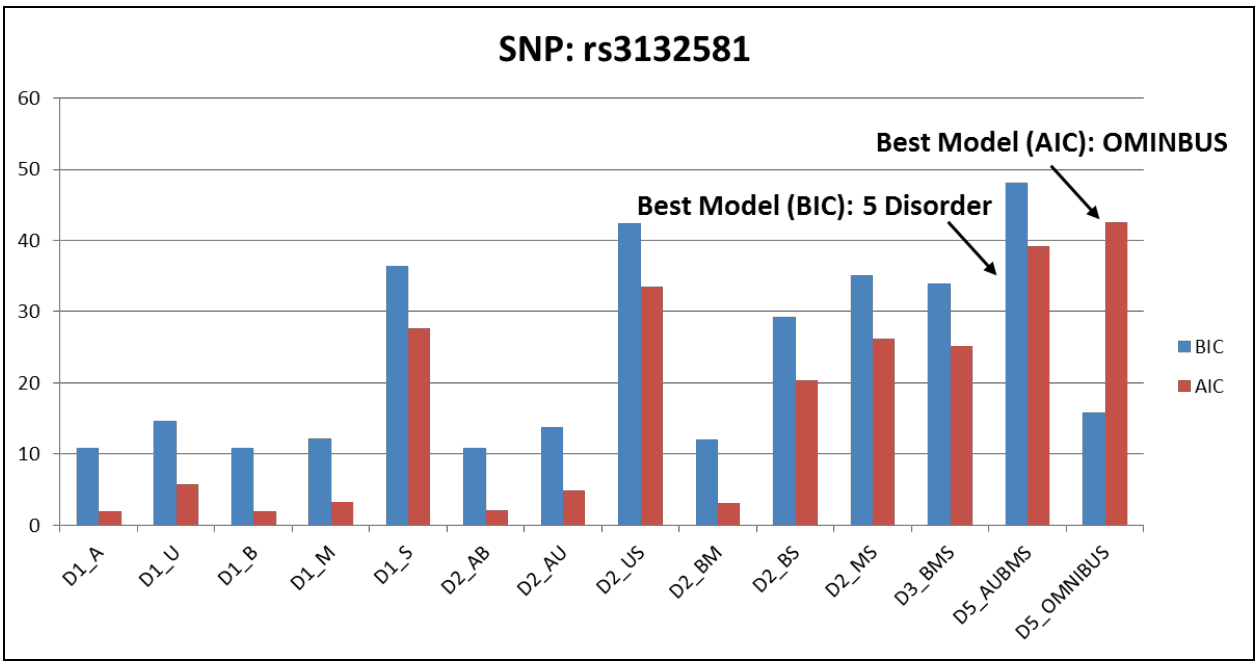


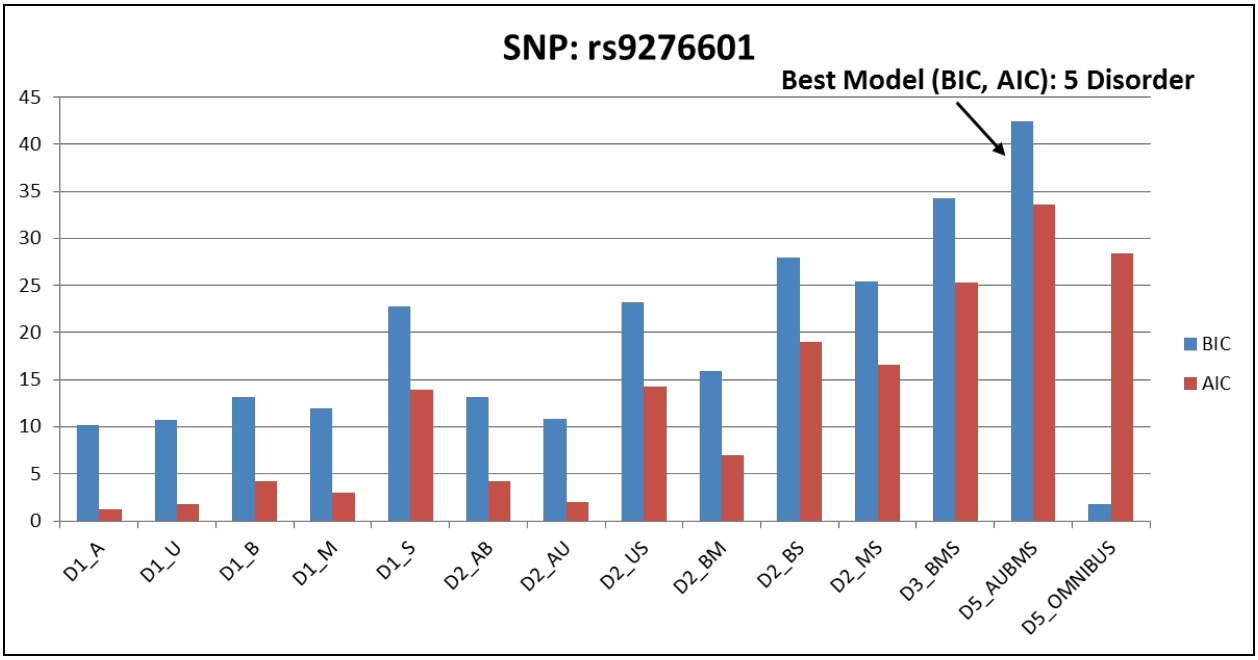


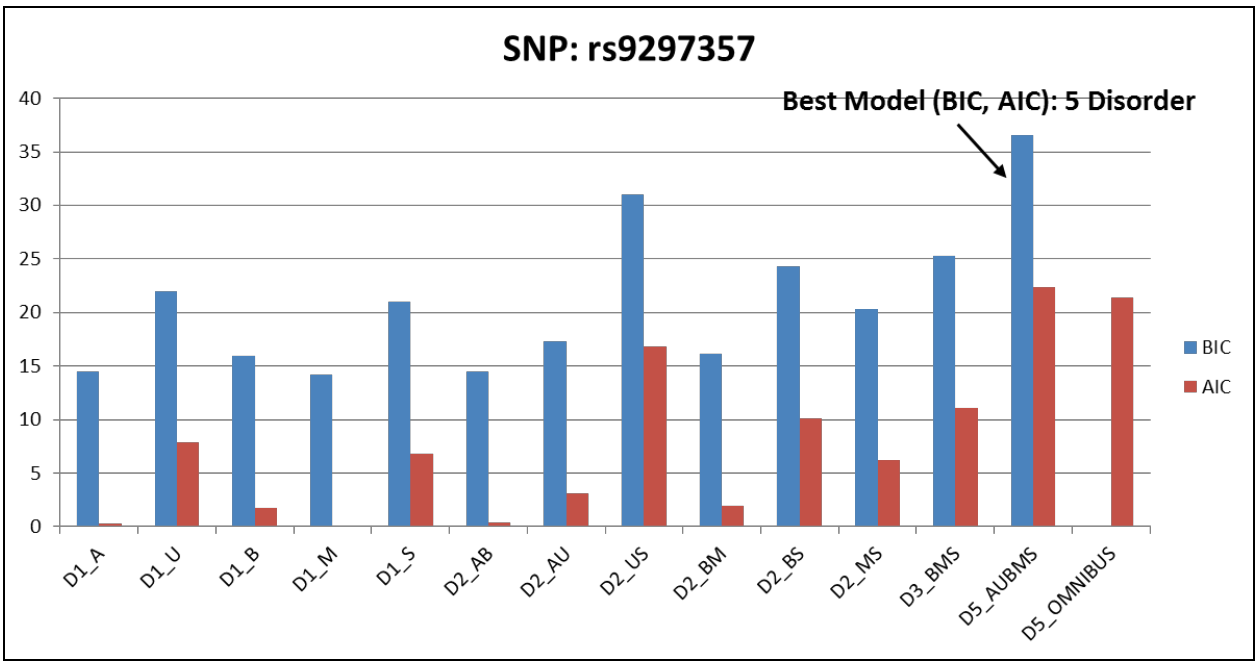
* SNP rs7096169 : the best and the second best model fitness scores are not significantly different (i.e., > 2), and therefore the best model prediction cannot be made for this SNP.

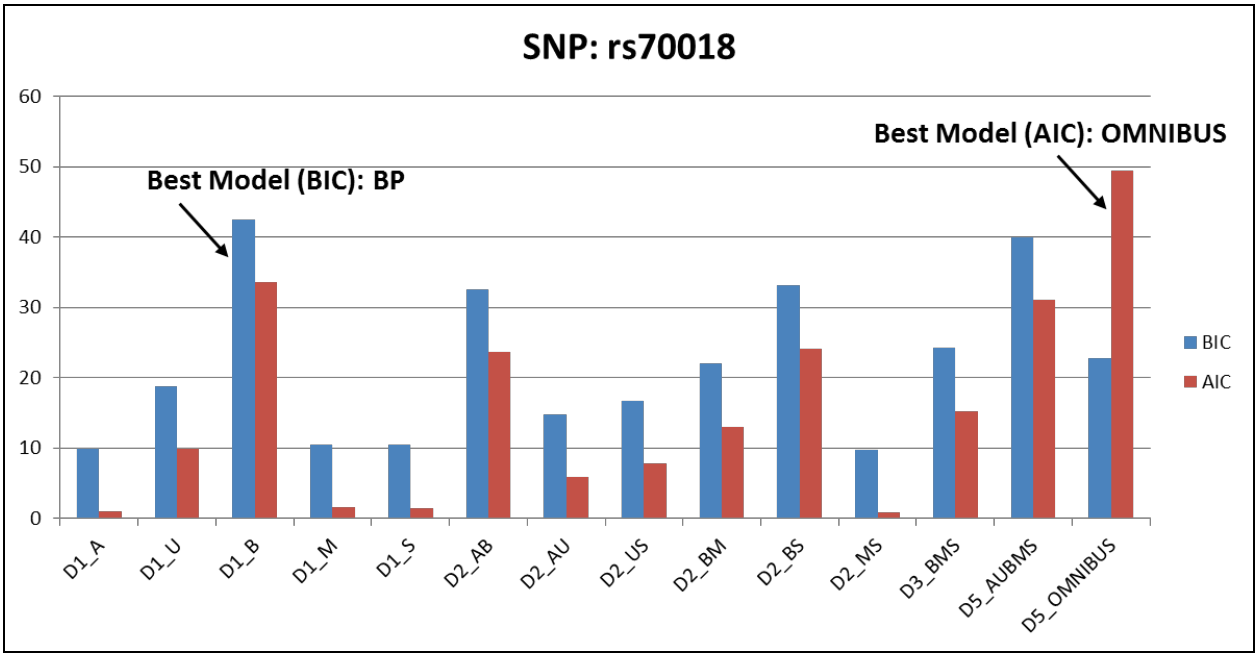

SNP: rs11152369
Best Model (BIC): Autism, SCZ
Best Model (AIC): OMINBUS
60
50
40
30
20
10
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

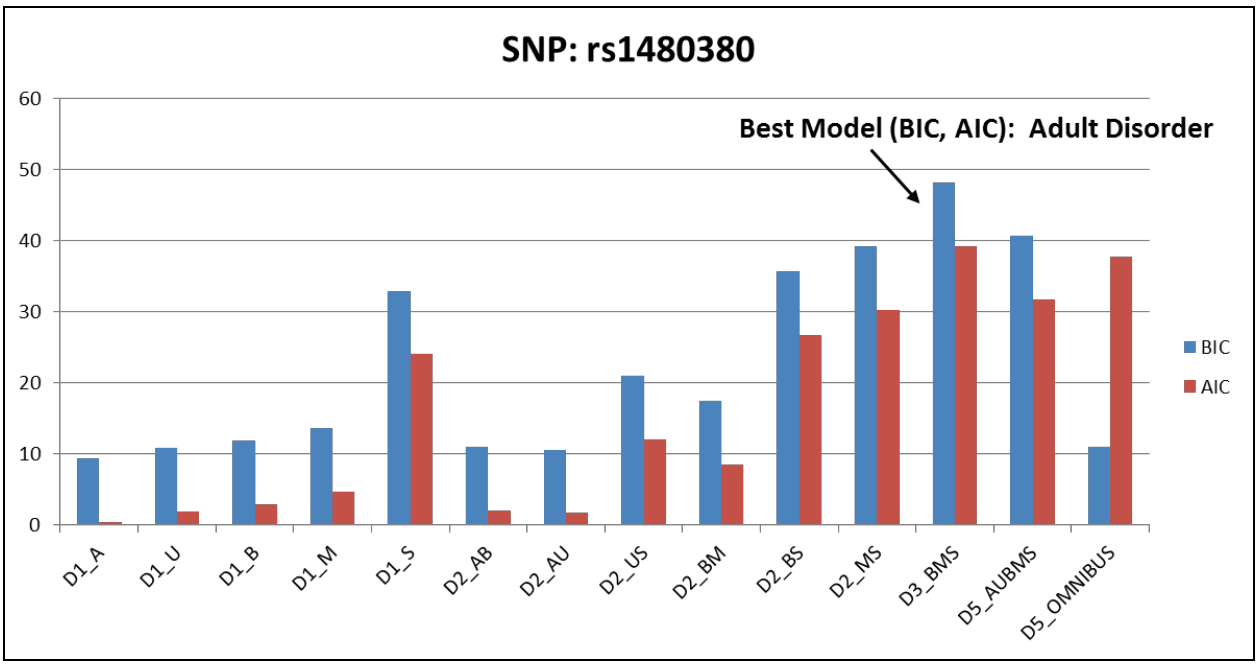

SNP: rs9275524
Best Model (AIC): OMINBUS
Best Model (BIC): 5 Disorder
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

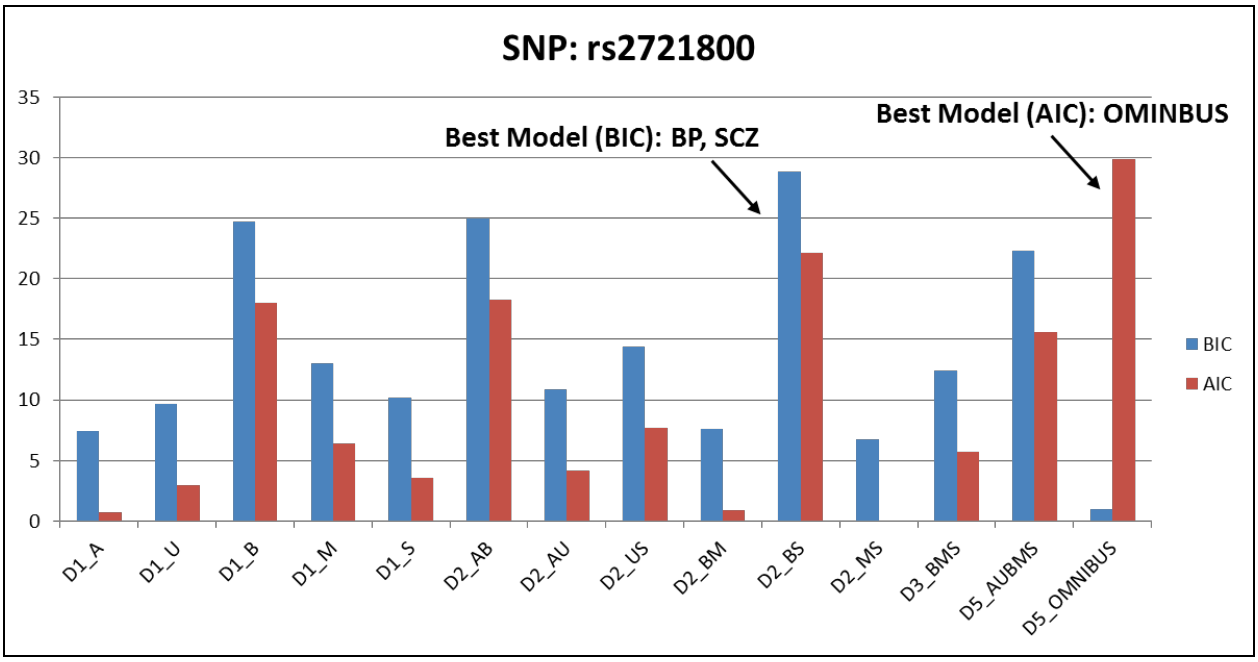

SNP: rs3132581
Best Model (AIC): OMINBUS
Best Model (BIC): 5 Disorder
60
50
40
30
20
10
0
BIC
AIC
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS

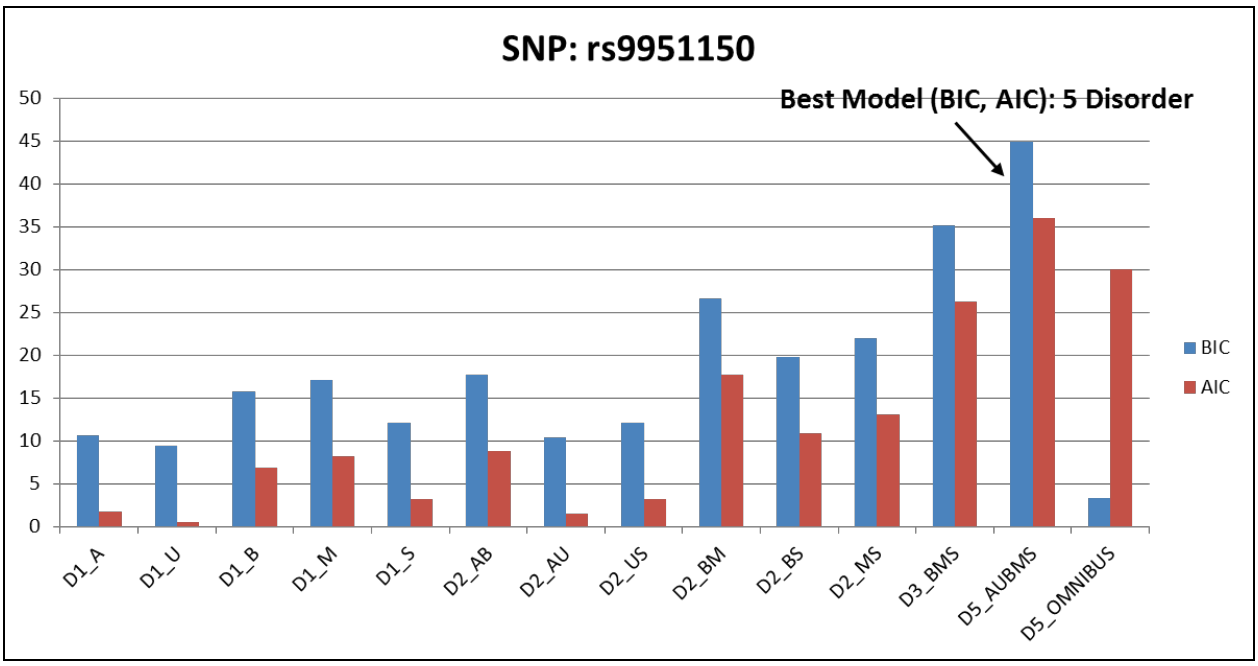

SNP: rs9276601
Best Model (BIC, AIC): 5 Disorder
45
40
35
30
25
20
15
10
5
0
BIC
AIC
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS

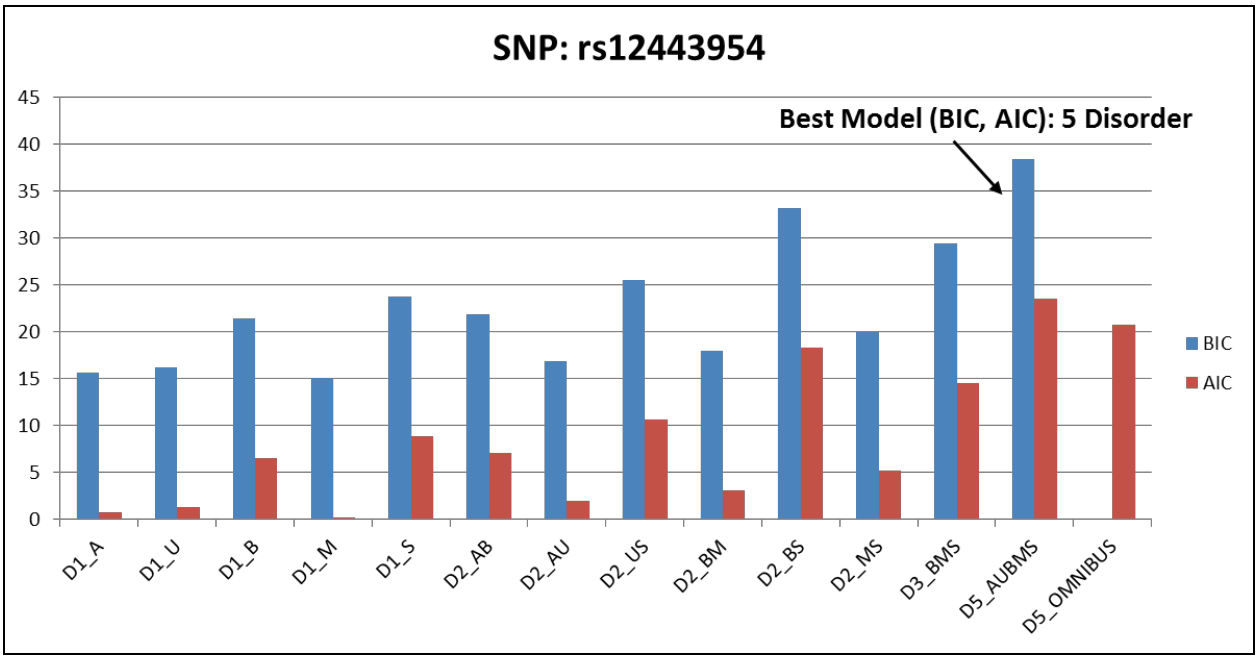

SNP: rs9297357
Best Model (BIC, AIC): 5 Disorder
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC


SNP: rs70018
Best Model (AIC): OMNIBUS
Best Model (BIC): BP
60
50
40
30
20
10
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC


SNP: rs1480380
Best Model (BIC, AIC): Adult Disorder
60
50
40
30
20
10
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

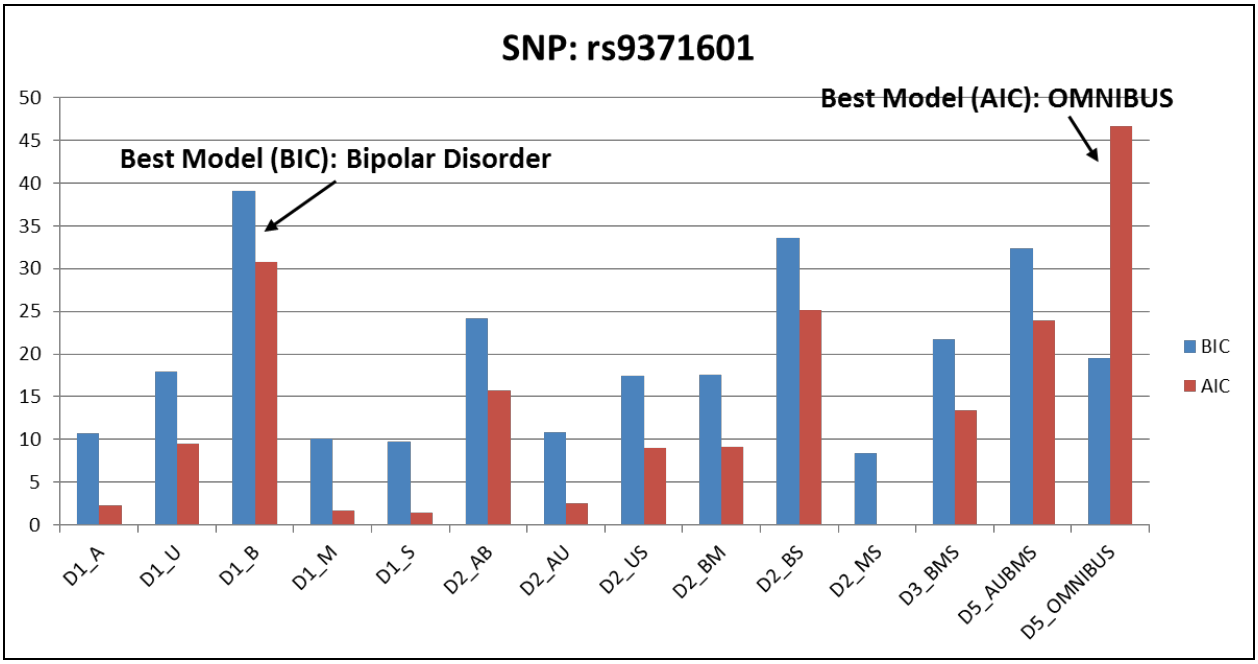

SNP: rs2721800
Best Model (BIC): BP, SCZ
Best Model (AIC): OMINBUS
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

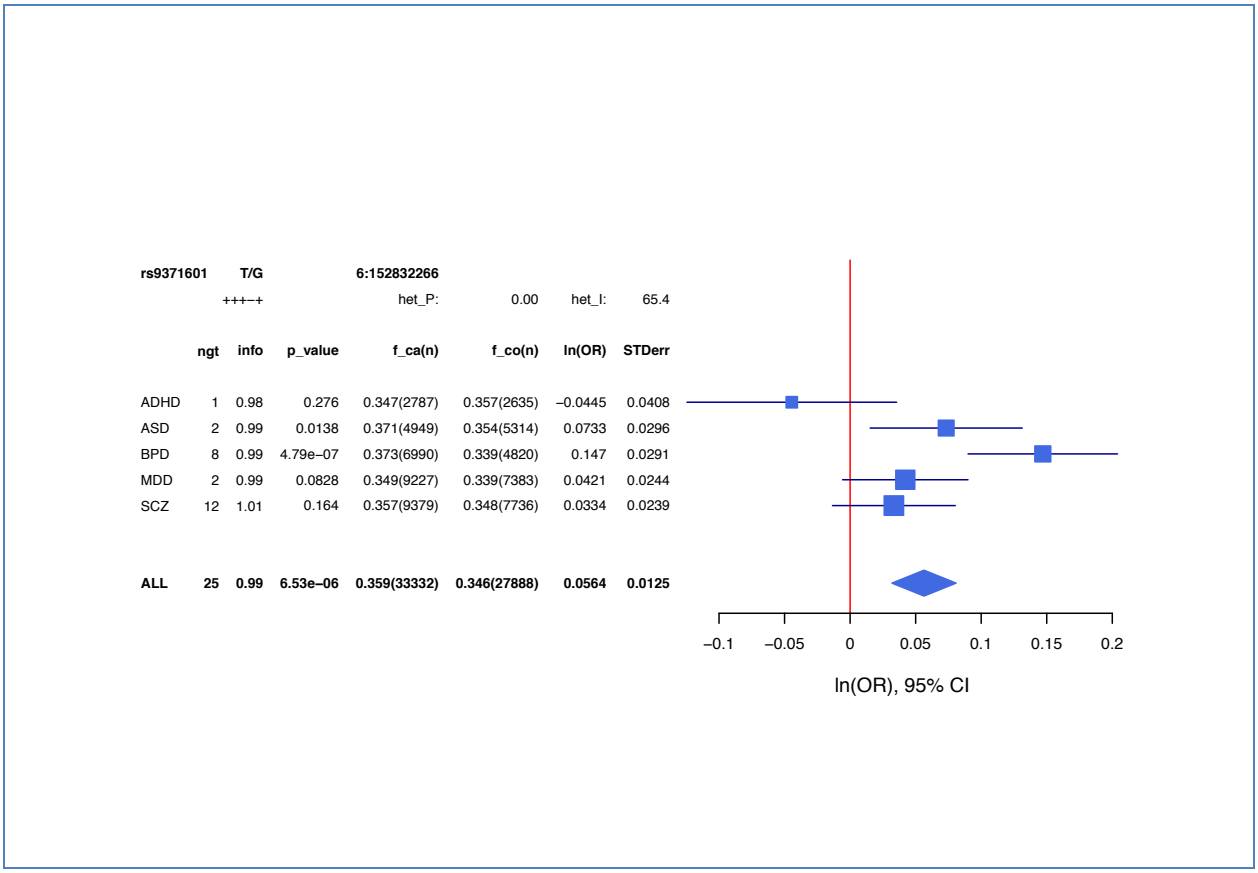

SNP: rs9951150
Best Model (BIC, AIC): 5 Disorder
50
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

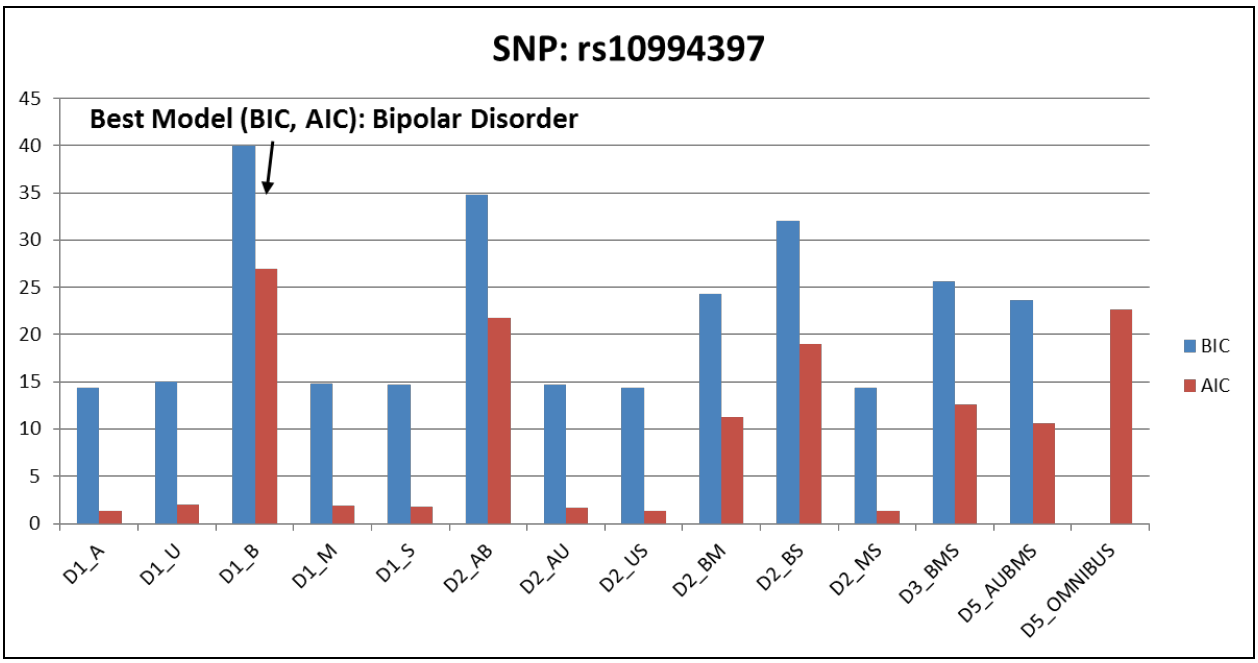

SNP: rs12443954
Best Model (BIC, AIC): 5 Disorder
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

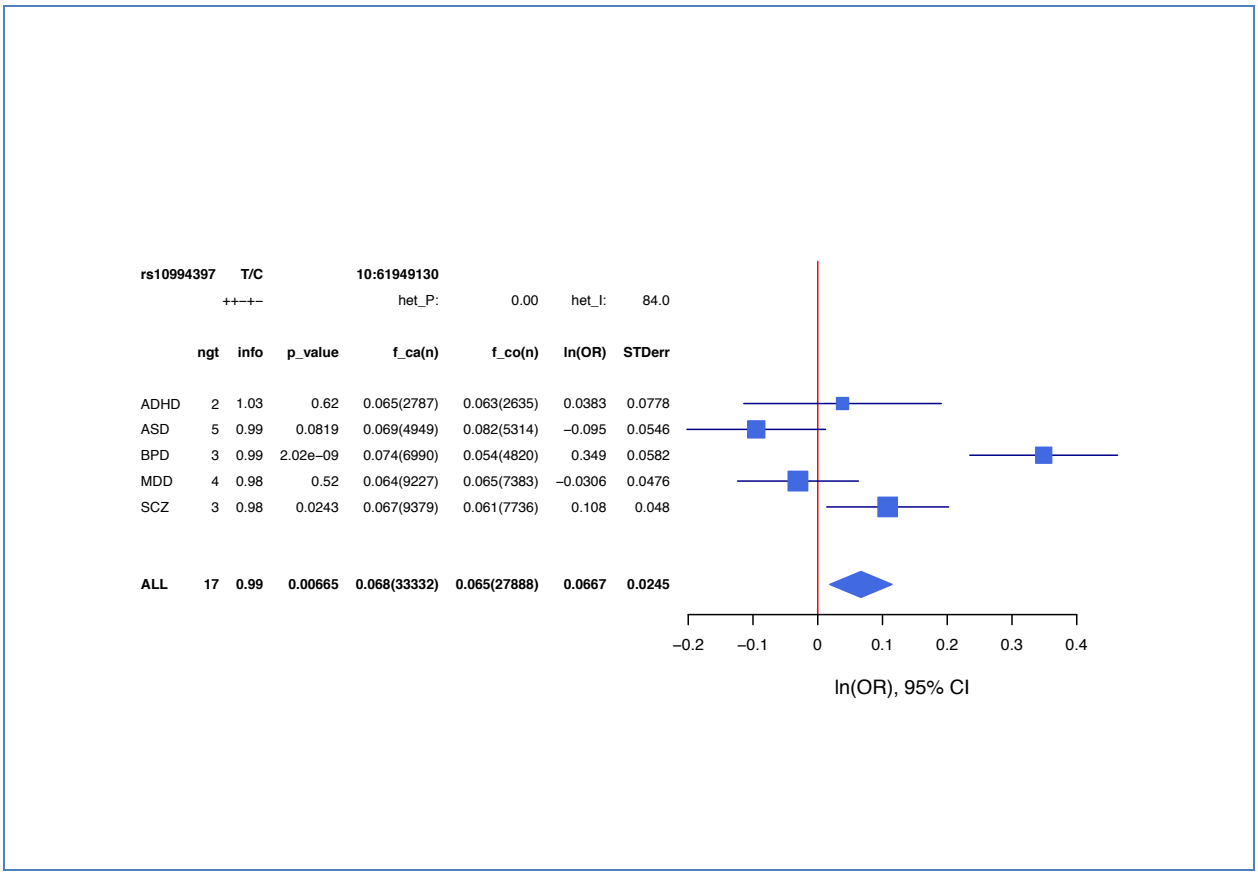
XIV. Figure S6. BIC/AIC results and Forest Plots for SNPs Associated with BPD in Prior PGC Analyses

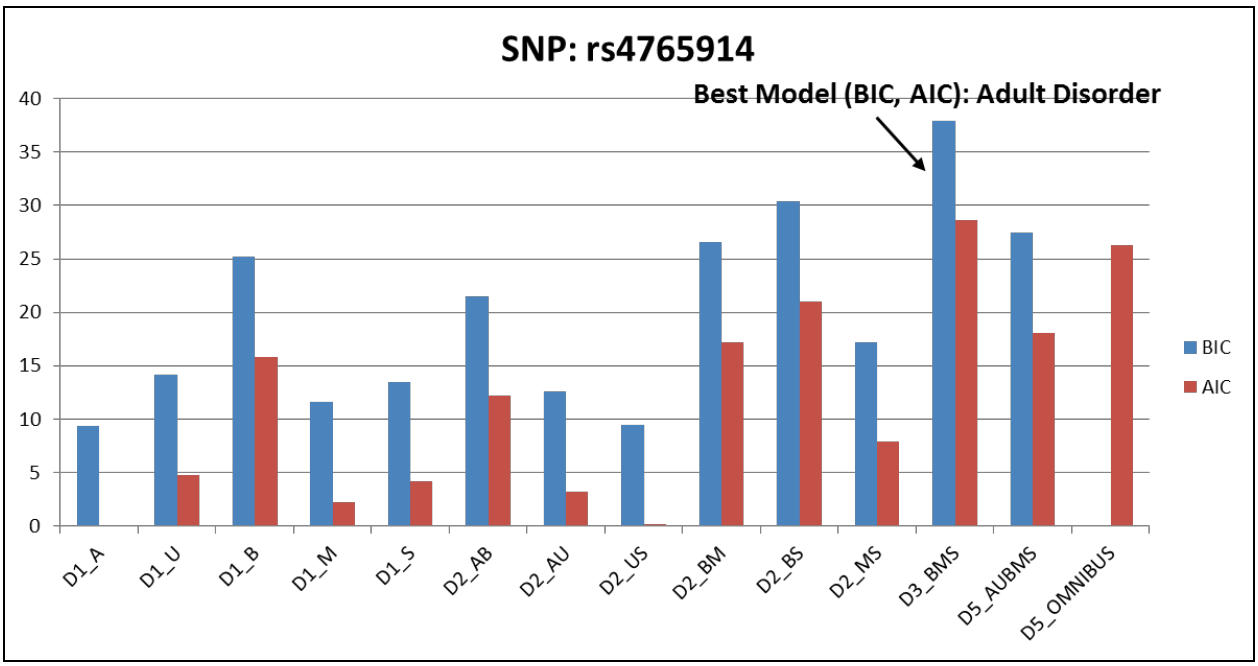
Two information criterion measures of disease subtype models for 4 SNPs in regions with association $p < 5 \times 10^{-8}$ in either the discovery or the combined PGC BP study [2]. Blue bar and red bar represent score differences across 21 models with respect to Bayesian information criterion (BIC) and Akaike information criterion (AIC) measures, respectively. X-axis represents 21 disease causal models, while Y-axis represents the relative difference in the information criterion score of each model compared to that of the highest score model (i.e., worst-fit model). For clarification, the best-fit model is marked by a circle in each plot. Note that the best-fit model could not be determined when the score difference between the best and the second-best model is at most 2. [17]

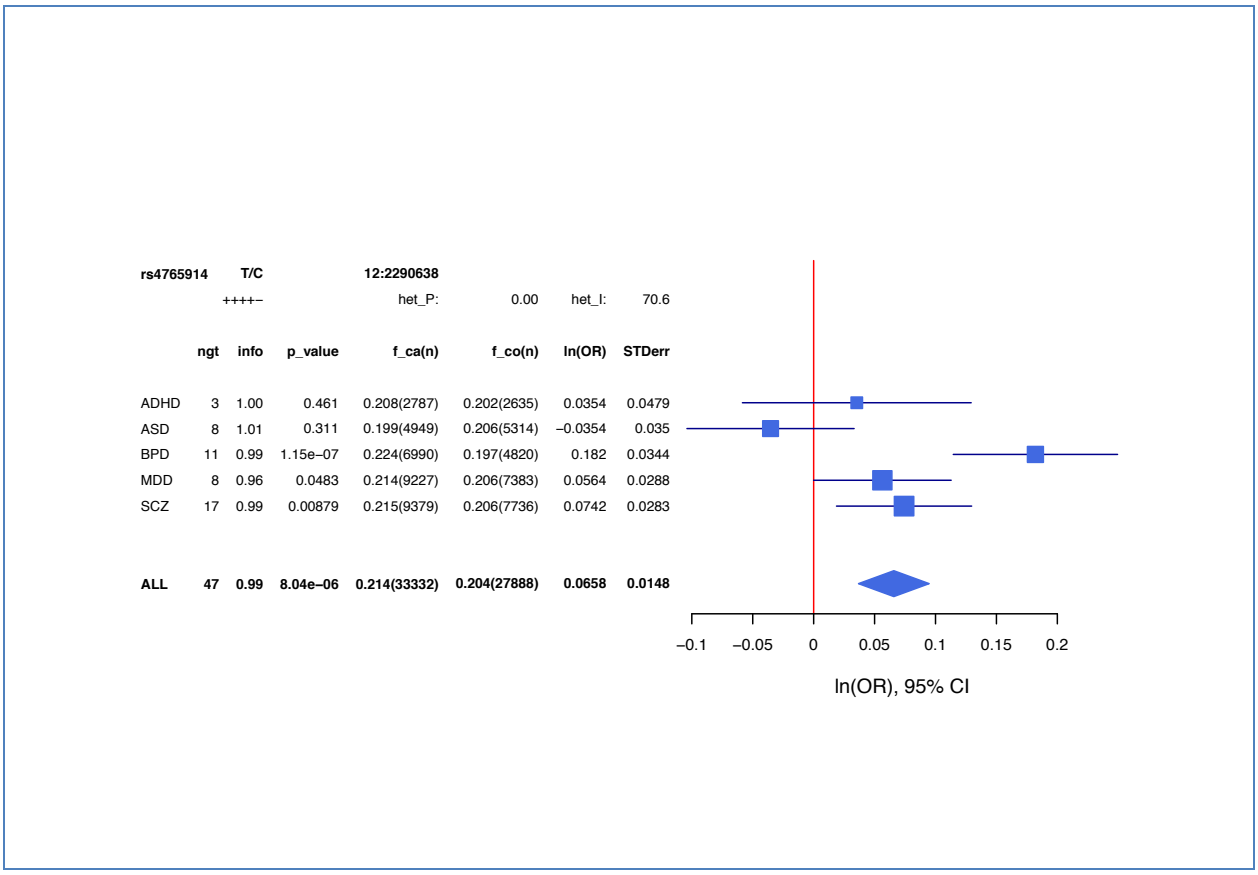

SNP: rs9371601
Best Model (AIC): OMNIBUS
Best Model (BIC): Bipolar Disorder
50
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC


rs9371601 T/G 6:152832266
+++−+ het_P: 0.00 het_I: 65.4
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 1 0.98 0.276 0.347(2787) 0.357(2635) −0.0445 0.0408
ASD 2 0.99 0.0138 0.371(4949) 0.354(5314) 0.0733 0.0296
BPD 8 0.99 4.79e−07 0.373(6990) 0.339(4820) 0.147 0.0291
MDD 2 0.99 0.0828 0.349(9227) 0.339(7383) 0.0421 0.0244
SCZ 12 1.01 0.164 0.357(9379) 0.348(7736) 0.0334 0.0239
ALL 25 0.99 6.53e−06 0.359(33332) 0.346(27888) 0.0564 0.0125
−0.1
−0.05
0
0.05
0.1
0.15
0.2
ln(OR), 95% CI

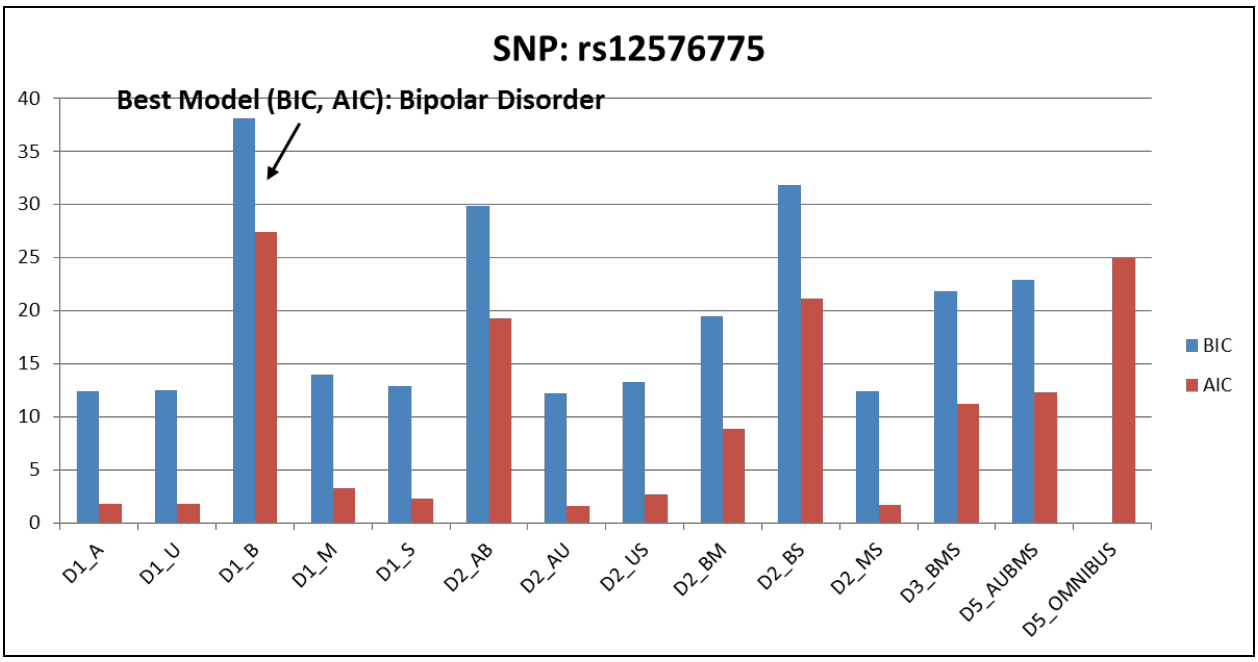

SNP: rs10994397
Best Model (BIC, AIC): Bipolar Disorder
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

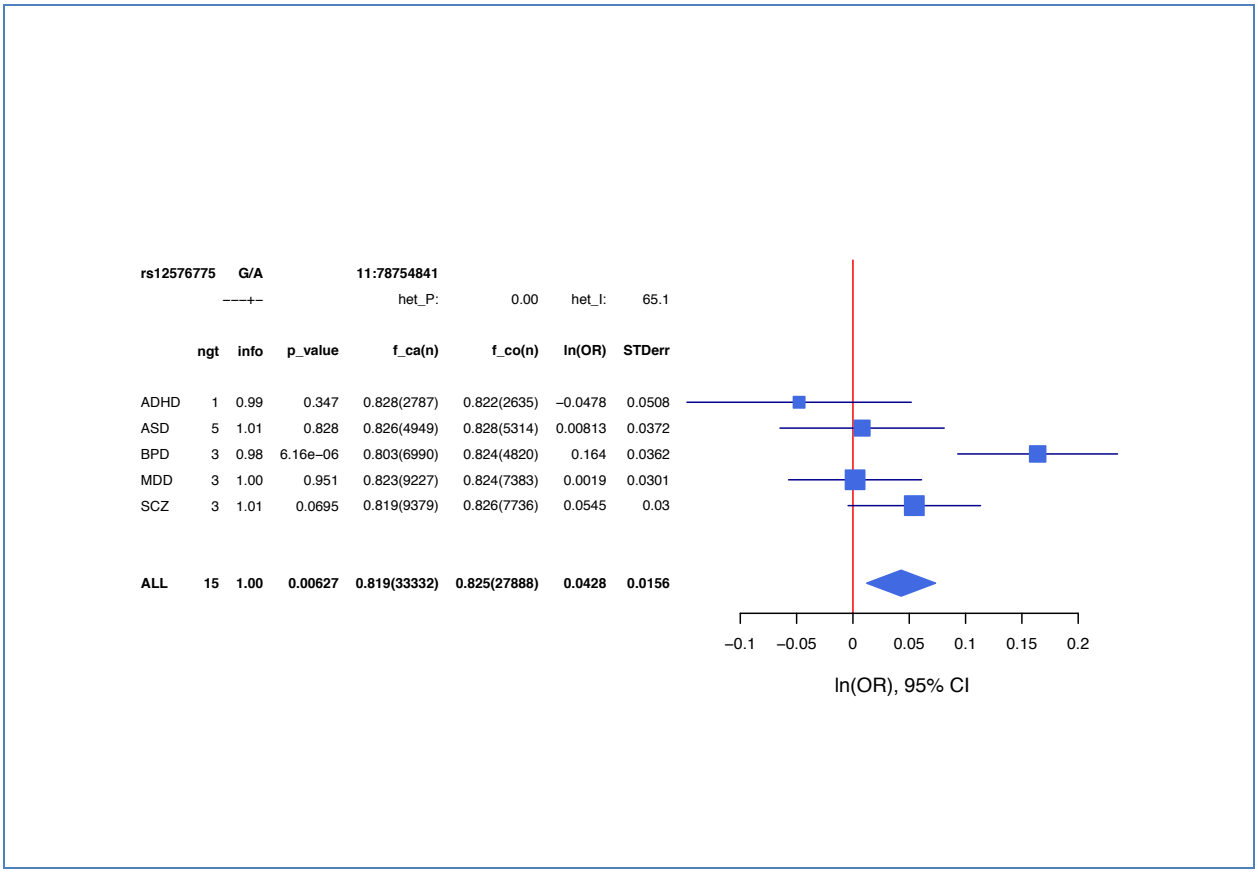

rs10994397 T/C 10:61949130
++−+− het_P: 0.00 het_I: 84.0
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 2 1.03 0.62 0.065(2787) 0.063(2635) 0.0383 0.0778
ASD 5 0.99 0.0819 0.069(4949) 0.082(5314) −0.095 0.0546
BPD 3 0.99 2.02e−09 0.074(6990) 0.054(4820) 0.349 0.0582
MDD 4 0.98 0.52 0.064(9227) 0.065(7383) −0.0306 0.0476
SCZ 3 0.98 0.0243 0.067(9379) 0.061(7736) 0.108 0.048
ALL 17 0.99 0.00665 0.068(33332) 0.065(27888) 0.0667 0.0245
−0.2
−0.1
0
0.1
0.2
0.3
0.4
ln(OR), 95% CI





rs4765914	T/C		12:2290638			
	++++–		het_P:	0.00	het_I:	70.6
	ngt	**info**	**p_value**	**f_ca(n)**	**f_co(n)**	**ln(OR)**	**STDerr**
ADHD	3	1.00	0.461	0.208(2787)	0.202(2635)	0.0354	0.0479
ASD	8	1.01	0.311	0.199(4949)	0.206(5314)	–0.0354	0.035
BPD	11	0.99	1.15e–07	0.224(6990)	0.197(4820)	0.182	0.0344
MDD	8	0.96	0.0483	0.214(9227)	0.206(7383)	0.0564	0.0288
SCZ	17	0.99	0.00879	0.215(9379)	0.206(7736)	0.0742	0.0283
ALL	**47**	**0.99**	**8.04e–06**	**0.214(33332)**	**0.204(27888)**	**0.0658**	**0.0148**



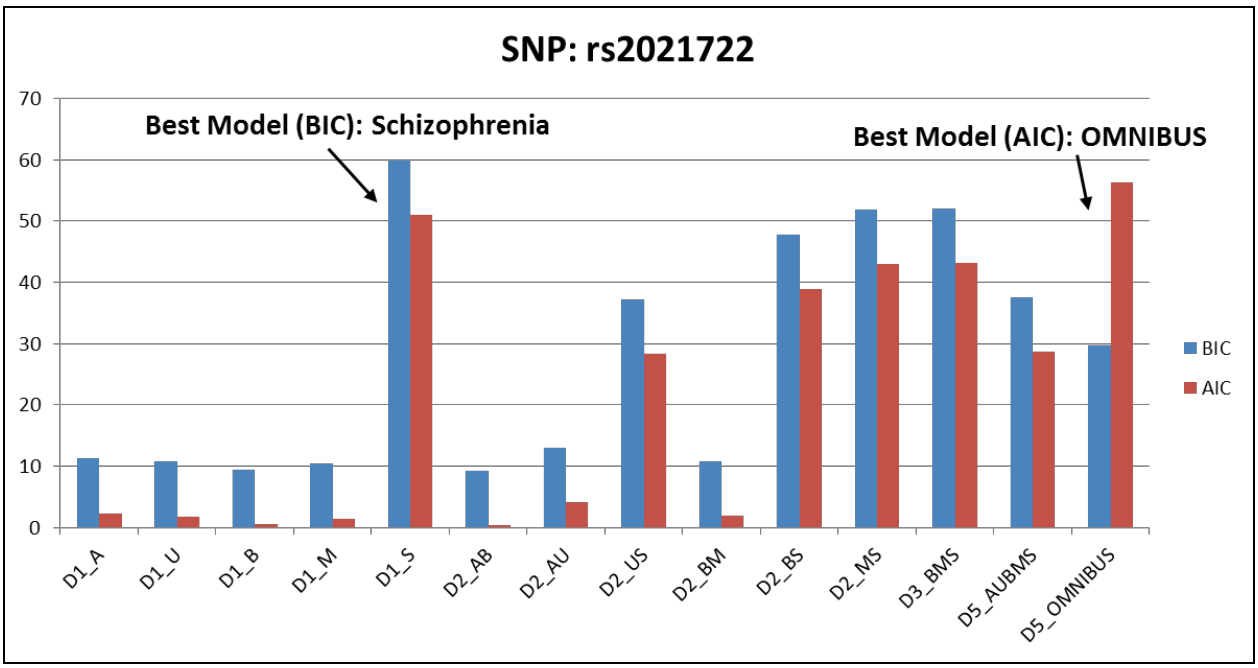


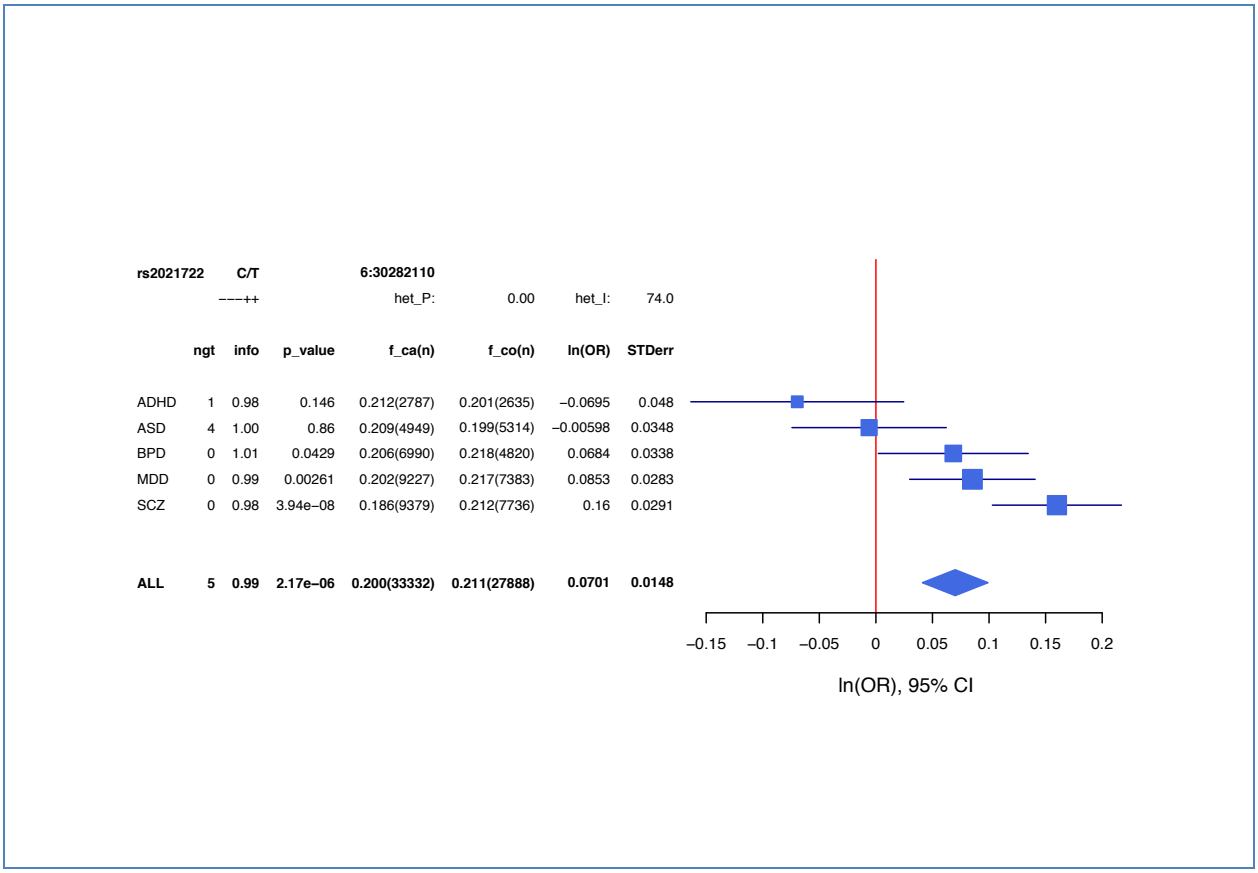
rs12576775	G/A		11:78754841			
	−−−+−		het_P:	0.00	het_I:	65.1

	ngt	info	p_value	f_ca(n)	f_co(n)	ln(OR)	STDerr
ADHD	1	0.99	0.347	0.828(2787)	0.822(2635)	−0.0478	0.0508
ASD	5	1.01	0.828	0.826(4949)	0.828(5314)	0.00813	0.0372
BPD	3	0.98	6.16e−06	0.803(6990)	0.824(4820)	0.164	0.0362
MDD	3	1.00	0.951	0.823(9227)	0.824(7383)	0.0019	0.0301
SCZ	3	1.01	0.0695	0.819(9379)	0.826(7736)	0.0545	0.03
ALL	**15**	**1.00**	**0.00627**	**0.819(33332)**	**0.825(27888)**	**0.0428**	**0.0156**

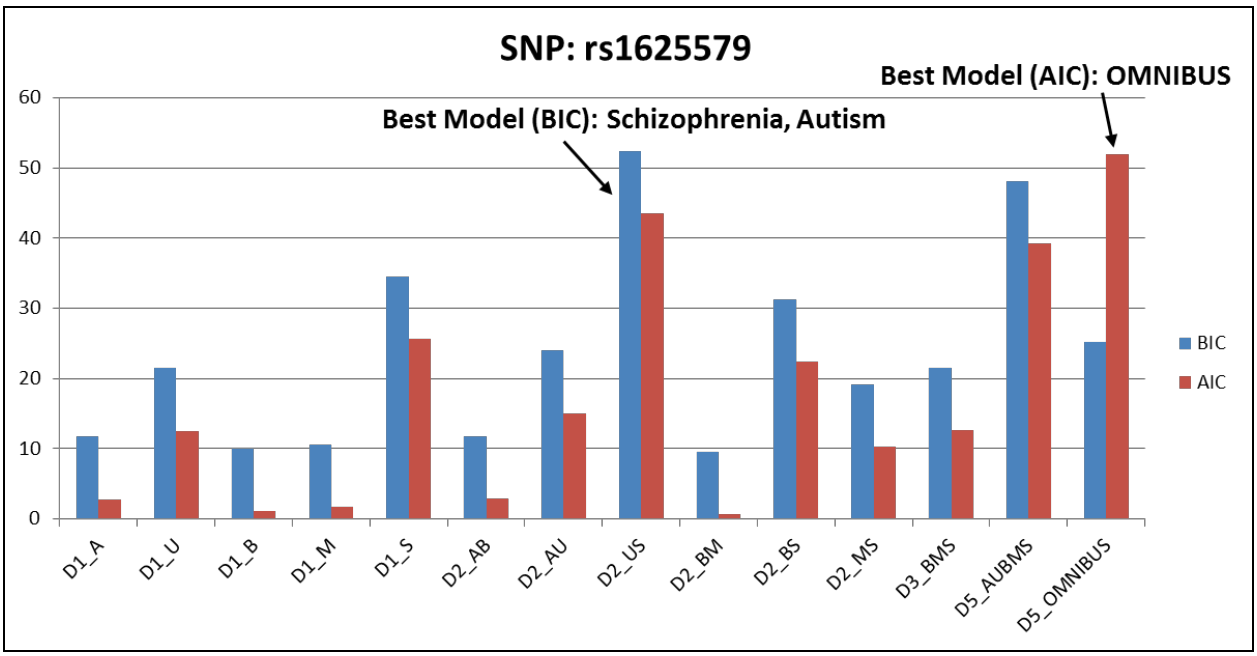
Figure S7. BIC/AIC results and Forest Plots for SNPs Associated with SCZ in Prior PGC Analyses

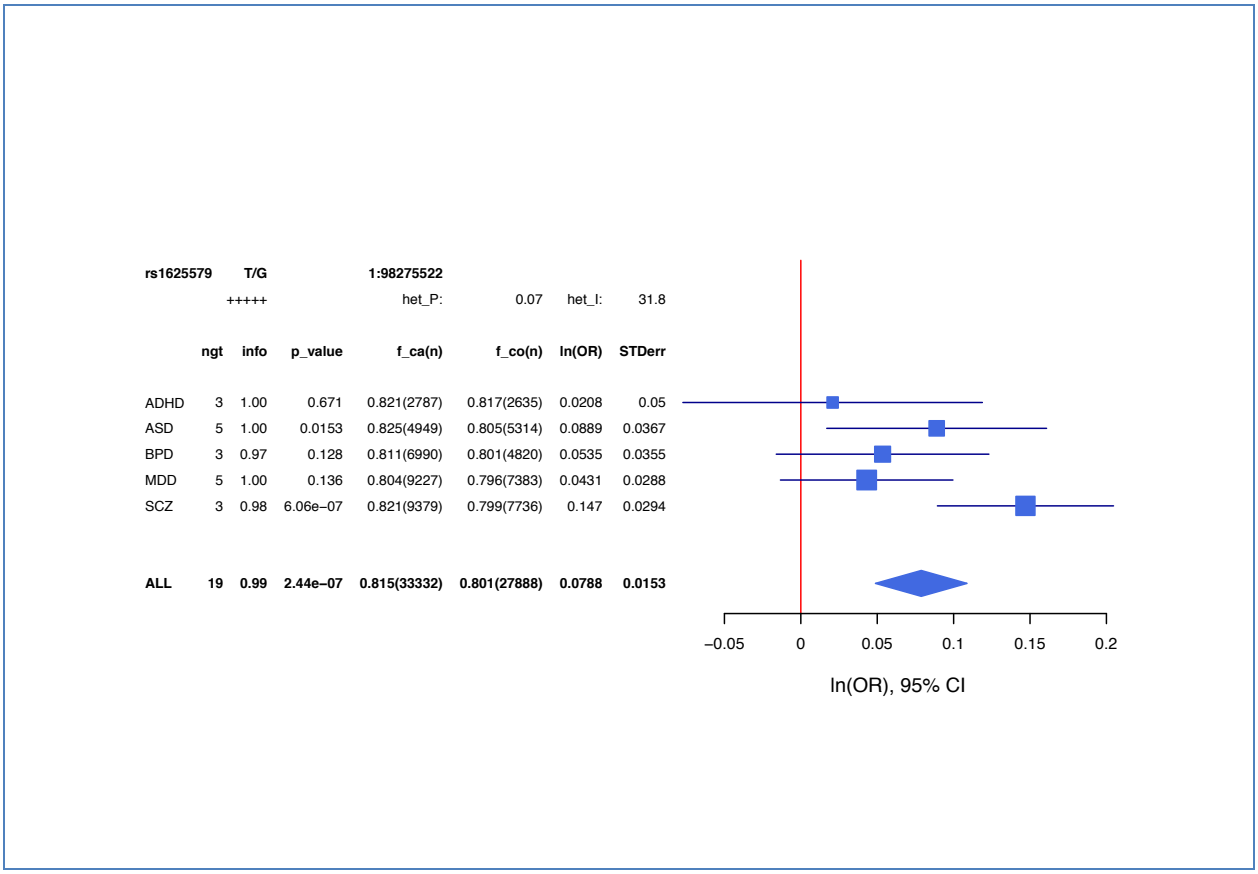
Two information criterion measures of disease subtype models for 10 SNPs in regions with the association $p < 5 \times 10^{-8}$ in either the discovery or the combined PGC schizophrenia study [3]. Blue bar and red bar represent score differences across 21 models with respect to Bayesian information criterion (BIC) and Akaike information criterion (AIC) measures, respectively. X-axis represents 21 disease causal models, while Y-axis represents the relative difference in the information criterion score of each model compared to that of the highest score model (i.e., worst-fit model). For clarification, the best-fit model is marked by a circle in each plot. Note that the best-fit model could not be determined when the score difference between the best and the second-best model is at most 2. [17]

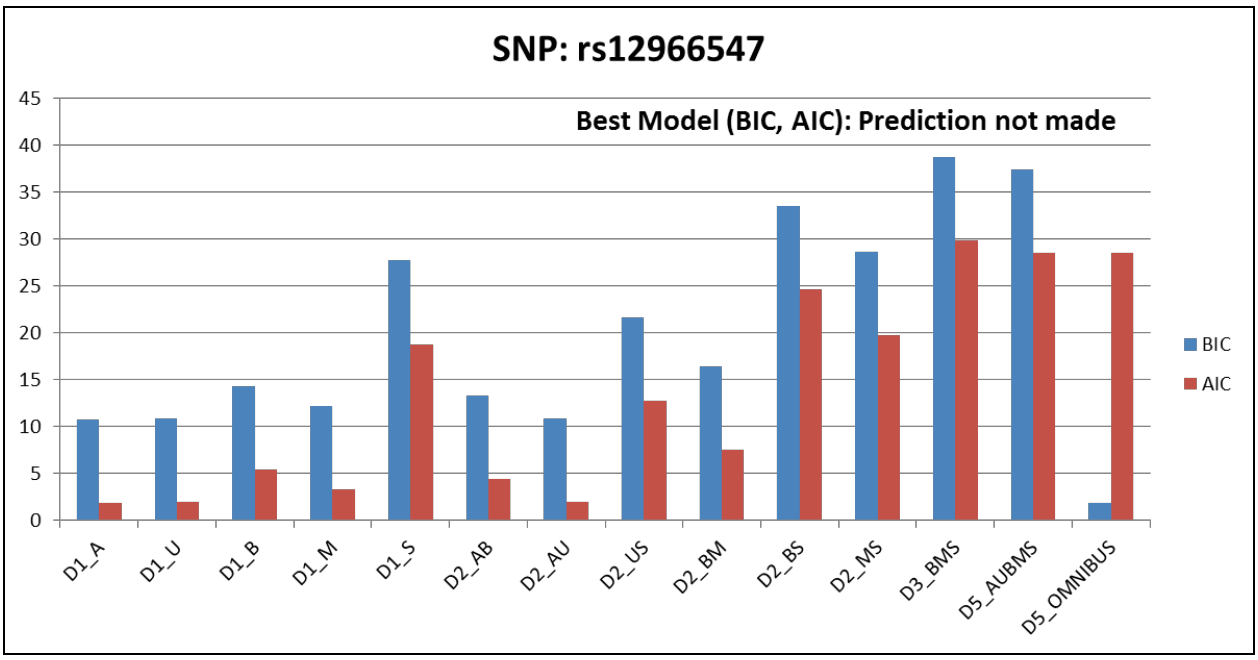


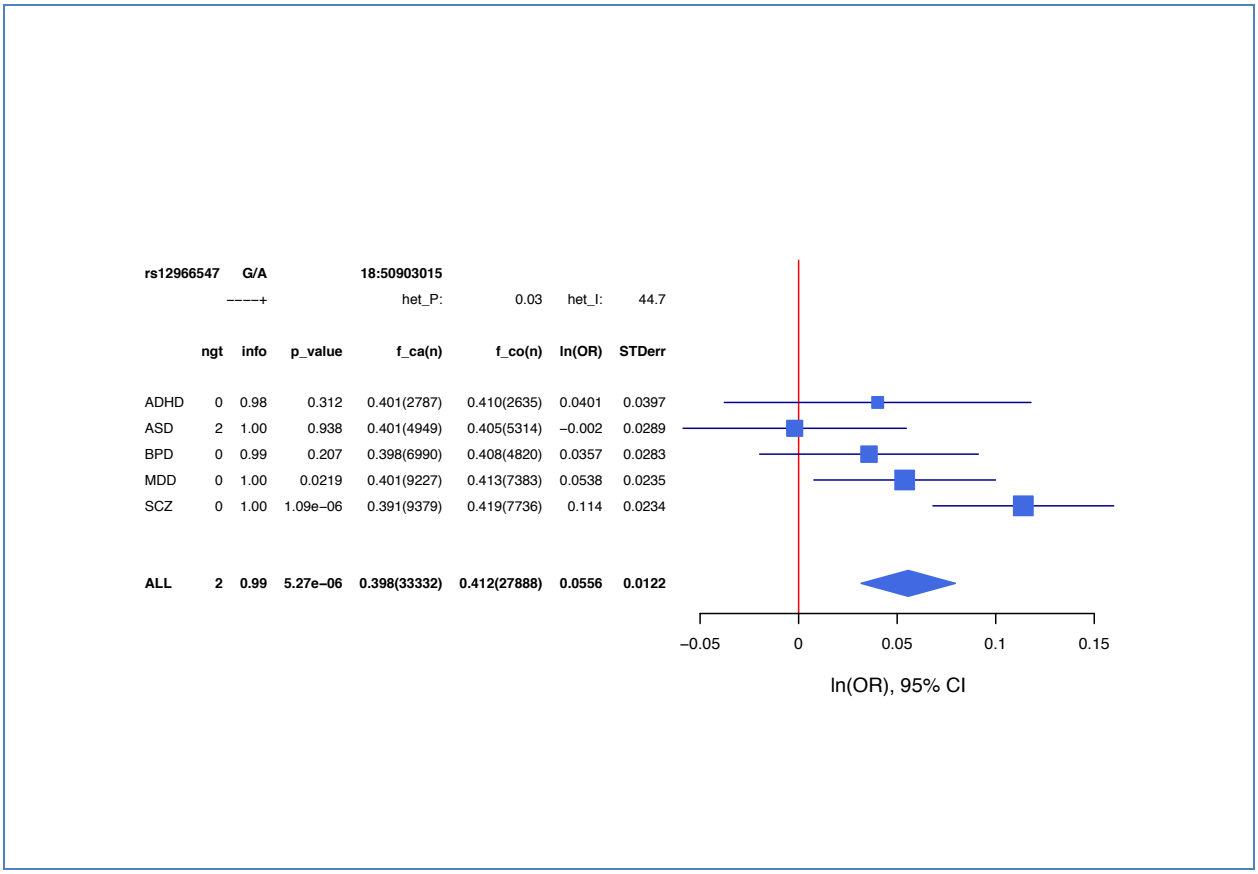


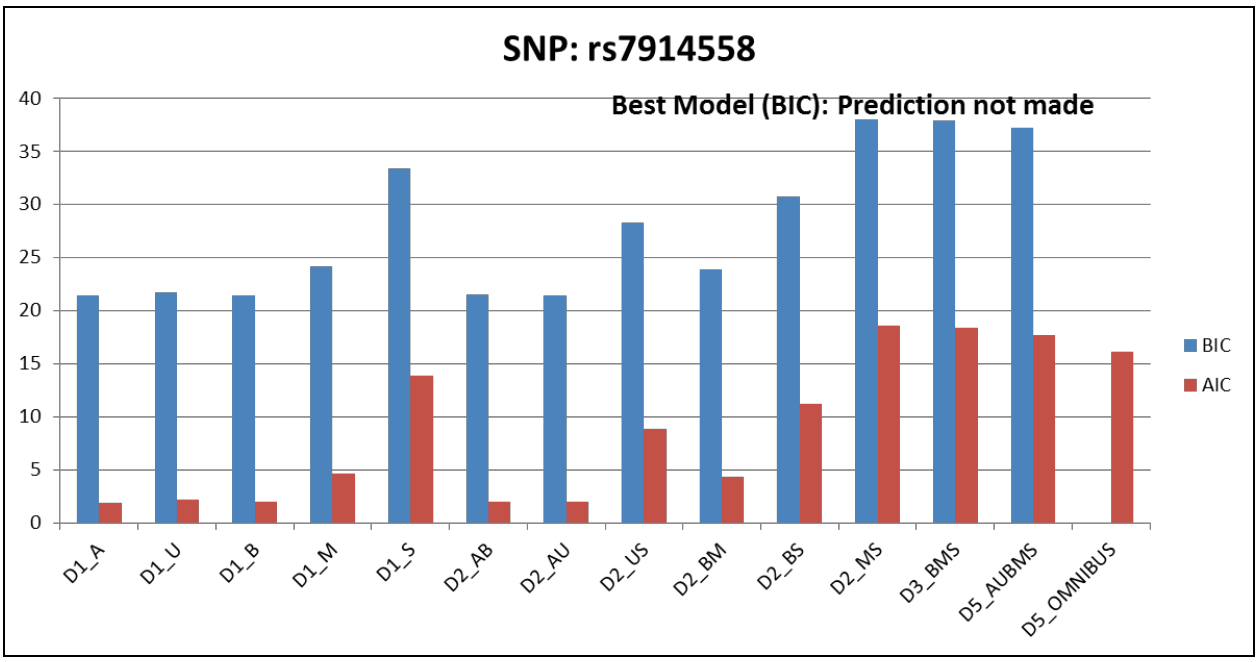
rs2021722	C/T		6:30282110			
	−−−++		het_P:	0.00	het_I:	74.0
	ngt	**info**	**p_value**	**f_ca(n)**	**f_co(n)**	**ln(OR)**	**STDerr**
ADHD	1	0.98	0.146	0.212(2787)	0.201(2635)	−0.0695	0.048
ASD	4	1.00	0.86	0.209(4949)	0.199(5314)	−0.00598	0.0348
BPD	0	1.01	0.0429	0.206(6990)	0.218(4820)	0.0684	0.0338
MDD	0	0.99	0.00261	0.202(9227)	0.217(7383)	0.0853	0.0283
SCZ	0	0.98	3.94e−08	0.186(9379)	0.212(7736)	0.16	0.0291
ALL	**5**	**0.99**	**2.17e−06**	**0.200(33332)**	**0.211(27888)**	**0.0701**	**0.0148**


SNP: rs1625579
Best Model (AIC): OMNIBUS
Best Model (BIC): Schizophrenia, Autism
60
50
40
30
20
10
0
BIC
AIC
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS

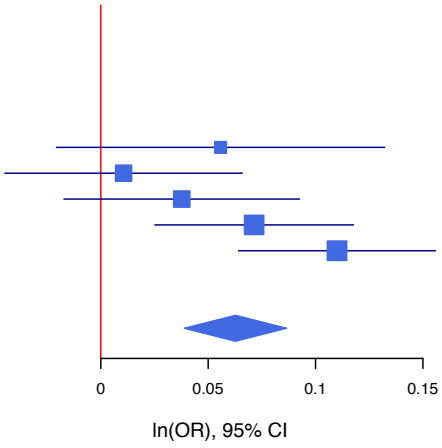

rs1625579 T/G 1:98275522
++++++ het_P: 0.07 het_I: 31.8
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 3 1.00 0.671 0.821(2787) 0.817(2635) 0.0208 0.05
ASD 5 1.00 0.0153 0.825(4949) 0.805(5314) 0.0889 0.0367
BPD 3 0.97 0.128 0.811(6990) 0.801(4820) 0.0535 0.0355
MDD 5 1.00 0.136 0.804(9227) 0.796(7383) 0.0431 0.0288
SCZ 3 0.98 6.06e−07 0.821(9379) 0.799(7736) 0.147 0.0294
ALL 19 0.99 2.44e−07 0.815(33332) 0.801(27888) 0.0788 0.0153
−0.05
0
0.05
0.1
0.15
0.2
ln(OR), 95% CI

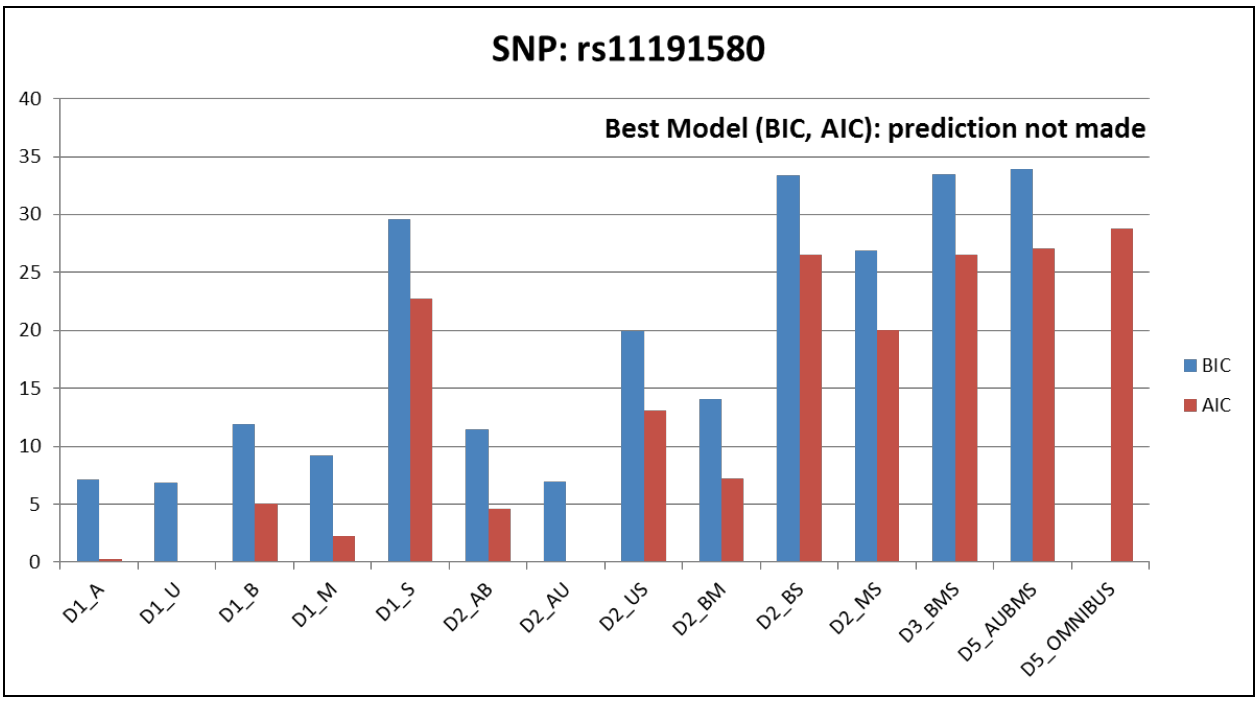

SNP: rs12966547
Best Model (BIC, AIC): Prediction not made
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

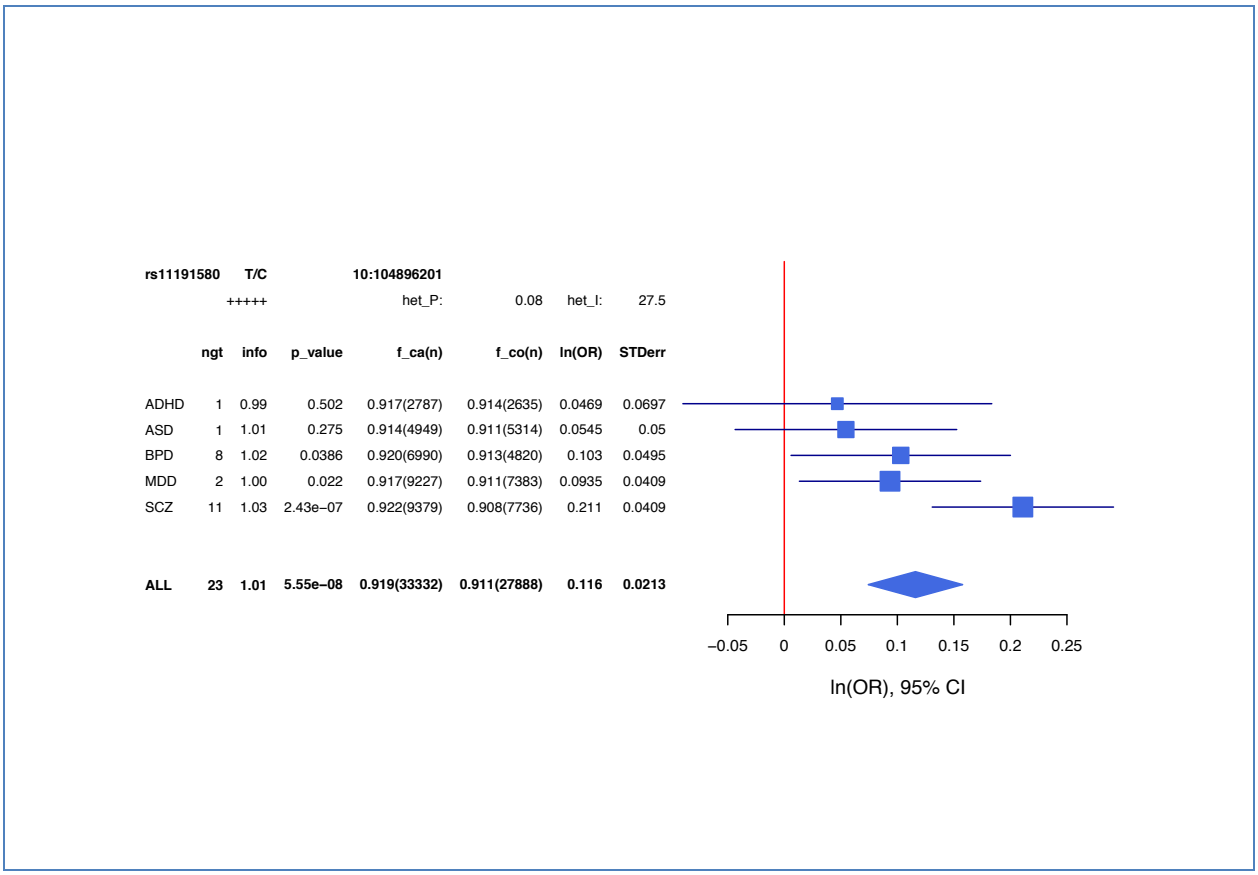

rs12966547 G/A 18:50903015
−−−−+ het_P: 0.03 het_I: 44.7
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 0 0.98 0.312 0.401(2787) 0.410(2635) 0.0401 0.0397
ASD 2 1.00 0.938 0.401(4949) 0.405(5314) −0.002 0.0289
BPD 0 0.99 0.207 0.398(6990) 0.408(4820) 0.0357 0.0283
MDD 0 1.00 0.0219 0.401(9227) 0.413(7383) 0.0538 0.0235
SCZ 0 1.00 1.09e−06 0.391(9379) 0.419(7736) 0.114 0.0234
ALL 2 0.99 5.27e−06 0.398(33332) 0.412(27888) 0.0556 0.0122
−0.05
0
0.05
0.1
0.15
ln(OR), 95% CI

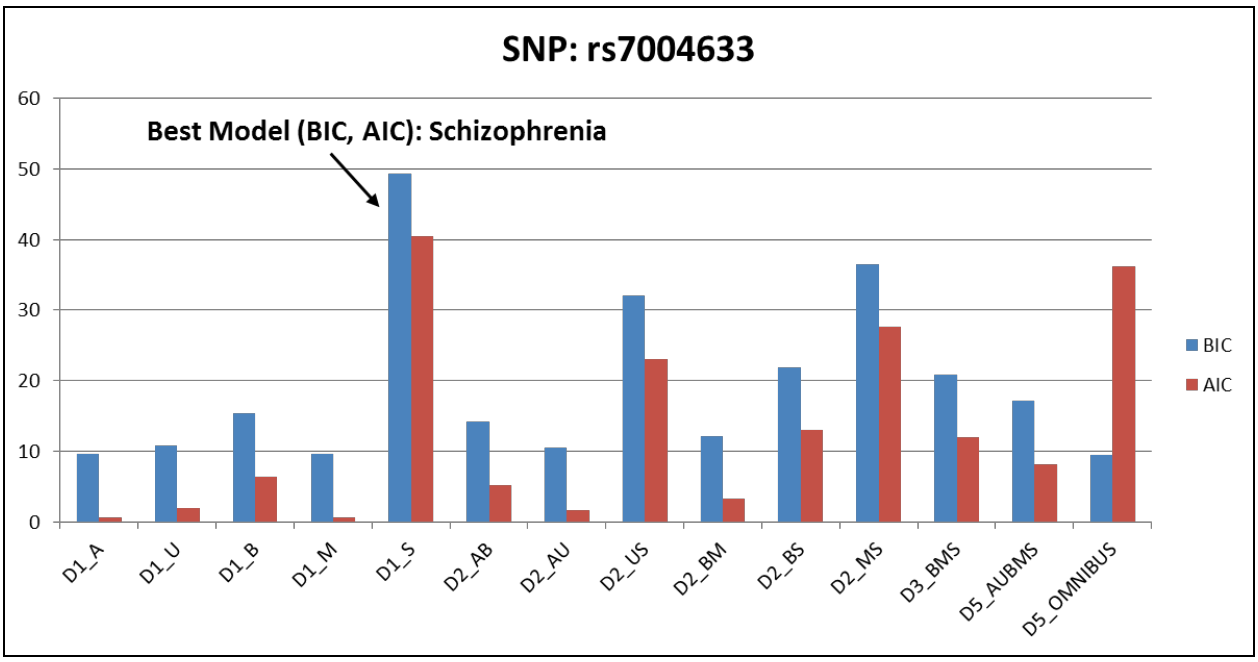


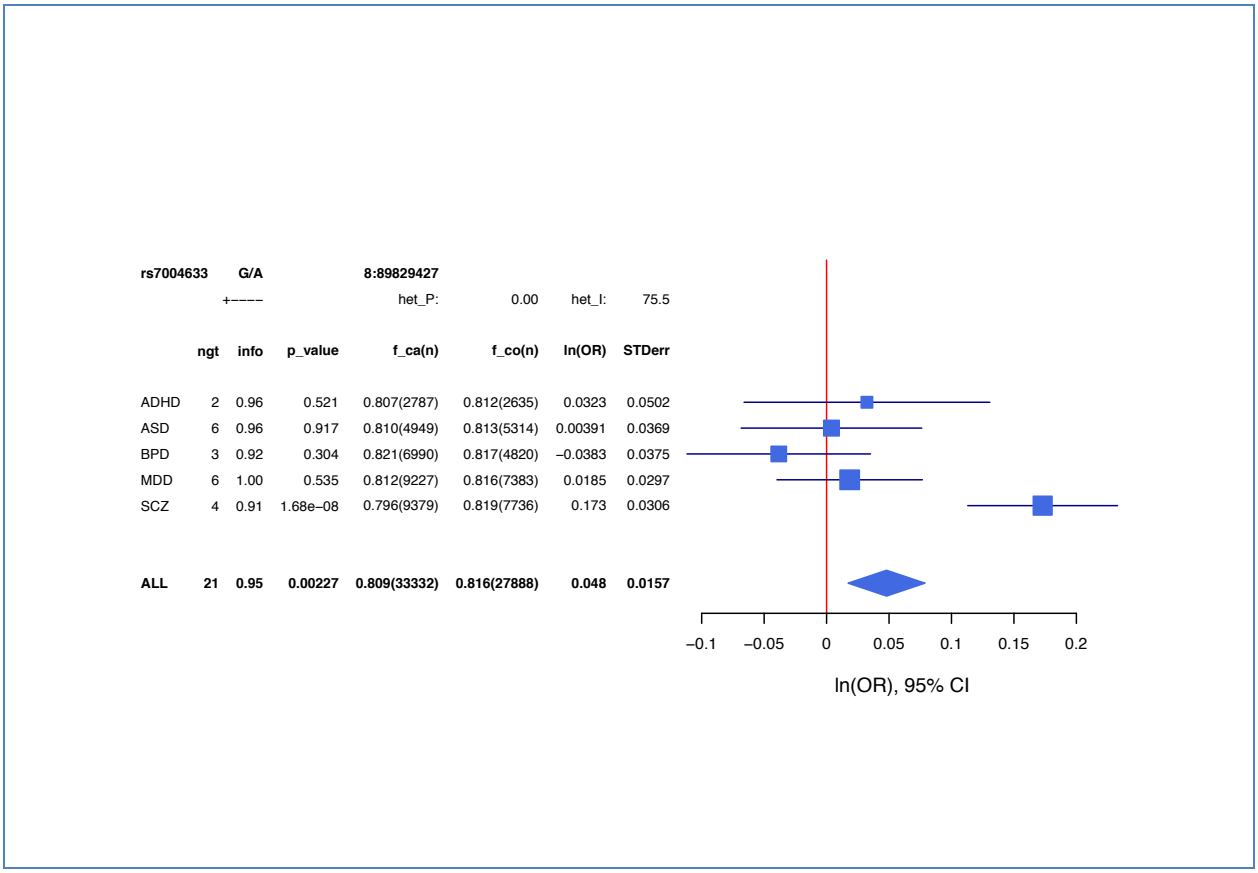
rs7914558	G/A		10:104765898				
	-----			het_P:	0.08	het_I:	29.2
	ngt	info	p_value	f_ca(n)	f_co(n)	ln(OR)	STDerr
ADHD	1	1.01	0.152	0.407(2787)	0.421(2635)	0.0558	0.039
ASD	4	1.03	0.707	0.412(4949)	0.413(5314)	0.0106	0.0282
BPD	0	1.01	0.178	0.402(6990)	0.408(4820)	0.0377	0.028
MDD	0	0.99	0.00245	0.399(9227)	0.411(7383)	0.0714	0.0236
SCZ	0	1.00	2.59e−06	0.394(9379)	0.415(7736)	0.11	0.0234
ALL	**5**	**1.01**	**2.27e−07**	**0.401(33332)**	**0.413(27888)**	**0.0627**	**0.0121**

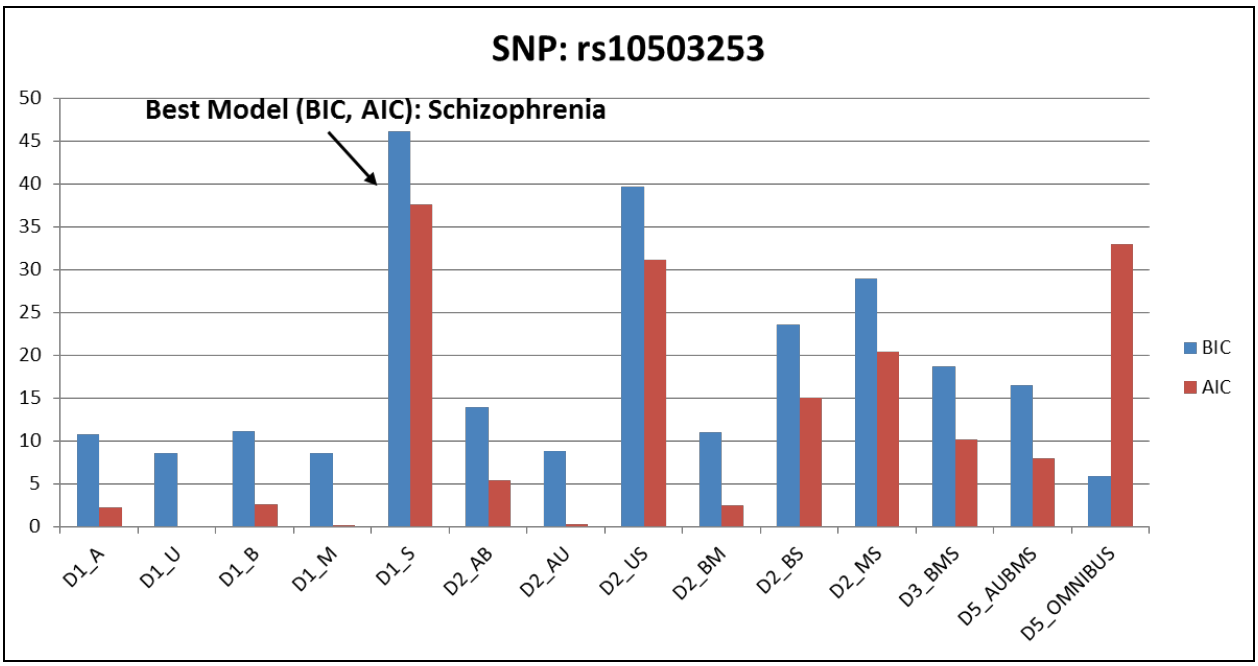


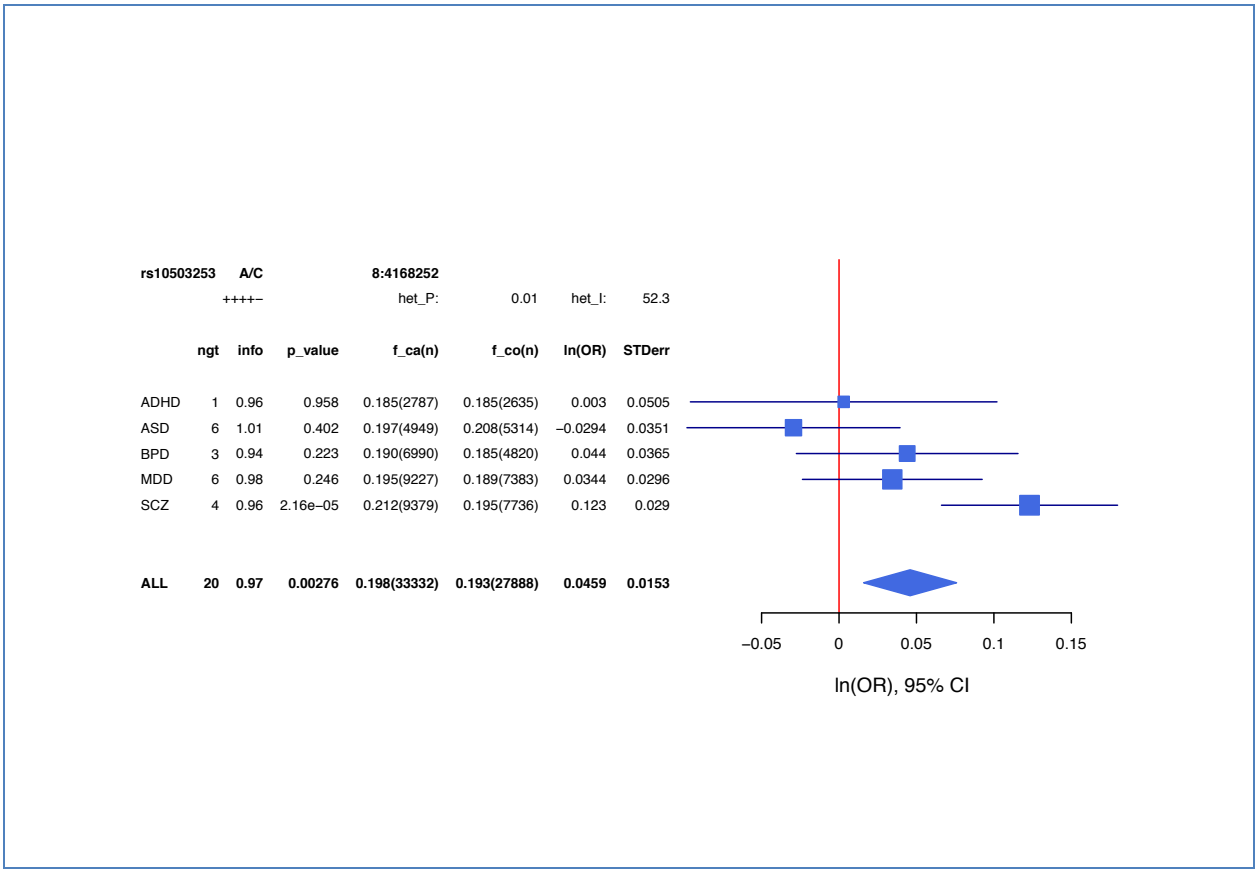

SNP: rs11191580
Best Model (BIC, AIC): prediction not made
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

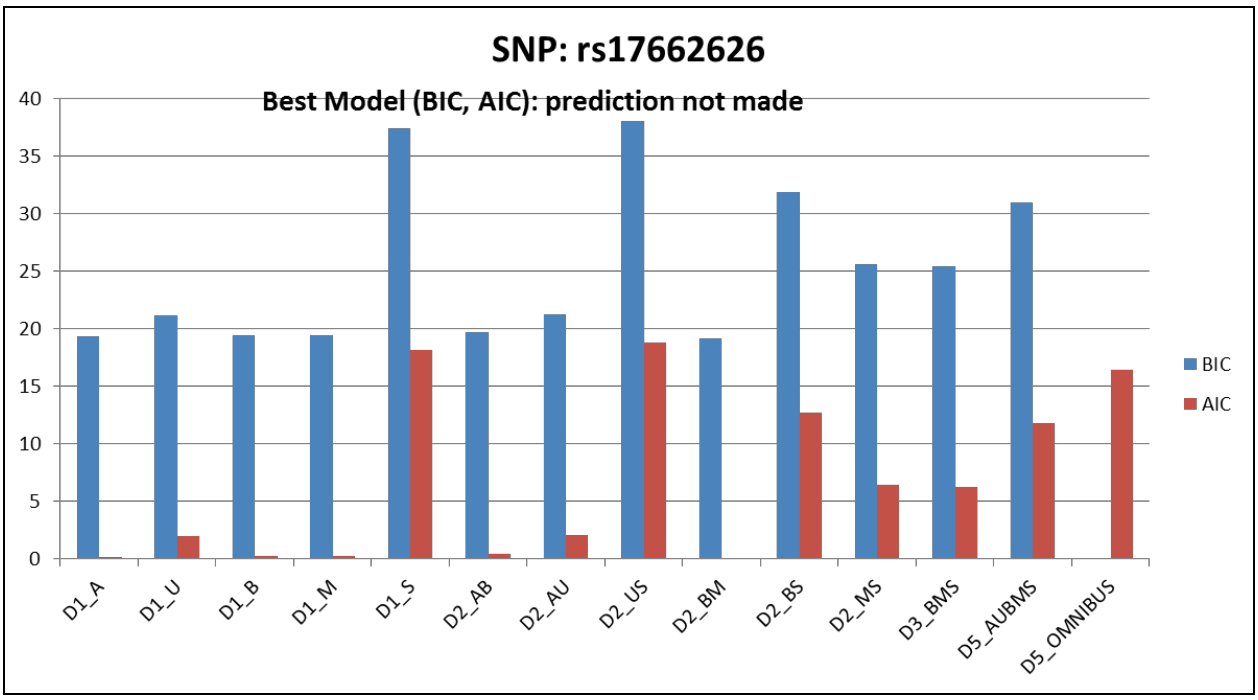

rs11191580 T/C 10:104896201
+++++ het_P: 0.08 het_I: 27.5
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 1 0.99 0.502 0.917(2787) 0.914(2635) 0.0469 0.0697
ASD 1 1.01 0.275 0.914(4949) 0.911(5314) 0.0545 0.05
BPD 8 1.02 0.0386 0.920(6990) 0.913(4820) 0.103 0.0495
MDD 2 1.00 0.022 0.917(9227) 0.911(7383) 0.0935 0.0409
SCZ 11 1.03 2.43e−07 0.922(9379) 0.908(7736) 0.211 0.0409
ALL 23 1.01 5.55e−08 0.919(33332) 0.911(27888) 0.116 0.0213
−0.05 0 0.05 0.1 0.15 0.2 0.25
ln(OR), 95% CI

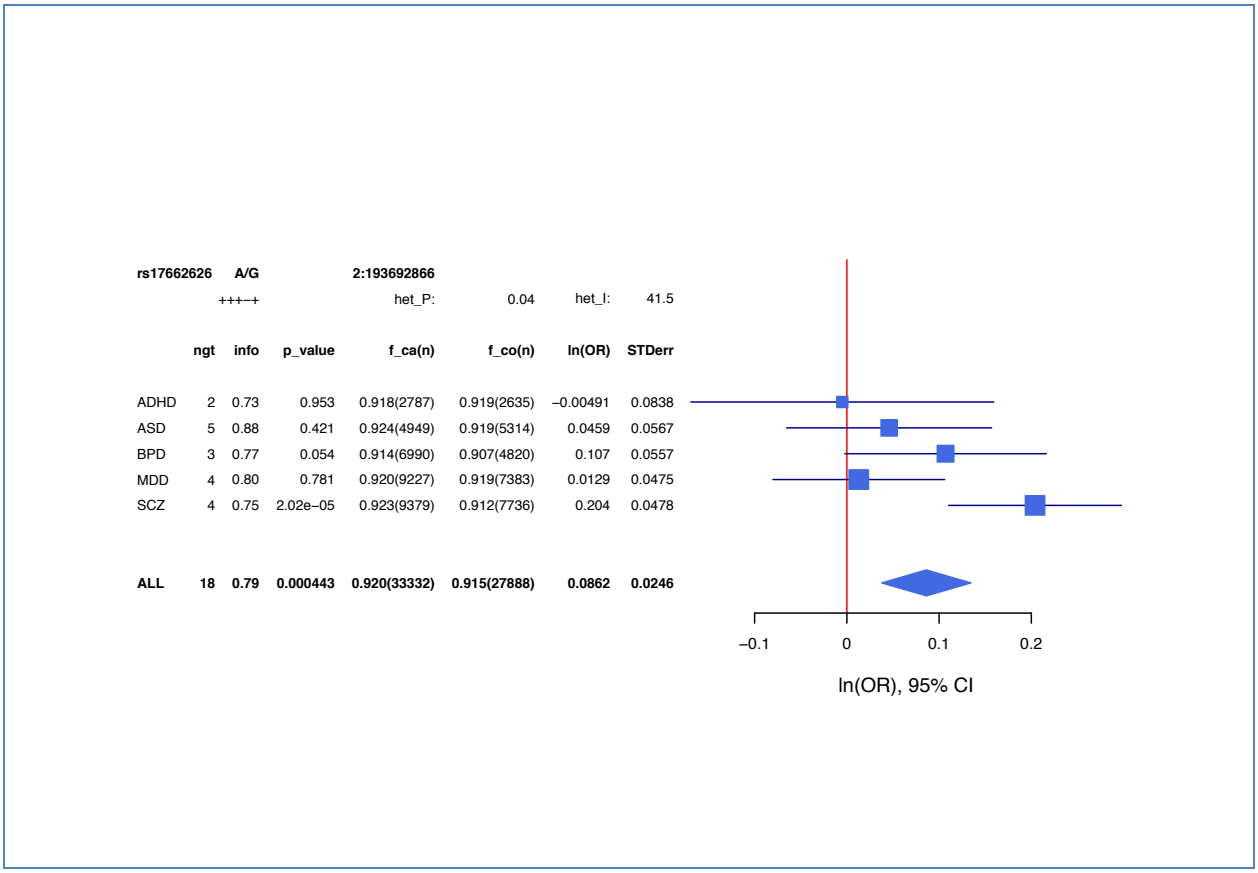

SNP: rs7004633
Best Model (BIC, AIC): Schizophrenia
60
50
40
30
20
10
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC


rs7004633 G/A 8:89829427
+−−−− het_P: 0.00 het_I: 75.5
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 2 0.96 0.521 0.807(2787) 0.812(2635) 0.0323 0.0502
ASD 6 0.96 0.917 0.810(4949) 0.813(5314) 0.00391 0.0369
BPD 3 0.92 0.304 0.821(6990) 0.817(4820) −0.0383 0.0375
MDD 6 1.00 0.535 0.812(9227) 0.816(7383) 0.0185 0.0297
SCZ 4 0.91 1.68e−08 0.796(9379) 0.819(7736) 0.173 0.0306
ALL 21 0.95 0.00227 0.809(33332) 0.816(27888) 0.048 0.0157
−0.1
−0.05
0
0.05
0.1
0.15
0.2
ln(OR), 95% CI

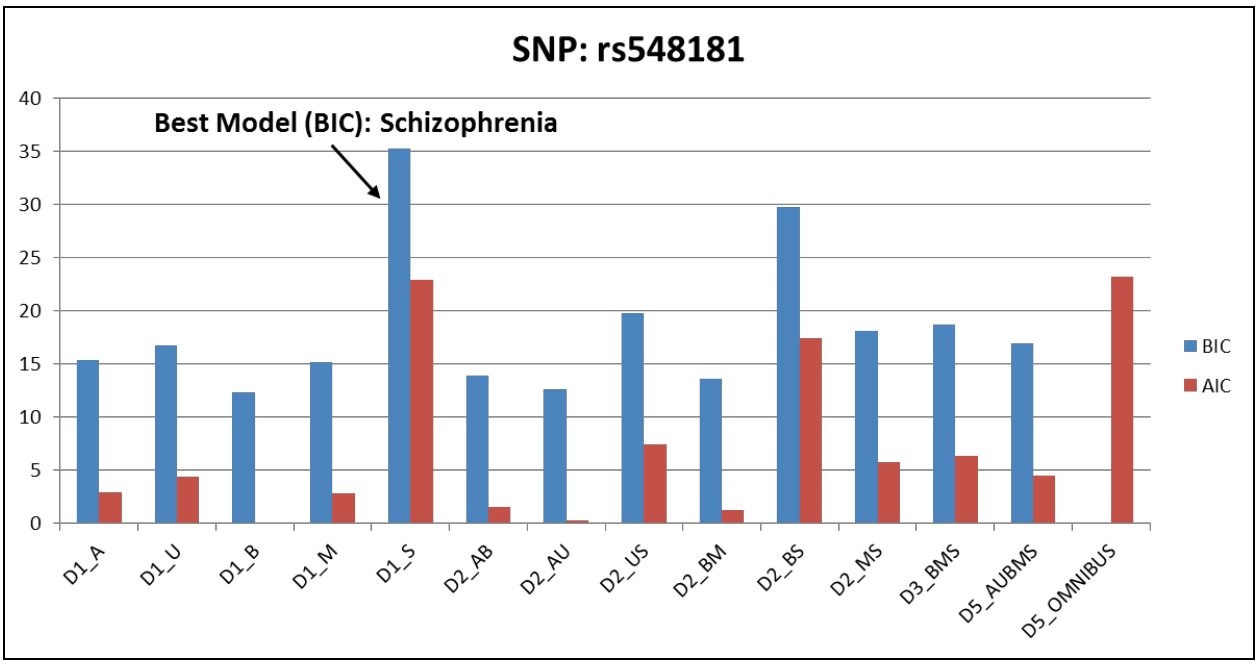

SNP: rs10503253
Best Model (BIC, AIC): Schizophrenia
50
45
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

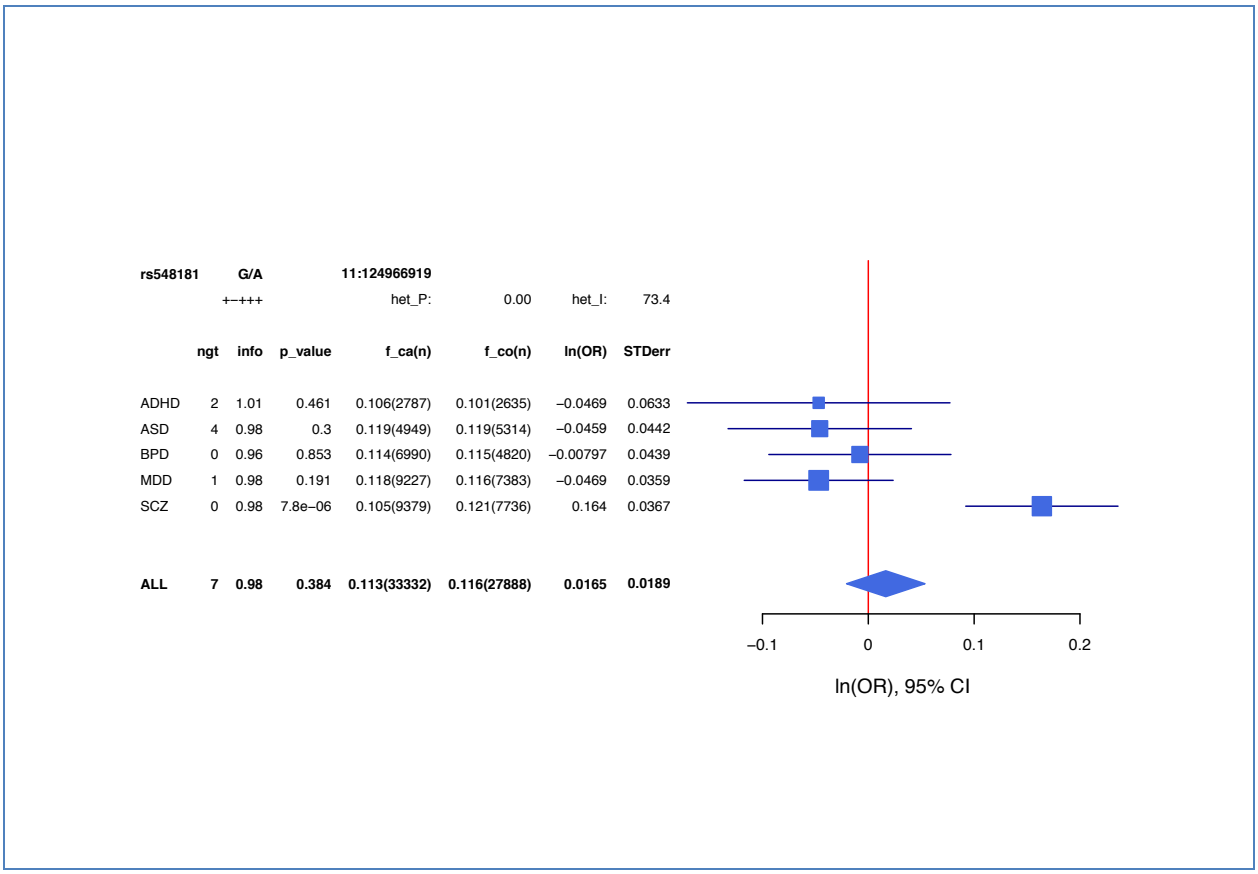

rs10503253 A/C 8:4168252
++++– het_P: 0.01 het_I: 52.3
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 1 0.96 0.958 0.185(2787) 0.185(2635) 0.003 0.0505
ASD 6 1.01 0.402 0.197(4949) 0.208(5314) –0.0294 0.0351
BPD 3 0.94 0.223 0.190(6990) 0.185(4820) 0.044 0.0365
MDD 6 0.98 0.246 0.195(9227) 0.189(7383) 0.0344 0.0296
SCZ 4 0.96 2.16e–05 0.212(9379) 0.195(7736) 0.123 0.029
ALL 20 0.97 0.00276 0.198(33332) 0.193(27888) 0.0459 0.0153
–0.05
0
0.05
0.1
0.15
ln(OR), 95% CI

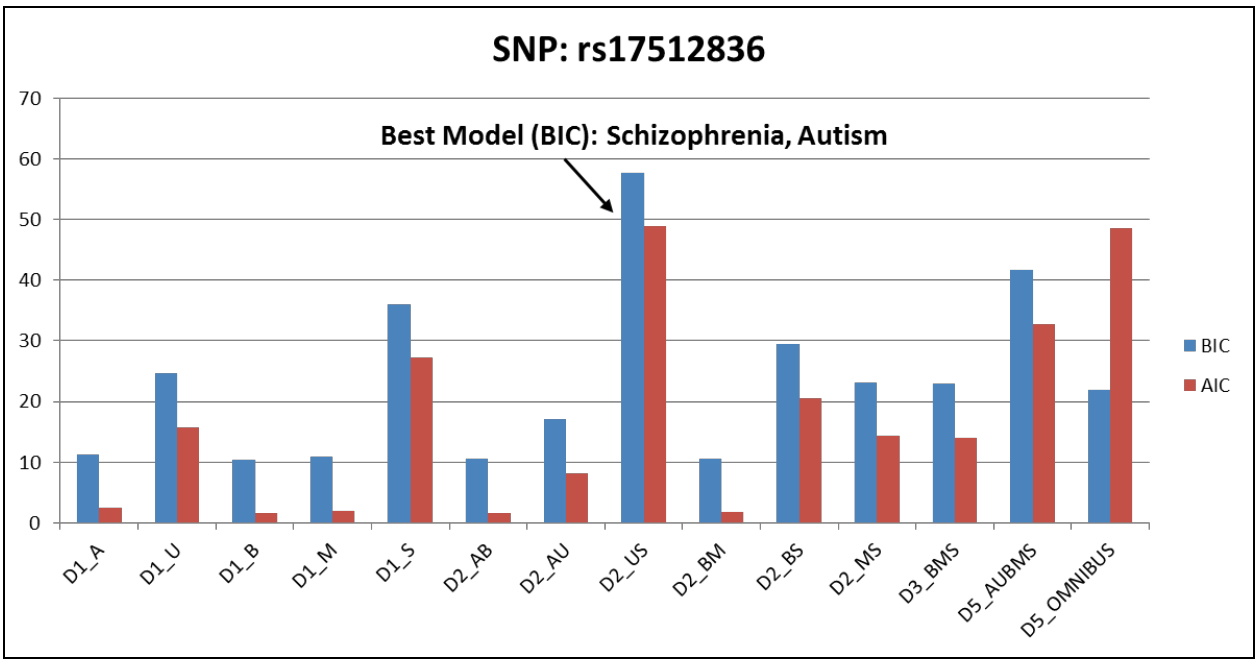


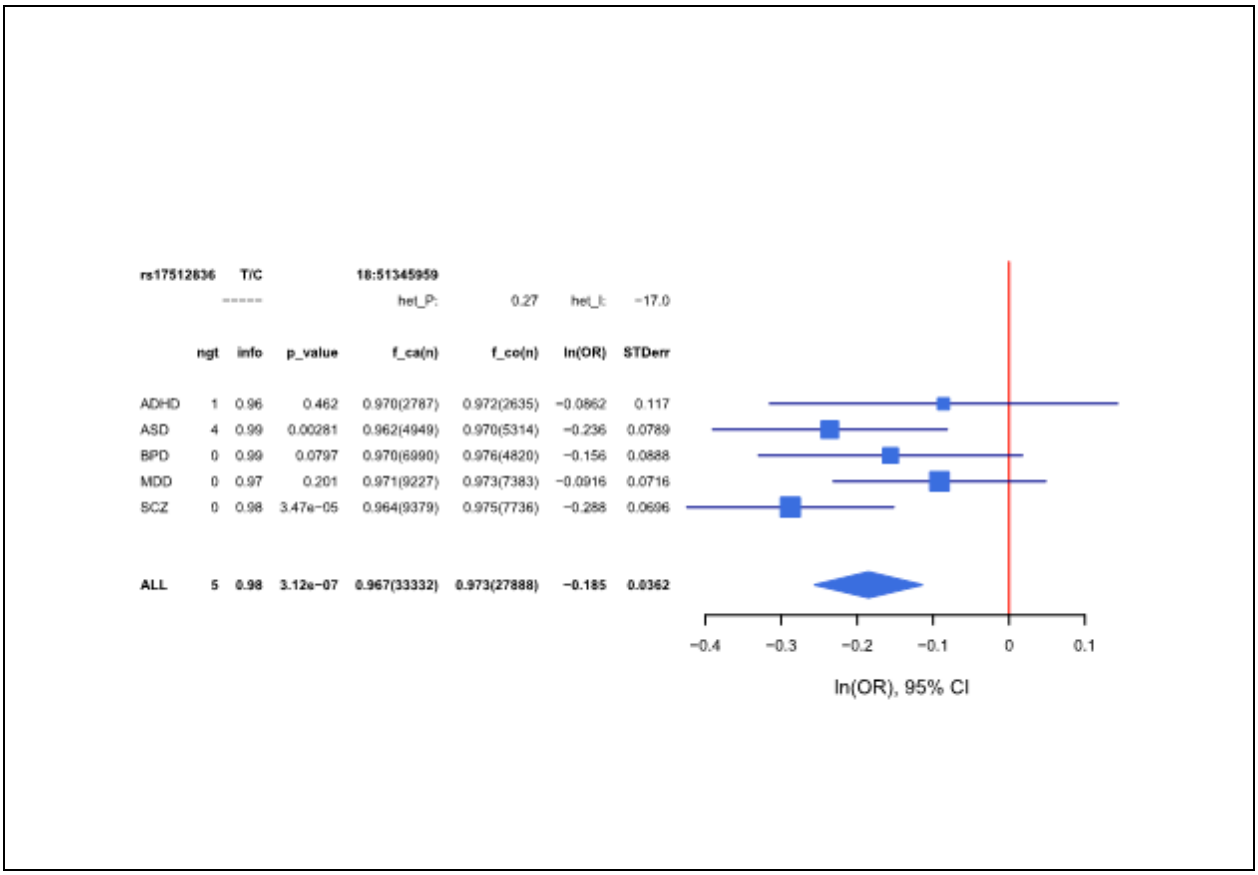


rs17662626	A/G		2:193692866			
	+++−+		het_P:	0.04	het_I:	41.5
	ngt	**info**	**p_value**	**f_ca(n)**	**f_co(n)**	**ln(OR)**	**STDerr**
ADHD	2	0.73	0.953	0.918(2787)	0.919(2635)	−0.00491	0.0838
ASD	5	0.88	0.421	0.924(4949)	0.919(5314)	0.0459	0.0567
BPD	3	0.77	0.054	0.914(6990)	0.907(4820)	0.107	0.0557
MDD	4	0.80	0.781	0.920(9227)	0.919(7383)	0.0129	0.0475
SCZ	4	0.75	2.02e−05	0.923(9379)	0.912(7736)	0.204	0.0478
ALL	**18**	**0.79**	**0.000443**	**0.920(33332)**	**0.915(27888)**	**0.0862**	**0.0246**


SNP: rs548181
Best Model (BIC): Schizophrenia
40
35
30
25
20
15
10
5
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC


rs548181 G/A 11:124966919
+−+++ het_P: 0.00 het_I: 73.4
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 2 1.01 0.461 0.106(2787) 0.101(2635) −0.0469 0.0633
ASD 4 0.98 0.3 0.119(4949) 0.119(5314) −0.0459 0.0442
BPD 0 0.96 0.853 0.114(6990) 0.115(4820) −0.00797 0.0439
MDD 1 0.98 0.191 0.118(9227) 0.116(7383) −0.0469 0.0359
SCZ 0 0.98 7.8e−06 0.105(9379) 0.121(7736) 0.164 0.0367
ALL 7 0.98 0.384 0.113(33332) 0.116(27888) 0.0165 0.0189
−0.1
0
0.1
0.2
ln(OR), 95% CI


SNP: rs17512836
Best Model (BIC): Schizophrenia, Autism
70
60
50
40
30
20
10
0
D1_A
D1_U
D1_B
D1_M
D1_S
D2_AB
D2_AU
D2_US
D2_BM
D2_BS
D2_MS
D3_BMS
D5_AUBMS
D5_OMNIBUS
BIC
AIC

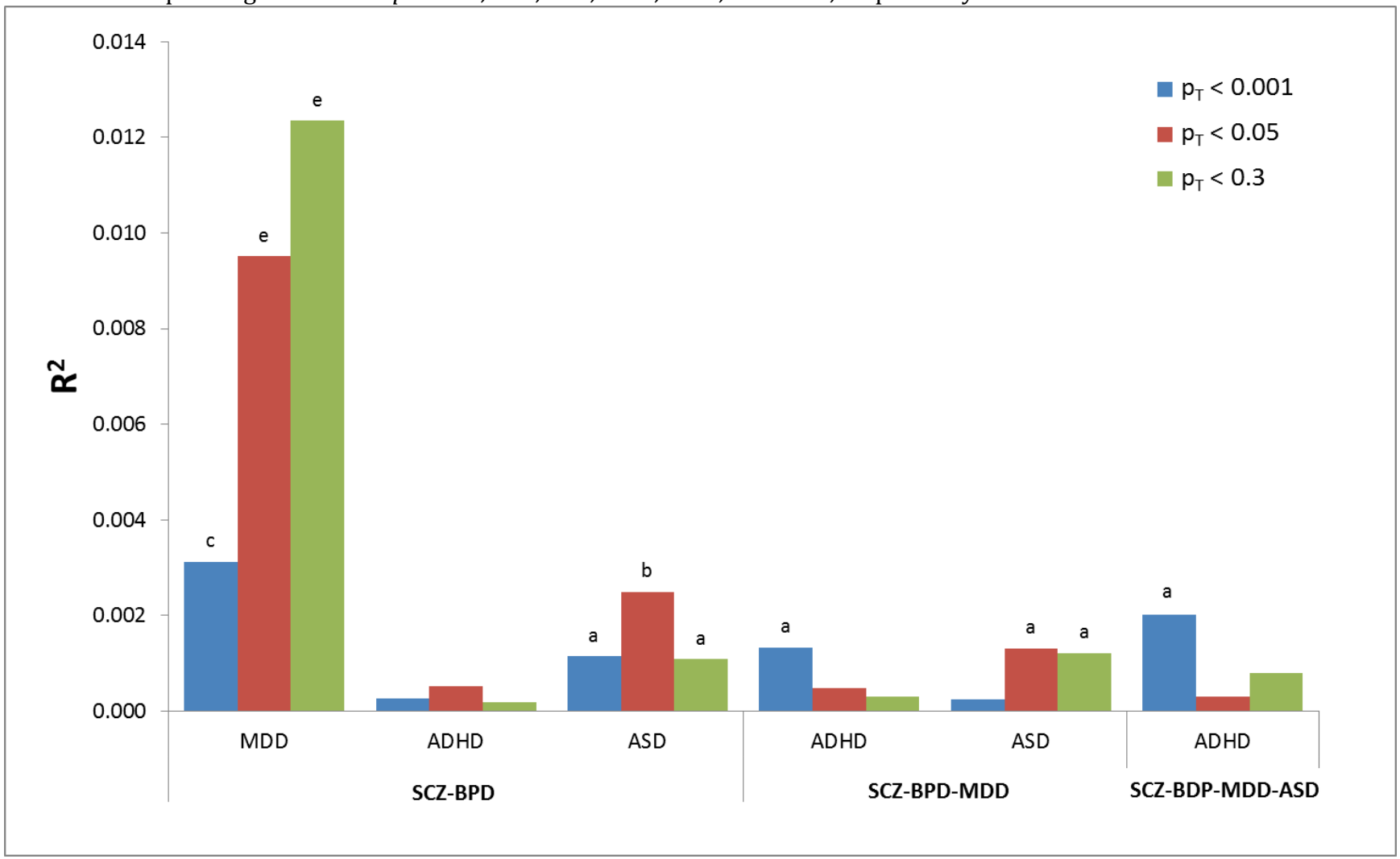

rs17512836 T/C 18:51345959
het_P: 0.27 het_I: 17.0
ngt info p_value f_ca(n) f_co(n) ln(OR) STDerr
ADHD 1 0.96 0.462 0.970(2787) 0.972(2635) −0.0862 0.117
ASD 4 0.99 0.00281 0.962(4949) 0.970(5314) −0.236 0.0789
BPD 0 0.99 0.0797 0.970(6990) 0.976(4820) −0.156 0.0888
MDD 0 0.97 0.201 0.971(9227) 0.973(7383) −0.0916 0.0716
SCZ 0 0.98 3.47e−05 0.964(9379) 0.975(7736) −0.288 0.0696
ALL 5 0.98 3.12e−07 0.967(33332) 0.973(27888) −0.185 0.0362
−0.4
−0.3
−0.2
−0.1
0
0.1
ln(OR), 95% CI

XV. Table S5: Polygenic Risk Profiling Results

	Proportion of variance explained by association P thresholds (PT)									
Disorder Pairs (Discovery_Target)	0.0001	0.001	0.01	0.05	0.1	0.2	0.3	0.4	0.5	1
ADHD_ASD	---	---	---	---	---	---	---	---	---	---
ADHD_BPD	---	---	---	---	---	---	---	---	---	---
ADHD_MDD	---	---	---	---	---	---	---	---	---	---
ADHD_SCZ	---	---	---	---	---	---	---	---	---	---
ASD_ADHD	---	---	---	---	---	---	---	---	---	---
ASD_BPD	---	---	0.00042[q]	0.00084[a]	0.00050[a]	0.00056[a]	0.00064[a]	0.00068[a]	0.00067[a]	0.00065[a]
ASD_MDD	---	0.00035[a]	0.00049[q]	---	---	---	---	---	---	---
ASD_SCZ	---	---	0.00042[q]	0.0012[b]	0.0011[a]	0.0010[a]	0.0012	0.0013	0.0013	0.0011[a]
BPD_ADHD	0.0018[a]	---	0.0017[q]	---	---	---	---	---	---	---
BPD_ASD	---	---	---	---	---	0.00073[a]	0.00069[a]	0.00077[a]	0.00068[a]	0.00063[a]
BPD_MDD	0.0010[a]	0.0010[a]	0.0030[c]	0.0039[d]	0.0041[d]	0.0039[d]	0.0042[d]	0.0047[d]	0.0048[d]	0.0049[d]
BPD_SCZ	0.0020[b]	0.0048[d]	0.012[e]	0.017[f]	0.021[f]	0.021[f]	0.022[f]	0.021[f]	0.021[f]	0.021[f]
MDD_ADHD	---	---	---	---	---	---	---	---	---	---
MDD_ASD	---	---	---	---	---	---	---	---	---	---
MDD_BPD	---	0.00054[q]	0.0019[b]	0.0040[c]	0.0051[c]	0.0056[d]	0.0056[d]	0.0061[d]	0.0062[d]	0.0.0067[d]
MDD_SCZ	---	0.00032[q]	0.0038[e]	0.0081[e]	0.0089[e]	0.0094[e]	0.0097[e]	0.010[e]	0.010[e]	0.011[e]
SCZ_ADHD	---	---	---	---	---	---	---	---	---	---
SCZ_ASD	---	0.0015[q]	---	0.00078[a]	0.00091[a]	0.00076[a]	0.00075[a]	0.00075[a]	0.00069[a]	0.00071[a]
SCZ_BPD	0.0044[c]	0.0065[d]	0.015[e]	0.023[e]	0.024[e]	0.025[f]	0.024[e]	0.024[e]	0.025[f]	0.025[f]
SCZ_MDD	0.00059[a]	0.0016[b]	0.0056[e]	0.0068[e]	0.0070[e]	0.0072[e]	0.0080[e]	0.0089[e]	0.0090[e]	0.0091[e]

Superscript letters indicate statistical significance for analysis, with a through f corresponding to values of $p < 0.05$, 10^{-4}, 10^{-8}, 10^{-12}, 10^{-16}, and 10^{-50}, respectively.

I. **XVI. Figure S8: Polygenic Risk Profiling for Combined Discovery Sets** Polygene risk scores were derived for combinations of disorders ("discovery sets") and applied sequentially to the remaining disorders ("target sets"). Results are grouped by each discovery set. The proportion of variance explained for the target disorder (estimated via Nagelkerke's pseudo R^2) is plotted on the y-axis. Color-coding denotes the gradient of *p*-value thresholds, p_T used to select training set SNPs. Lower case letters above the bars indicate statistical significance for analysis, with "a" through "f" corresponding to values of $p < 0.05$, 10^{-4}, 10^{-8}, 10^{-12}, 10^{-16}, and 10^{-50}, respectively.



XVII. Table S6: Top 50 Pathway Analysis Results

Pathway Gene No	Associated Interval No	Empirical P-Value	Corrected P-value	GO Term	GO Name
67	20	9.9999E-06	0.036593	GO:0005262	calcium channel activity
120	19	2.99997E-05	0.069186	GO:0032259	methylation
136	22	3.99996E-05	0.082983	GO:0009952	anterior/posterior pattern specification
110	18	6.99993E-05	0.135173	GO:0043414	macromolecule methylation
71	13	0.000179998	0.276545	GO:0009855	determination of bilateral symmetry
46	11	0.000189998	0.289742	GO:0000186	activation of MAPKK activity
72	13	0.000199998	0.304339	GO:0009799	specification of symmetry
168	22	0.000239998	0.346131	GO:0006730	one-carbon metabolic process
169	22	0.000239998	0.346131	GO:0008168	methyltransferase activity
175	22	0.000329997	0.431114	GO:0016741	transferase activity, transferring one-carbon groups
66	12	0.000339997	0.438912	GO:0007368	determination of left/right symmetry
8	4	0.000409996	0.4977	GO:0008239	dipeptidyl-peptidase activity
33	8	0.000469995	0.538692	GO:0001947	heart looping
33	8	0.000469995	0.538692	GO:0003143	embryonic heart tube morphogenesis
33	8	0.000469995	0.538692	GO:0061371	determination of heart left/right asymmetry
88	14	0.000489995	0.55169	GO:0008757	S-adenosylmethionine-dependent methyltransferase activity
192	21	0.000529995	0.577684	GO:0010498	proteasomal protein catabolic process
41	9	0.000579994	0.606479	GO:0042054	histone methyltransferase activity
108	18	0.000679993	0.654669	GO:0048736	appendage development

108	18	0.000679993	0.654669	GO:0060173	limb development
38	8	0.000699993	0.663467	GO:0043021	ribonucleoprotein complex binding
48	9	0.000749993	0.688462	GO:0061136	regulation of proteasomal protein catabolic process
8	4	0.000789992	0.707259	GO:0034123	positive regulation of toll-like receptor signaling pathway
102	17	0.00096999	0.774845	GO:0035107	appendage morphogenesis
102	17	0.00096999	0.774845	GO:0035108	limb morphogenesis
66	11	0.00097999	0.779444	GO:0032526	response to retinoic acid
72	12	0.00103999	0.79784	GO:0034399	nuclear periphery
172	26	0.00108999	0.810238	GO:0005244	voltage-gated ion channel activity
172	26	0.00108999	0.810238	GO:0022832	voltage-gated channel activity
75	12	0.00110999	0.814837	GO:0006479	protein methylation
75	12	0.00110999	0.814837	GO:0008213	protein alkylation
187	20	0.00118999	0.833633	GO:0043161	proteasomal ubiquitin-dependent protein catabolic process
56	10	0.00131999	0.861628	GO:0016571	histone methylation
65	11	0.00131999	0.861628	GO:0016363	nuclear matrix
25	6	0.00148999	0.888022	GO:0008173	RNA methyltransferase activity
5	3	0.00151998	0.893821	GO:0032988	ribonucleoprotein complex disassembly
17	5	0.00171998	0.917217	GO:0043022	ribosome binding
5	3	0.00176998	0.921816	GO:0035197	siRNA binding
167	21	0.00179998	0.925815	GO:0060562	epithelial tube morphogenesis
80	12	0.00183998	0.930414	GO:0033189	response to vitamin A
57	10	0.00202998	0.944611	GO:0008276	protein methyltransferase activity
12	4	0.00208998	0.94981	GO:0051205	protein insertion into membrane
18	5	0.00224998	0.957409	GO:0031519	PcG protein complex
41	8	0.00226998	0.957808	GO:0034968	histone lysine methylation
12	4	0.00245998	0.968606	GO:0001510	RNA methylation

124	16	0.00283997	0.979404	GO:0016607	nuclear speck
132	19	0.00300997	0.984003	GO:0030031	cell projection assembly
137	16	0.00300997	0.984003	GO:0015931	nucleobase-containing compound transport
32	8	0.00309997	0.984803	GO:0051925	regulation of calcium ion transport via voltage-gated calcium channel activity

Pathway Gene No: number of human reference hg18 genes annotated by a corresponding GO term; **Associated Gene No**: number of LD-independent association intervals that intersect with at least one reference gene annotated by a corresponding GO term; **Empirical P-Value**: gene set enrichment *p* value assessed by a random interval-based permutation procedure; **Corrected P-Value**: gene set enrichment *p* value adjusted by multiple testing of 6,600 GO terms; **GO Term**: Gene Ontology Accession identifier; **GO Name**: Name of GO Term.

XVIII. Table S7: Calcium Channel Activity Gene Set Results

Gene Symbol	Genic Region	Index SNP	Index SNP P	Gene Name
CACNA2D4, CACNA1C	chr12:1891122..2817115	rs1024582	1.87E-08	calcium channel, voltage-dependent, alpha 2/delta subunit 4; calcium channel, voltage-dependent, L type, alpha 1C subunit;
CACNB2	chr10:18394605..18840798	rs2799573	4.29E-08	calcium channel, voltage-dependent, beta 2 subunit
CACNA1S	chr1:200998641..201116694	rs2297909	2.27E-06	calcium channel, voltage-dependent, L type, alpha 1S subunit
GRIN2A	chr16:9837260..10311611	rs8058295	7.12E-06	glutamate receptor, ionotropic, N-methyl D-aspartate 2A
CALHM1	chr10:105203143..105253645	rs11191732	8.65E-06	calcium homeostasis modulator 1
CACNA1D	chr3:53493682..53856490	rs893363	1.91E-05	calcium channel, voltage-dependent, L type, alpha 1D subunit
ITPR2	chr12:26480341..27021131	rs12815170	2.37E-05	inositol 1,4,5-triphosphate receptor, type 2

TRPM3	chr9:73139948..74096820	rs4745072	7.82E-05	transient receptor potential cation channel, subfamily M, member 3
GPM6A	chr4:176544084..176958815	rs1021226	0.0002478	glycoprotein M6A
CACNA1E	chr1:181347237..181787219	rs577528	0.0002697	calcium channel, voltage-dependent, R type, alpha 1E subunit
CATSPER4	chr1:26482051..26539459	rs1335762	0.0002714	cation channel, sperm associated 4
TMEM37	chr2:120152476..120206096	rs7578714	0.0003744	transmembrane protein 37
GRIN3A	chr9:104321634..104535862	rs7047153	0.0003822	glutamate receptor, ionotropic, N-methyl-D-aspartate 3A
TPCN2	chr11:68781349..68868072	rs679596	0.000407	two pore segment channel 2
TRPC4	chr13:38200772..38479562	rs2008441	0.000545	transient receptor potential cation channel, subfamily C, member 4
ITPR1	chr3:4500031..4899524	rs11708874	0.0006516	inositol 1,4,5-triphosphate receptor, type 1
CATSPER2	chr15:43910700..43995316	rs694461	0.0007349	cation channel, sperm associated 2
CATSPER1	chr11:65774222..65828988	rs539046	0.0007813	cation channel, sperm associated 1
PSEN1	chr14:73568154..73697109	rs7142086	0.0008547	presenilin 1
CACNA2D2	chr3:50390232..50576675	rs2526752	0.0008854	calcium channel, voltage-dependent, alpha 2/delta subunit 2

Gene Symbol: HUGO Gene Nomenclature Committee (HGNC) gene symbol; **Genic Region:** UCSD Genome Browser hg.18-based chromosomal locus of each gene (including 35kb upstream& 10kb downstream of transcription starting & ending position); **Index SNP:** SNP identifier with the most significant meta analysis *p* value within the corresponding genic region; **Index SNP P**: meta analysis *p* value of the index SNP.

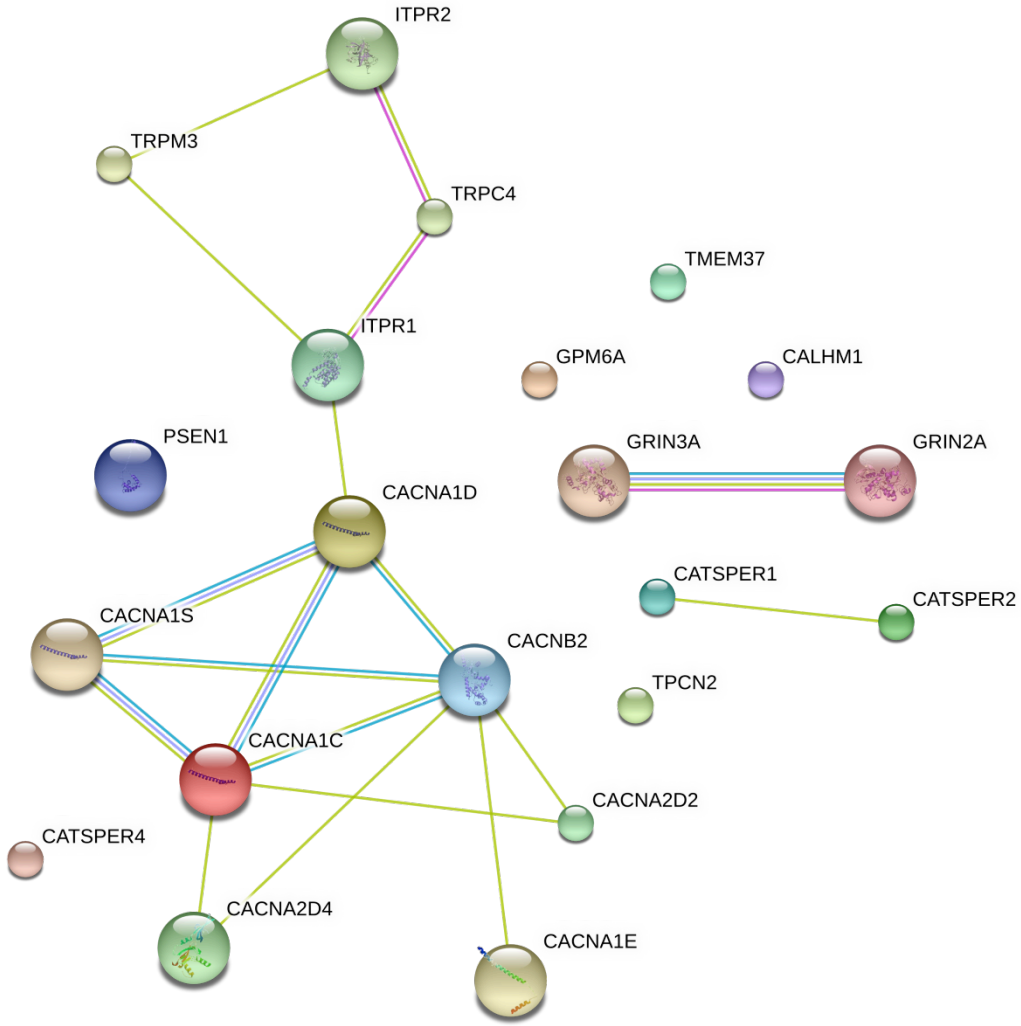
XIX. Table S8. List of 73 calcium channel activity genes that are analyzed as 67 gene segments in the INRICH pathway analysis (http:atgu.mgh.harvard.edu/inrich).

ENSEMBL ID	Chromosome	Start BP	End BP	Symbol	Pathway Analysis
ENSG00000188782	1	26354638	26412046	CATSPER4	included
ENSG00000198216	1	179613860	180053842	CACNA1E	included
ENSG00000081248	1	199265264	199383317	CACNA1S	included
ENSG00000198626	1	235237127	236205982	RYR2	included
ENSG00000171227	2	119868946	119922566	TMEM37	included
ENSG00000135914	2	231671187	231733076	HTR2B	included
ENSG00000144481	2	234455781	234602905	TRPM8	included
ENSG00000150995	3	4475031	4874524	ITPR1	included
ENSG00000007402	3	50365236	50551679	CACNA2D2	included
ENSG00000157388	3	53468722	53831530	CACNA1D	included
ENSG00000157445	3	54096613	55093624	CACNA2D3	included
ENSG00000144935	3	143890605	144019420	TRPC1	included
ENSG00000174343	4	39997102	40061991	CHRNA9	included
ENSG00000118762	4	89112843	89227953	PKD2	included
ENSG00000138741	4	123009631	123127359	TRPC3	included
ENSG00000138685	4	123932312	124048841	FGF2	included
ENSG00000150625	4	176781078	177195809	GPM6A	included
ENSG00000152705	5	134296494	134385291	CATSPER3	included
ENSG00000069018	5	135566897	135764124	TRPC7	included
ENSG00000096433	6	33661499	33782329	ITPR3	included
ENSG00000165125	7	142269077	142328629	TRPV6	included
ENSG00000127412	7	142305388	142376027	TRPV5	included
ENSG00000104321	8	73084705	73185406	TRPA1	included
ENSG00000155886	9	19495977	19811926	SLC24A2	included
ENSG00000083067	9	72329768	73286640	TRPM3	included

ENSG00000119121	9	76517230	76727830	TRPM6	included
ENSG00000198785	9	103361455	103575683	GRIN3A	included
ENSG00000148408	9	139857061	140148897	CACNA1B	included
ENSG00000165995	10	18434611	18880804	CACNB2	included
ENSG00000185933	10	105193133	105243635	CALHM1	included
ENSG00000129749	11	3633392	3684190	CHRNA10	included
ENSG00000149534	11	59577309	59630020	MS4A2	included
ENSG00000175294	11	65530798	65585564	CATSPER1	included
ENSG00000162341	11	68537925	68624648	TPCN2	included
ENSG00000110218	11	93466741	93564786	PANX1	included
ENSG00000166266	11	107349668	107493713	CUL5	included
ENSG00000151062	12	1761383	1933263	CACNA2D4	included as a combined
ENSG00000151067	12	1915212	2687376	CACNA1C	
ENSG00000150086	12	13595410	14059320	GRIN2B	included
ENSG00000123104	12	26371608	26912398	ITPR2	included
ENSG00000111199	12	108695272	108790595	TRPV4	included
ENSG00000186815	12	112108237	112230773	TPCN1	included
ENSG00000182500	12	120513837	120574966	ORAI1	included
ENSG00000133107	13	37098772	37377562	TRPC4	included
ENSG00000185989	13	113755295	113951188	RASA3	included
ENSG00000080815	14	72637907	72766862	PSEN1	included
ENSG00000134160	15	29070555	29275768	TRPM1	included
ENSG00000198838	15	31355468	31955591	RYR3	included
ENSG00000166762	15	41697992	41782608	CATSPER2	included
ENSG00000092439	15	48626642	48801304	TRPM7	included
ENSG00000196557	16	1108241	1221772	CACNA1H	included
ENSG00000183454	16	9744761	10219112	GRIN2A	included
ENSG00000006116	16	24139374	24291623	CACNG3	included
ENSG00000167723	17	3350545	3443039	TRPV3	included
ENSG00000196689	17	3405492	3482141	TRPV1	included
ENSG00000108405	17	3736639	3801709	P2RX1	included

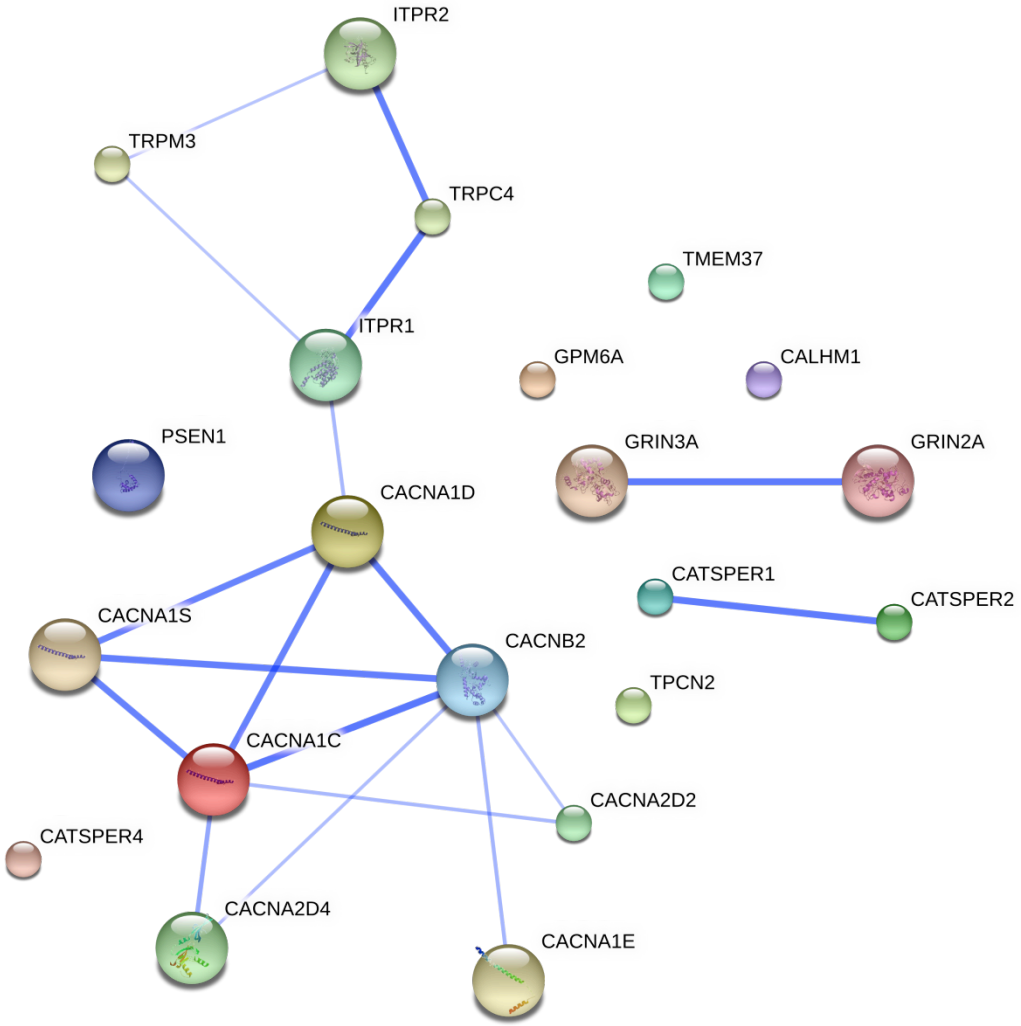
ENSG00000187688	17	16224612	16291042	TRPV2	included
ENSG00000067191	17	34573234	34642428	CACNB1	included
ENSG00000006283	17	45958427	46069834	CACNA1G	included
ENSG00000075429	17	62226696	62322065	CACNG5	included
ENSG00000075461	17	62356474	62469980	CACNG4	included as a combined
ENSG00000108878	17	62436167	62493375	CACNG1	
ENSG00000141837	19	13168255	13513274	CACNA1A	included
ENSG00000196218	19	43581179	43780044	RYR1	included
ENSG00000130529	19	54317863	54416903	TRPM4	included
ENSG00000105605	19	59069400	59149007	CACNG7	included as a combined
ENSG00000142408	19	59123105	59195281	CACNG8	
ENSG00000130433	19	59152353	59217735	CACNG6	
ENSG00000142185	21	44559473	44697392	TRPM2	included
ENSG00000166862	22	35279913	35464549	CACNG2	included
ENSG00000100346	22	38261703	38425688	CACNA1I	included
ENSG00000102001	X	48938466	49011777	CACNA1F	excluded
ENSG00000072315	X	110894198	111247660	TRPC5	excluded

Ensembl ID: Ensembl gene identifier; **Start BP**: starting base pair position of genic region including the 35kb upstream from transcription starting sites; **End BP**: ending base pair position of genic region including the 10kb downstream from transcription ending sites; **Symbol**: HGNC gene symbol; **Pathway Analysis**: to indicate whether the gene was included/excluded in the INRICH analysis.



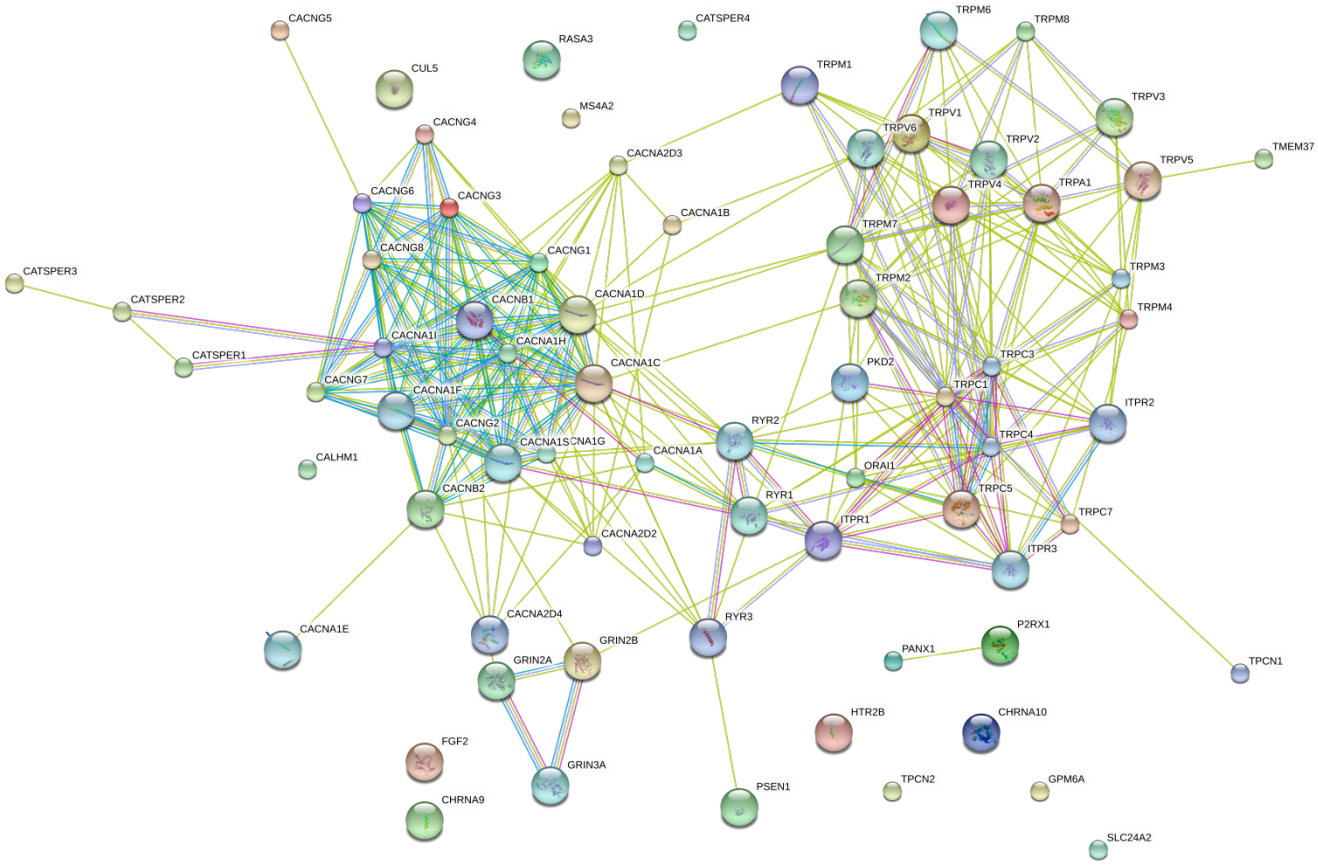
XX. Figure S9. Functional relationship between 21 calcium channel activity genes that showed significant association with 5 disorders at $p < 10^{-3}$ in the primary meta analysis (http://string-db.org)[13]. The network view summarizes the network of predicted associations for a particular group of proteins. The network nodes are proteins. The edges represent the predicted functional associations, supported by the existence of the seven types of evidence

used in predicting the associations. A red line indicates the presence of fusion evidence; a green line - neighborhood evidence; a blue line - coocurrence evidence; a purple line - experimental evidence; a yellow line - textmining evidence; a light blue line - database evidence; a black line - coexpression evidence. Detailed functional evidence is summarized in Table S9.



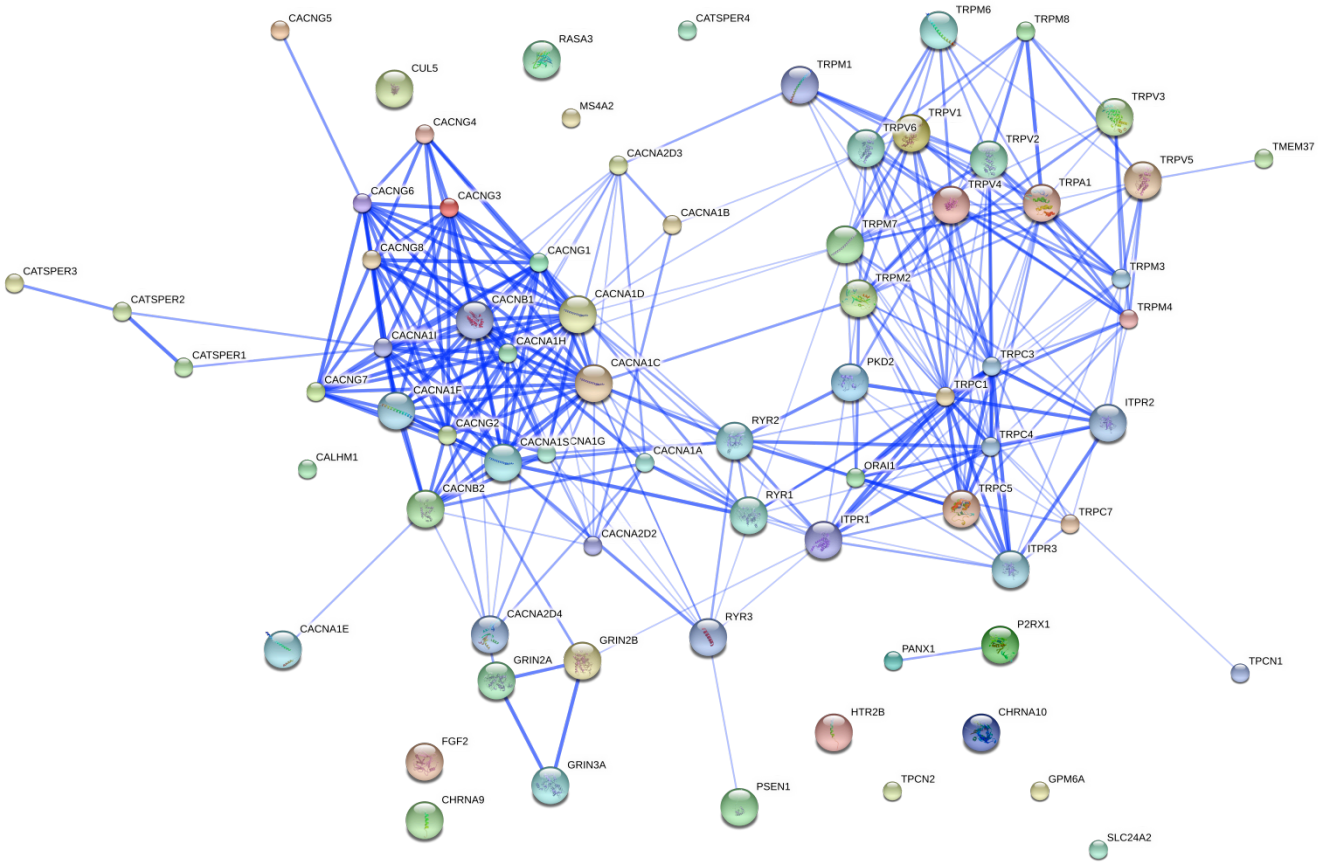
XXI. Figure S10. Functional relationship between 21 calcium channel activity genes that showed significant association with 5 disorders at $p < 10^{-3}$ in the primary meta analysis (http://string-db.org)[13]. The network view summarizes the network of predicted associations for a particular group of proteins. The network nodes are proteins. The edges represent the predicted functional associations, supported by the existence of the seven types of evidence

used in predicting the associations. Edge thickness represents the predicted functional score between protein pairs. Detailed functional evidence and scores are summarized in Table S9.



XXII. Figure S11. **Functional relationships between 73 calcium channel activity genes defined by Gene Ontology (http://amigo.geneontology.org/cgi-bin/amigo/term_details?term=GO:0005262) .** The network view from the STRING database (http://string-db.org)[13] summarizes the network of predicted associations for a particular group of proteins. The network nodes are proteins. The edges represent the predicted functional associations, supported by the existence of the seven types of evidence used in predicting the associations. A red line indicates the presence of fusion

evidence; a green line - neighborhood evidence; a blue line – co-ocurrence evidence; a purple line - experimental evidence; a yellow line – text mining evidence; a light blue line - database evidence; a black line – co-expression evidence. Detailed functional evidence and scores are summarized in Table S10.



XXIII. Figure S12. Functional relationships between 73 calcium channel activity genes defined by Gene Ontology (http://amigo.geneontology.org/cgi-bin/amigo/term_details?term=GO:0005262). The network view from the STRING database (http://string-db.org)[13] summarizes the network of predicted associations for a particular group of proteins. The network nodes are proteins. The edges represent the predicted functional associations, supported by the existence of the seven types of evidence used in predicting the associations. Edge thickness represents the predicted functional score between protein pairs. Detailed functional evidence and scores are summarized in Table S10.

XXIV. Table S9. Detailed functional association score from the STRING database (http://string-db.org/) [13] between 21 calcium channel activity genes that showed association with 5 disorders at $p < 10^{-3}$ in the primary meta analysis.

Node1	Node2	Homology Score	Experimental Score	Knowledge Score	Text Mining Score	Combined Score
CACNB2	CACNA1C	0	0	0.9	0.86	0.985
ITPR1	TRPC4	0	0.821	0	0.81	0.963
GRIN3A	GRIN2A	0.758	0.538	0.9	0.879	0.961
CATSPER1	CATSPER2	0	0	0	0.954	0.954
CACNB2	CACNA1D	0	0	0.9	0.362	0.931
CACNA1S	CACNB2	0	0	0.9	0.33	0.928
ITPR2	TRPC4	0	0.62	0	0.81	0.923
CACNA1D	CACNA1C	0.955	0	0.9	0.804	0.903
CACNA1S	CACNA1C	0.953	0	0.9	0.747	0.903
CACNA1S	CACNA1D	0.952	0	0.9	0.602	0.902
CACNA2D4	CACNA1C	0	0	0	0.643	0.643
CACNA1E	CACNB2	0	0	0	0.539	0.539
ITPR1	CACNA1D	0	0	0	0.522	0.522
CACNA2D2	CACNA1C	0	0	0	0.515	0.515
CACNA2D4	CACNB2	0	0	0	0.475	0.475
CACNA2D2	CACNB2	0	0	0	0.444	0.443
ITPR1	TRPM3	0	0	0	0.433	0.433
ITPR2	TRPM3	0	0	0	0.433	0.433

Homology Score: calculated based on the degree of homology of the interactors. **Experimental Score**: based on a list of significant protein interaction datasets, gathered from other protein-protein interaction databases: BIND, DIP, GRID, HPRD, IntAct, MINT, and PID; **Knowledge Score**: based on pathway and functional annotation databases: Biocarta, BioCyc, GO, KEGG, and Reactome; **Text Mining Score**: based on a list of significant protein interaction groups, extracted from the abstracts of scientific literature; **Combined Score**: computed by combining the probabilities from the different evidence channels, correcting for the probability of randomly observing an interaction. For a more detailed description refer to von Mering C et al.[14]

XXV. Table S10. Detailed functional association score from the STRING database[13] for 73 calcium channel activity genes (GO:0005262)

Node1	Node2	Homology Score	Experimental Score	Knowledge Score	Text Mining Score	Combined Score
CACNG8	CACNG6	0	0	0.9	0.956	0.995
CACNG8	CACNG1	0	0	0.9	0.956	0.995
CACNG6	CACNG7	0	0	0.9	0.954	0.995
CACNG1	CACNG3	0	0	0.9	0.96	0.995
TRPC1	TRPC5	0.921	0.913	0.9	0.993	0.991
TRPC3	TRPC1	0.872	0.913	0.9	0.993	0.991
TRPC3	ITPR3	0	0.925	0	0.878	0.99
CACNB1	CACNA1D	0	0	0.9	0.871	0.986
CACNA1D	CACNG1	0	0	0.9	0.873	0.986
TRPC1	PKD2	0	0.62	0	0.964	0.985
CACNB2	CACNA1C	0	0	0.9	0.86	0.985
CACNA1D	CACNG8	0	0	0.9	0.856	0.984
CACNA1D	CACNG7	0	0	0.9	0.856	0.984
CACNB1	CACNA1S	0	0	0.9	0.834	0.982
CACNA1S	RYR1	0	0.62	0	0.953	0.981
CACNA1D	CACNG6	0	0	0.9	0.822	0.981
CACNA1S	CACNG1	0	0	0.9	0.812	0.979
ORAI1	TRPC1	0	0	0	0.979	0.979
TRPM8	TRPA1	0	0	0	0.979	0.979
CACNA1D	CACNG3	0	0	0.9	0.811	0.979
TRPC4	TRPC1	0.922	0.785	0.9	0.967	0.978
ITPR3	TRPC1	0	0.816	0	0.885	0.977
ITPR1	TRPC3	0	0.821	0	0.878	0.976
CACNG6	CACNG3	0	0	0.9	0.732	0.971
CACNB1	CACNG1	0	0	0	0.965	0.965
ITPR1	TRPC4	0	0.821	0	0.81	0.963

TRPC4	ITPR3	0	0.816	0	0.81	0.962
GRIN3A	GRIN2A	0.758	0.538	0.9	0.879	0.961
TRPC3	ORAI1	0	0.62	0	0.905	0.961
GRIN3A	GRIN2B	0.758	0.538	0.9	0.867	0.96
CACNG4	CACNG1	0	0	0	0.96	0.96
TRPC3	TRPC5	0.882	0.552	0.9	0.961	0.957
TRPC4	TRPC3	0.88	0.552	0.9	0.993	0.957
ITPR2	TRPC1	0	0.62	0	0.895	0.957
TRPC4	TRPC5	0.955	0.552	0.9	0.964	0.954
CATSPER1	CATSPER2	0	0	0	0.954	0.954
ITPR1	TRPC1	0	0.559	0	0.879	0.943
CACNB2	CACNA1D	0	0	0.9	0.362	0.931
CACNG1	CACNG7	0.669	0	0.9	0.953	0.931
CACNA1F	CACNB2	0	0	0.9	0.344	0.93
ITPR2	TRPC3	0	0	0	0.929	0.929
CACNA1G	CACNA1C	0.654	0	0.9	0.84	0.928
CACNA1S	CACNB2	0	0	0.9	0.33	0.928
CACNB1	CACNA1C	0	0	0.9	0.319	0.927
CACNA1G	CACNA1D	0.662	0	0.9	0.835	0.927
CACNA1H	CACNA1C	0.664	0	0.9	0.777	0.925
ITPR2	TRPC4	0	0.62	0	0.81	0.923
CACNA1H	CACNA1D	0.68	0	0.9	0.648	0.919
CACNG8	CACNG7	0.805	0	0.9	0.954	0.918
CACNB1	CACNA1A	0	0.62	0	0.792	0.916
CACNG7	CACNG3	0.812	0	0.9	0.85	0.915
CACNA1I	CACNA1C	0.706	0	0.9	0.522	0.914
CACNG2	CACNA1D	0	0	0.9	0.18	0.912
CACNA1C	CACNG1	0	0	0.9	0.175	0.912
CACNA1I	CACNG1	0	0	0.9	0.167	0.911
CACNB1	CACNA1G	0	0	0.9	0.173	0.911
CACNB1	CACNA1H	0	0	0.9	0.171	0.911

CACNA1I	CACNB1	0	0	0.9	0.166	0.911
CACNA1H	CACNG1	0	0	0.9	0.172	0.911
CACNG2	CACNG1	0	0	0.9	0.161	0.91
CACNA1F	CACNG1	0	0	0.9	0.159	0.91
CACNB1	CACNA1F	0	0	0.9	0.158	0.91
CACNG6	CACNG1	0.902	0	0.9	0.945	0.909
CACNA1G	CACNB2	0	0	0.9	0.147	0.908
CACNA1S	CACNG3	0	0	0.9	0.138	0.908
CACNA1C	CACNG3	0	0	0.9	0.138	0.908
CACNA1C	CACNG7	0	0	0.9	0.138	0.908
CACNA1S	CACNG8	0	0	0.9	0.138	0.908
CACNA1S	CACNG7	0	0	0.9	0.138	0.908
CACNG8	CACNA1C	0	0	0.9	0.138	0.908
CACNA1S	CACNA1G	0.693	0	0.9	0.33	0.908
CACNA1H	CACNG7	0	0	0.9	0.135	0.907
CACNA1H	CACNG8	0	0	0.9	0.135	0.907
CACNA1I	CACNG8	0	0	0.9	0.129	0.907
CACNA1S	CACNA1H	0.698	0	0.9	0.305	0.907
CACNA1I	CACNG7	0	0	0.9	0.129	0.907
CACNA1I	CACNG3	0	0	0.9	0.129	0.907
CACNA1H	CACNG3	0	0	0.9	0.135	0.907
CACNA1I	CACNA1G	0.931	0	0.9	0.96	0.906
CACNA1I	CACNA1H	0.935	0	0.9	0.962	0.906
CACNA1F	CACNG8	0	0	0.9	0.122	0.906
GRIN2A	GRIN2B	0.939	0	0.9	0.994	0.906
CACNA1F	CACNG3	0	0	0.9	0.122	0.906
CACNA1G	CACNA1H	0.935	0	0.9	0.958	0.906
CACNA1F	CACNG7	0	0	0.9	0.122	0.906
CACNA1C	CACNG6	0	0	0.9	0.127	0.906
CACNG2	CACNA1C	0	0	0.9	0.127	0.906
CACNG4	CACNG3	0.947	0	0.9	0.957	0.905

CACNA1S	CACNG6	0	0	0.9	0.114	0.905
CACNG8	CACNG3	0.939	0	0.9	0.857	0.905
CACNA1H	CACNG6	0	0	0.9	0.111	0.905
CACNA1H	CACNG2	0	0	0.9	0.105	0.904
ITPR2	ITPR3	0.956	0	0.9	0.94	0.904
CACNB1	CACNB2	0.951	0	0.9	0.867	0.904
CACNA1I	CACNG6	0	0	0.9	0.105	0.904
CACNG8	CACNG4	0.947	0	0.9	0.929	0.904
CACNA1S	CACNG2	0	0	0.9	0.108	0.904
CACNA1H	CACNB2	0	0	0.9	0.105	0.904
CACNA1F	CACNA1C	0.948	0	0.9	0.673	0.903
CACNA1I	CACNA1D	0.699	0	0.9	0.18	0.903
CACNA1I	CACNG2	0	0	0.9	0.099	0.903
CACNA1D	CACNA1C	0.955	0	0.9	0.804	0.903
CACNA1I	CACNB2	0	0	0.9	0.099	0.903
CACNA1F	CACNG6	0	0	0.9	0.097	0.903
CACNA1S	CACNA1C	0.953	0	0.9	0.747	0.903
CACNA1F	CACNA1D	0.953	0	0.9	0.837	0.903
CACNA1F	CACNA1G	0.691	0	0.9	0.147	0.902
CACNA1S	CACNA1D	0.952	0	0.9	0.602	0.902
CACNA1F	CACNG2	0	0	0.9	0.091	0.902
CACNA1F	CACNA1H	0.705	0	0.9	0.105	0.901
CACNA1I	CACNA1F	0.731	0	0.9	0.099	0.901
CACNA1I	CACNA1S	0.741	0	0.9	0.108	0.901
CACNA1F	CACNA1S	0.946	0	0.9	0.317	0.901
CACNG2	CACNG8	0.941	0	0.9	0.095	0.9
CACNG2	CACNG6	0	0	0.9	0.07	0.9
CACNG2	CACNG7	0.818	0	0.9	0.095	0.9
CACNG2	CACNG3	0.963	0	0.9	0.095	0.9
CACNG2	CACNG4	0.949	0	0.9	0.095	0.9
RYR2	TRPC5	0	0	0.9	0.016	0.899

TRPC4	RYR2	0	0	0.9	0.037	0.899
RYR2	CACNA1C	0	0.62	0	0.727	0.889
CACNA1A	RYR1	0	0	0.8	0.434	0.879
CACNG4	CACNG6	0	0	0	0.861	0.861
RYR2	PKD2	0	0	0	0.858	0.858
CACNA1D	CACNG4	0	0	0	0.856	0.856
TRPC1	TRPM4	0	0	0	0.854	0.854
CACNB1	CACNG4	0	0	0	0.851	0.851
CACNB1	CACNG7	0	0	0	0.851	0.851
CACNB1	CACNG8	0	0	0	0.851	0.851
CACNB1	CACNG3	0	0	0	0.851	0.851
TRPC4	ORAI1	0	0	0	0.847	0.847
TRPM2	TRPV1	0	0	0	0.833	0.833
ORAI1	TRPC5	0	0	0	0.83	0.83
TRPV2	TRPM8	0	0	0	0.828	0.828
TRPC3	TRPV1	0	0	0	0.827	0.827
RYR2	CACNA1S	0	0	0	0.822	0.822
CACNB1	CACNG6	0	0	0	0.819	0.819
RYR3	CACNA1S	0	0	0	0.815	0.815
CACNA1A	CACNB2	0	0	0	0.796	0.796
CACNA2D2	CACNA1A	0	0	0	0.788	0.788
TRPV2	TRPM4	0	0	0	0.787	0.787
TRPV4	TRPM4	0	0	0	0.784	0.784
TRPC1	TRPV4	0	0	0	0.785	0.784
TRPC3	TRPV4	0	0	0	0.782	0.782
TRPM1	TRPV1	0	0	0	0.781	0.781
TRPM4	TRPV1	0	0	0	0.78	0.78
ITPR1	RYR2	0.449	0.62	0	0.78	0.78
TRPC1	TRPV1	0	0	0	0.78	0.78
TRPV2	TRPM7	0	0	0	0.779	0.779
TRPM3	TRPV4	0	0	0	0.779	0.778

TRPC4	TRPV2	0	0	0	0.778	0.778
TRPM7	TRPV4	0	0	0	0.779	0.778
TRPM7	TRPV1	0	0	0	0.777	0.776
TRPM2	TRPV4	0	0	0	0.772	0.772
CATSPER2	CATSPER3	0	0	0	0.773	0.772
ORAI1	TRPM2	0	0	0	0.771	0.771
TRPV2	TRPM2	0	0	0	0.77	0.77
TRPM3	TRPV2	0	0	0	0.769	0.769
TRPC3	TRPV2	0	0	0	0.76	0.76
TRPV6	TRPC1	0	0	0	0.756	0.756
TRPM7	TRPA1	0	0	0	0.755	0.754
TRPA1	TRPM4	0	0	0	0.753	0.752
TRPM3	TRPV3	0	0	0	0.744	0.744
ITPR3	TRPM2	0	0.548	0	0.465	0.742
TRPV6	TRPM4	0	0	0	0.743	0.742
TRPV3	TRPM4	0	0	0	0.739	0.738
CACNG6	CACNG5	0	0	0	0.738	0.738
TRPM6	TRPV6	0	0	0	0.737	0.736
TRPM8	TRPV5	0	0	0	0.736	0.736
TRPV3	TRPM2	0	0	0	0.731	0.73
TRPM6	TRPV4	0	0	0	0.731	0.73
RYR1	CACNG1	0	0	0	0.729	0.729
TRPM3	TRPV1	0	0	0	0.721	0.72
TRPM3	TRPV5	0	0	0	0.721	0.72
TRPM3	TRPA1	0	0	0	0.72	0.72
TRPM2	CACNA1C	0	0	0	0.713	0.713
TRPV5	TRPM4	0	0	0	0.711	0.71
TRPM1	TRPV2	0	0	0	0.708	0.708
TRPC3	TRPV6	0	0	0	0.704	0.704
CACNA2D2	CACNA1B	0	0	0	0.697	0.697
CACNA2D3	CACNA1B	0	0	0	0.697	0.697

CACNG2	GRIN2B	0	0	0	0.697	0.697
TRPV4	PKD2	0	0	0	0.687	0.687
ITPR2	TRPM7	0	0	0	0.682	0.682
TRPV6	TRPM8	0	0	0	0.68	0.679
TRPM1	TRPV4	0	0	0	0.679	0.679
TRPC4	TRPA1	0	0	0	0.674	0.674
TRPM1	TRPA1	0	0	0	0.674	0.673
TRPC3	RYR1	0	0	0	0.67	0.669
RYR1	TRPC1	0	0	0	0.657	0.657
CACNA1A	CACNA2D3	0	0	0	0.653	0.653
RYR2	CACNA1D	0	0	0	0.648	0.648
TRPV2	TRPV1	0.919	0.62	0	0.931	0.648
TRPV6	TRPM2	0	0	0	0.644	0.643
CACNA2D4	CACNA1C	0	0	0	0.643	0.643
GRIN2A	CACNG2	0	0	0	0.642	0.642
TRPM6	TRPM7	0.939	0.62	0	0.985	0.642
ITPR1	TRPC5	0	0.559	0	0.233	0.639
TRPC1	TRPA1	0	0	0	0.637	0.637
CACNA1I	CATSPER2	0.646	0.62	0	0.176	0.636
ITPR1	ITPR3	0.954	0.62	0	0.927	0.636
RYR2	RYR1	0.957	0.62	0	0.992	0.636
ITPR1	ORAI1	0	0	0	0.636	0.635
RYR3	RYR2	0.96	0.62	0	0.988	0.635
TRPM2	TRPV5	0	0	0	0.634	0.633
CACNA1I	CATSPER1	0.56	0.62	0	0.133	0.632
TRPM1	CACNA2D3	0	0	0	0.632	0.631
ITPR3	TRPC5	0	0.548	0	0.233	0.63
TRPM6	TRPA1	0	0	0	0.629	0.629
PANX1	P2RX1	0	0	0	0.628	0.627
CACNA2D4	CACNA1A	0	0	0	0.621	0.621
TRPM2	TRPA1	0	0	0	0.62	0.619

RYR2	CACNA1G	0	0	0	0.614	0.614
CACNB1	RYR1	0	0	0	0.599	0.599
TRPM3	TRPV6	0	0	0	0.593	0.593
TRPC5	TRPM4	0	0	0	0.59	0.589
ITPR3	TRPC7	0	0.548	0	0.144	0.587
TRPM6	TRPV2	0	0	0	0.586	0.586
TRPM6	TRPV1	0	0	0	0.572	0.571
CACNA1G	CACNA2D3	0	0	0	0.568	0.568
CACNA2D2	CACNA1G	0	0	0	0.568	0.568
ORAI1	TRPC7	0	0	0	0.566	0.566
TRPC3	TRPC7	0.966	0.552	0	0.891	0.565
TRPV2	TRPC5	0	0	0	0.565	0.565
TRPM8	TRPV1	0.445	0	0	0.955	0.558
TRPC3	TRPV5	0	0	0	0.557	0.556
TRPC3	TRPM4	0.452	0	0	0.953	0.551
CACNA1E	CACNB2	0	0	0	0.539	0.539
TMEM37	TRPV5	0	0	0	0.53	0.53
ORAI1	TRPM7	0	0	0	0.528	0.528
ITPR1	CACNA1A	0	0	0	0.528	0.528
ITPR1	CACNA1D	0	0	0	0.522	0.522
CACNA2D2	CACNA1C	0	0	0	0.515	0.515
ORAI1	CACNA1C	0	0	0	0.505	0.505
TRPM8	TRPV3	0.486	0	0	0.917	0.502
TRPC5	PKD2	0	0	0	0.5	0.5
TRPC3	TRPM2	0.51	0	0	0.946	0.496
TRPA1	TRPV1	0.469	0	0	0.877	0.495
TRPC5	TRPA1	0	0	0	0.493	0.493
ITPR3	ORAI1	0	0	0	0.492	0.491
ITPR2	ORAI1	0	0	0	0.492	0.491
RYR2	CACNG1	0	0	0	0.486	0.486
RYR3	CACNG1	0	0	0	0.486	0.486

CACNG2	CACNA1B	0	0	0	0.484	0.484
TRPM1	TRPC1	0.519	0	0	0.932	0.481
CACNA2D2	CACNA1I	0	0	0	0.481	0.481
CACNA1I	CACNA2D3	0	0	0	0.481	0.481
TRPM8	TRPV4	0.459	0	0	0.828	0.476
CACNA2D4	CACNB2	0	0	0	0.475	0.475
CACNB2	CACNA2D3	0	0	0	0.475	0.475
CACNA1H	CACNA2D3	0	0	0	0.474	0.474
CACNA2D2	CACNA1H	0	0	0	0.474	0.474
TRPV2	TRPA1	0.471	0	0	0.836	0.472
ITPR2	TRPV6	0	0	0	0.468	0.468
TRPC4	TRPV4	0.44	0	0	0.785	0.467
ITPR1	RYR1	0.451	0	0	0.797	0.466
ITPR1	TRPM2	0	0	0	0.465	0.465
ITPR2	TRPM2	0	0	0	0.465	0.465
TRPM2	TRPC7	0.507	0	0	0.876	0.464
TRPM2	TRPC5	0.524	0	0	0.907	0.464
RYR3	PSEN1	0	0	0	0.463	0.463
CACNA1A	CACNG1	0	0	0	0.463	0.463
RYR2	TRPC1	0	0	0	0.463	0.463
TRPC4	TRPM2	0.527	0	0	0.908	0.463
RYR3	CACNA1C	0	0	0	0.462	0.462
RYR1	CACNA1C	0	0	0	0.462	0.462
TRPM6	TRPV5	0.45	0	0	0.779	0.456
ITPR2	TRPV5	0	0	0	0.455	0.455
TRPA1	TRPV4	0.497	0	0	0.836	0.452
TRPC5	TRPV4	0.44	0	0	0.758	0.452
CACNA2D3	CACNA1C	0	0	0	0.447	0.447
TRPV2	TRPC1	0.471	0	0	0.786	0.445
ITPR3	TRPM7	0	0	0	0.444	0.443
CACNA2D2	CACNB2	0	0	0	0.444	0.443

TRPM7	TRPC1	0.48	0	0	0.79	0.441
TRPM2	TRPC1	0.572	0	0	0.942	0.439
TRPC4	TRPV1	0.463	0	0	0.765	0.439
TRPC4	TRPM4	0.474	0	0	0.776	0.438
CACNA2D3	CACNG1	0	0	0	0.437	0.436
CACNA2D4	CACNG1	0	0	0	0.437	0.436
TRPC3	TRPA1	0.433	0	0	0.72	0.435
TRPC7	TRPM4	0	0	0	0.436	0.435
TRPV6	CACNA1D	0	0	0	0.434	0.434
TRPM3	ITPR3	0	0	0	0.433	0.433
ITPR1	TRPM3	0	0	0	0.433	0.433
ITPR2	TRPM3	0	0	0	0.433	0.433
TRPC3	TRPM7	0.485	0	0	0.78	0.432
TRPM3	ORAI1	0	0	0	0.43	0.43
TRPM7	TRPV3	0.447	0	0	0.726	0.429
TRPM7	TRPV5	0.451	0	0	0.725	0.426
CACNB1	CACNA2D3	0	0	0	0.424	0.424
CACNA1B	TRPV1	0	0	0	0.424	0.424
CACNB1	CACNA2D4	0	0	0	0.424	0.424
TRPV3	TRPA1	0.534	0	0	0.836	0.423
ITPR1	GRIN2B	0	0	0	0.419	0.419
ITPR3	RYR1	0.451	0	0	0.709	0.417
ITPR2	RYR1	0.453	0	0	0.709	0.416
TRPC4	TRPV6	0.468	0	0	0.723	0.414
ITPR1	RYR3	0.45	0	0	0.703	0.414
TPCN1	TRPC1	0	0	0	0.413	0.413
TRPM1	TRPC3	0.495	0	0	0.757	0.413
RYR3	CACNA1G	0	0	0	0.41	0.41
TRPC4	TRPM7	0.513	0	0	0.776	0.41
TRPM7	CACNA1D	0	0	0	0.407	0.407
CACNA2D2	CACNG1	0	0	0	0.407	0.407

TRPM3	TRPC1	0.485	0	0	0.728	0.405
RYR3	CACNA1H	0	0	0	0.404	0.404
TRPC7	PKD2	0	0	0	0.403	0.402
RYR3	TRPM7	0	0	0	0.402	0.401
CACNA1H	TRPM7	0	0	0	0.402	0.401
TRPV6	TRPM7	0.465	0	0	0.695	0.401

Homology Score: calculated based on the degree of homology of the interactors. **Experimental Score**: based on a list of significant protein interaction datasets, gathered from other protein-protein interaction databases: BIND, DIP, GRID, HPRD, IntAct, MINT, and PID; **Knowledge Score**: based on pathway and functional annotation databases: Biocarta, BioCyc, GO, KEGG, and Reactome; **Text Mining Score**: based on a list of significant protein interaction groups, extracted from the abstracts of scientific literature; **Combined Score**: computed by combining the probabilities from the different evidence channels, correcting for the probability of randomly observing an interaction. For a more detailed description refer to von Mering C et al.[14].

1. Purcell S, Neale B, Todd-Brown K, Thomas L, Ferreira MA, Bender D, et al. PLINK: a tool set for whole-genome association and population-based linkage analyses. Am J Hum Genet. 2007; **81**(3): 559-75.
2. Price AL, Patterson NJ, Plenge RM, Weinblatt ME, Shadick NA, Reich D. Principal components analysis corrects for stratification in genome-wide association studies. Nature genetics. 2006; **38**(8): 904-9.
3. Browning SR, Browning BL. Rapid and accurate haplotype phasing and missing-data inference for whole-genome association studies by use of localized haplotype clustering. Am J Hum Genet. 2007; **81**(5): 1084-97.
4. Sklar P, Ripke S, Scott LJ, Andreassen OA, Cichon S, Craddock N, et al. Large-scale genome-wide association analysis of bipolar disorder identifies a new susceptibility locus near ODZ4. Nat Genet. 2011.
5. Ripke S, Sanders AR, Kendler KS, Levinson DF, Sklar P, Holmans PA, et al. Genome-wide association study identifies five new schizophrenia loci. Nat Genet. 2011.
6. Lee PH, Bergen SE, Perlis RH, Sullivan PF, Sklar P, Smoller JW, et al. Modifiers and subtype-specific analyses in whole-genome association studies: a likelihood framework. Human heredity. 2011; **72**(1): 10-20.
7. Huang J, Perlis RH, Lee PH, Rush AJ, Fava M, Sachs GS, et al. Cross-Disorder Genomewide Analysis of Schizophrenia, Bipolar Disorder, and Depression. Am J Psychiatry. 2010.
8. Lee PH, O'Duslaine C, Thomas B, Purcell S. INRICH: Interval-based enrichment analysis for genome wide association studies. in prep.
9. Subramanian A, Tamayo P, Mootha VK, Mukherjee S, Ebert BL, Gillette MA, et al. Gene set enrichment analysis: a knowledge-based approach for interpreting genome-wide expression profiles. Proceedings of the National Academy of Sciences of the United States of America. 2005; **102**(43): 15545-50.
10. Nicolae D, Gamazon E, Zhang W, Duan S, Dolan ME, Cox NJ. Trait-Associated SNPs Are More Likely to Be eQTLs: Annotation to Enhance Discovery from GWAS. PLoS Genet. 2010; **6**(4): e1000888.
11. Myers A, Gibbs JR, Webster JA, Rohrer K, Zhao A, Marlowe L, Kaleem M, Leung D, Bryden L, Nath P, Zismann VL, Joshipura K, Huentelman MJ, Hu-Lince D, Coon KD, Craig DW, Pearson JV, Holmans P, Heward CB, Reiman EM, Stephan D, Hardy J. A survey of genetic human cortical gene expression. Nat Genet. 2007; **39**(12): 1494-9.
12. Webster J, Gibbs JR, Clarke J, Ray M, Zhang W, Holmans P, Rohrer K, Zhao A, Marlowe L, Kaleem M, McCorquodale DS 3rd, Cuello C, Leung D, Bryden L, Nath P, Zismann VL, Joshipura K, Huentelman MJ, Hu-Lince D, Coon KD, Craig DW, Pearson JV; NACC-Neuropathology Group, Heward CB, Reiman EM, Stephan D, Hardy J, Myers AJ. Genetic Control of Human Brain Transcript Expression in Alzheimer Disease. Am J Hum Genet. 2009; **84**(4): 445-58.
13. Colantuoni C, Lipska BK, Ye T, Hyde TM, Tao R, Leek JT, Colantuoni EA, Elkahloun AG, Herman MM, Weinberger DR, Kleinman JE. Temporal dynamics and genetic control of transcription in the human prefrontal cortex. Nature. 2011; **478**(7370): 519-23.
14. Schadt E, Molony C, Chudin E, Hao K, Yang X, Lum PY, Kasarskis A, Zhang B, Wang S, Suver C, Zhu J, Millstein J, Sieberts S, Lamb J, GuhaThakurta D, Derry J, Storey JD, Avila-Campillo I, Kruger MJ, Johnson JM, Rohl CA, van Nas A, Mehrabian M, Drake

TA, Lusis AJ, Smith RC, Guengerich FP, Strom SC, Schuetz E, Rushmore TH, Ulrich R. Mapping the genetic architecture of gene expression in human liver. PLoS Biol. 2008; **6**(5): e107.
15. Ding J, Gudjonsson JE, Liang L, Stuart PE, Li Y, Chen W, Weichenthal M, Ellinghaus E, Franke A, Cookson W, Nair RP, Elder JT, Abecasis GR. Gene Expression in Skin and Lymphoblastoid Cells: Refined Statistical Method Reveals Extensive Overlap in cis-eQTL Signals. Am J Hum Genet. 2010; **87**(6): 779-89.
16. Stranger B, Nica AC, Forrest MS, Dimas A, Bird CP, Beazley C, Ingle CE, Dunning M, Flicek P, Koller D, Montgomery S, Tavaré S, Deloukas P, Dermitzakis ET. Population genomics of human gene expression. Nat Genet. 2007; **39**(10): 1217-24.
17. Burnham KP, Anderson DR. Model Selection and Multimodel Inference: A Practical Information-Theoretic Approach, 2nd Ed. New York: Spring-Verlag New York, Inc.; 2002.

PGC Workgroup(s)	First name	Last name	Affiliation	Country	email
CDG	Jordan W.	Smoller; MD; ScD*	Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital; Stanley Center for Psychiatric Research, Broad Institute, Boston, MA	USA	jsmoller@hms.harvard.edu
CDG; Analysis; SCZ	Stephan	Ripke; MD*	Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.; Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	ripke@chgr.mgh.harvard.edu
Analysis, BPD, SCZ	Phil Hyoun	Lee; PhD	Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital; Boston, MA	USA	phlee@pngu.mgh.harvard.edu
CDG; ADHD; Analysis, SCZ	Benjamin	Neale; PhD	Massachusetts General Hospital/Broad Institute	USA	bneale@broadinstitute.org
CDG; BPD	John I.	Nurnberger; MD	Indiana University	USA	jnurnber@iupui.edu
CDG; ASD	Susan	Santangelo; PhD	Massachusetts General Hospital	USA	ssantangelo@pngu.mgh.harvard.edu
CDG; MDD; SCZ	Patrick F.	Sullivan; MD	University of North Carolina at Chapel Hill	USA	pfsulliv@med.unc.edu
CDG	Roy H.	Perlis, MD, MSc	Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital; Stanley Center for Psychiatric Research, Broad Institute, Boston, MA	USA	rperlis@partners.org
Analysis, BPD, SCZ, CDG	Shaun M	Purcell, PhD	Division of Psychiatric Genomics, Department of Psychiatry, Mount Sinai School of Medicine, New York, New York, USA.; Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.; Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	shaun@atgu.mgh.harvard.edu
CDG, SCZ	Ayman	Fanous	Washington VA Medical Center; Department of Psychiatry; Georgetown University School of Medicine; Washington; DC; USA; Department of Psychiatry; Virginia Commonwealth University School of Medicine; Richmond; Virginia; USA	USA	ahfanous@vcu.edu
CDG, Analysis	Michael C.	Neale, PhD	Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University	USA	neale@vcu.edu
CDG; MDD; SCZ; BPD	Marcella	Rietschel, MD	Central Institute of Mental Health, Medical Faculty Mannheim, Univ. of Heidelberg J5, 68159 Mannheim, Germany	Germany	marcella.rietschel@zi-mannheim.de
CDG; MDD; SCZ; BPD	Thomas G.	Schulze	(1) Department of Psychiatry & Psychotherapy; University of Göttingen; Göttingen; (2) Department of Psychiatry & Behavioral Sciences; Johns Hopkins University; Baltimore; MD	1) Germany; 2) USA	1) thomas.schulze@med.uni-goettingen.de; 2) thomas_g_schulze@jhu.edu
CDG, ADHD	Anita	Thapar, MD	Institute of Psychological Medicine and Clinical Neurosciences,MRC Centre in Neuropsychiatric Genetics and Genomics, Cardiff University School of Medicine	UK	Thapar@cardiff.ac.uk
ASD; ADHD	Richard	Anney; PhD	Trinity College Dublin	Ireland	anneyr@TCD.IE
ADHD	Jan K.	Buitelaar; MD; PhD	Radboud University Medical Centre; Donders Institute for Brain; Cognition and Behaviour; department of Cognitive Neuroscience; Nijmegen; The Netherlands	NL	j.buitelaar@psy.umcn.nl
PGC Coordinating Committee; ADHD	Stephen V.	Faraone, Ph.D.	SUNY Upstate Medical University	USA	sfaraone@childpsychresearch.org
MDD	Witte J	Hoogendijk	Erasmus Medical Center	The Netherlands	w.hoogendijk@erasmusmc.nl
MDD, SCZ, Coordinating Committee	Douglas F	Levinson	Department of Psychiatry and Behavioral Sciences; Stanford University; Stanford; California; USA	USA	dflev@stanford.edu
ADHD	Klaus-Peter	Lesch; MD	University of Wuerzburg	Germany	kplesch@mail.uni-wuerzburg.de
PGC	Brien	Riley	Depts. of Psychiatry and Human & Molecular Genetics, Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond VA	USA	bpriley@vcu.edu
ADHD	Russell	Schachar	The Hospital for Sick Children, University of Toronto	Canada	russell.schachar@sickkids.ca
ADHD	Edmund	Sonuga-Barke PhD	Developmental Brain & Behaviour Laboratory, Psychology University of Southampton; Department of Experimental Clinical & Health Psychology, Ghent University	UK	ejb3@soton.ac.uk
BPD	Devin	Absher	HudsonAlpha Institute for Biotechnology, Huntsville, Alabama, USA.	USA	
SCZ	Ingrid	Agartz	KG Jebsen Centre for Psychosis Research, Institute of Clinical Medicine; University of Oslo; Department of Research; Diakonhjemmet Hospital; Oslo; Norway	Norway	ingrid.agartz@medisin.uio.no
BPD	Huda	Akil	Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, Michigan, USA.	USA	
SCZ	Farooq	Amin	Department of Psychiatry and Behavioral Sciences; Emory University and Atlanta Veterans Affairs Medical Center; Atlanta; Georgia; USA	USA	famin@emory.edu
SCZ, BPD	Ole A	Andreassen	KG Jebsen Centre for Psychosis Research; Institute of Clinical Medicine; University of Oslo; Division of Mental Health and Addiction, Oslo University Hospital, Oslo, Norway	Norway	o.a.andreassen@medisin.uio.no
BPD	Adebayo	Anjorin	Molecular Psychiatry Laboratory, Mental Health Sciences Unit, University College London, London, UK.	UK	
ASD	Dan	Arking; PhD	Johns Hopkins University	USA	arking@JHMI.EDU
ADHD	Philip	Asherson MD	King's College London, Institute of Psychiatry	UK	philip.asherson@kcl.ac.uk
SCZ	Maria H.	Azevedo, MD	Faculty of Medicine, University of Coimbra, Coimbra, Portugal	Portugal	mazevedo@fmed.uc.pt
BPD	Lena	Backlund	Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.	Sweden	
BPD	Judith A	Badner	Department of Psychiatry, University of Chicago, Chicago, Illinois, USA.	USA	
ADHD	Tobias	Banaschewski	Department of Child and Adolescent Psychiatry; Central Institute of Mental Health; Medical Faculty Mannheim, University of Heidelberg; Mannheim; Germany	Germany	tobias.banaschewski@zi-mannheim.de
BPD	Jack D	Barchas	Department of Psychiatry, Weill Medical College, Cornell University, New York, New York, USA.	USA	
MDD	Michael R	Barnes	GlaxoSmithKline	UK	dr.michael.r.barnes@gmail.com

BPD	Thomas B	Barrett	Portland Veterans Affairs Medical Center, Portland, Oregon, USA.	USA	
SCZ, BPD	Nicholas	Bass	Molecular Psychiatry Laboratory; Research Department of Mental Health Sciences; University College London Medical School; Windeyer Institute of Medical Sciences; London; England; UK	UK	rejunjb@ucl.ac.uk
BPD	Michael	Bauer	Department of Psychiatry and Psychotherapy; ENBREC Group; University Hospital Carl Gustav Carus; Dresden; Germany.	Germany	
BPD	Frank	Bellivier	INSERM, U955, Psychiatrie Génétique, Créteil, France.; Université Paris Est, Faculté de Médecine, Créteil, France.; Assistance Publique–Hôpitaux de Paris (AP-HP); Hôpital H. Mondor–A. Chenevier; Département de Psychiatrie; Créteil; France.; ENBREC group; Fondation Fondamental; Créteil; France.	France	
BPD	Sarah E	Bergen	Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.; Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	sbergen@broadinstitute.org
BPD	Wade	Berrettini	Department of Psychiatry, University of Pennsylvania, Philadelphia, Pennsylvania, USA.	USA	wadeb@mail.med.upenn.edu
MDD	Thomas	Bettecken	Max Planck Institute of Psychiatry; Munich	Germany	bettecken@mpipsykl.mpg.de
ADHD	Joseph	Biederman; MD	Massachusetts General Hospital; Harvard Medical School	USA	jbiederman@partners.org
MDD	Elisabeth B	Binder	Max Planck Institute of Psychiatry; Munich	Germany	binder@mpipsykl.mpg.de
SCZ	Donald W	Black	Department of Psychiatry; University of Iowa Carver College of Medicine; Iowa City; Iowa; USA	USA	donald-black@uiowa.edu
SCZ, BPD, MDD	Douglas H R	Blackwood	Division of Psychiatry; University of Edinburgh; Royal Edinburgh Hospital; Edinburgh; Scotland; UK	UK	d.blackwood@ed.ac.uk
BPD	Cinnamon S	Bloss	The Scripps Translational Science Institute and Scripps Health, La Jolla, California, USA.	USA	
BPD	Michael	Boehnke	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.	USA	
MDD	Dorret I	Boomsma	VU University; Amsterdam	The Netherlands	dorret@psy.vu.nl;
BPD, MDD	Gerome	Breen	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.; University of Aberdeen, Institute of Medical Sciences, Foresterhill, Aberdeen, UK.	UK	gerome.breen@kcl.ac.uk
MDD; BPD	René	Breuer	Department of Genetic Epidemiology in Psychiatry; Central Institute of Mental Health; University of Heidelberg; Mannheim; Germany	Germany	rene.breuer@zi-mannheim.de
SCZ	Nancy G	Buccola	School of Nursing; Louisiana State University Health Sciences Center; New Orleans; Louisiana; USA	USA	NBucco@lsuhsc.edu
BPD	William E	Bunner	Department of Psychiatry and Human Behavior, University of California, Irvine, California, USA.	USA	
BPD	Margit	Burmeister	Department of Human Genetics, Department of Psychiatry, Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, Michigan, USA.	USA	
ASD	Joseph D	Buxbaum; PhD	Mt. Sinai School of Medicine	USA	joseph.buxbaum@MSSM.EDU
BPD, SCZ	William F	Byerley	Department of Psychiatry; University of California at San Francisco and NCIRE (Northern California Institute for Research And Education); San Francisco; California; USA	USA	william.byerley@ucsf.edu
BPD	Sian	Caesar	Department of Psychiatry, School of Clinical and Experimental Medicine, Birmingham University, Birmingham, UK.	UK	
SCZ	Rita M	Cantor	UCLA Center for Neurobehavioral Genetics; University of California at Los Angeles; Department of Human Genetics; University of California at Los Angeles; Los Angeles; California; USA	USA	RCantor@mednet.ucla.edu
ASD	Aravinda	Chakravarti; PhD	Johns Hopkins University	USA	aravinda@JHMI.EDU
BPD	Kim	Chambert	Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	
SCZ	Khalid	Choudhury, PhD	University College London	UK	khalid.choudhury@ucl.ac.uk
MDD; BPD; SCZ	Sven	Cichon	(1) Dept. of Genomics; Life & Brain Center; University of Bonn; Bonn; (2) Institute of Neuroscience and Medicine (INM-1); Research Center Juelich; Juelich; (3) Institute of Human Genetics; Unviersity of Bonn; Bonn; Germany	Germany	sven.cichon@uni-bonn.de
SCZ	C. Robert	Cloninger, MD	Washington University	USA	crcloninger44@gmail.com
BPD	David A	Collier	Social; Genetic and Developmental Psychiatry Centre; Institute of Psychiatry; King's College; London; England; UK	UK	David.Collier@iop.kcl.ac.uk
ASD	Edwin	Cook; MD	University of Illinois	USA	ecook@PSYCH.UIC.EDU
ASD	Hilary	Coon; PhD	University of Utah	USA	hilary.coon@UTAH.EDU
BPD, SCZ	Aiden	Corvin	Neuropsychiatric Genetics Research Group; Department of Psychiatry and Institute of Molecular Medicine; Trinity College Dublin; Dublin; Ireland	Ireland	acorvin@tcd.ie
BPD, MDD	William H	Coryell	University of Iowa	USA	william-coryell@uiowa.edu
BPD	David W	Craig	The Translational Genomics Research Institute, Phoenix, Arizona, USA.	USA	
MDD	Ian W	Craig	Institute of Psychiatry; King's College London	UK	ian.craig@kcl.ac.uk
SCZ	David	Curtis, MD, PhD	East London NHS Foundation Trust/QMUL	UK	davidcurtis@nhs.net
MDD	Darina	Czamara	Max Planck Institute of Psychiatry; Munich	Germany	darina@mpipsykl.mpg.de
Analysis, ASD, BPD, SCZ, Coordinating Committee	Mark	Daly	Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.; Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	mjdaly@atgu.mgh.harvard.edu
SCZ	Susmita	Datta, PhD	University College London	UK	sdatta@hotmail.com
BPD	Richard	Day	Division of Neuroscience, Medical Research Institute, University of Dundee, Ninewells Hospital & Medical School, Dundee, UK.	IL	
MDD	Eco J	De Geus	VU University; Amsterdam	The Netherlands	eco@psy.vu.nl

MDD; BPD	Franziska	Degenhardt	(1) Dept. of Genomics; Life & Brain Center; University of Bonn; Bonn; (2) Institute of Human Genetics; Unviersity of Bonn; Bonn; Germany	Germany	franziska.degenhardt@uni-bonn.de
ASD	Bernie	Devlin; PhD	University of Pennsylvania	USA	devlinbj@UPMC.EDU
BPD, SCZ	Srdjan	Djurovic	KG Jebsen Centre for Psychosis Research, Institute of Clinical Medicine, University of Oslo, Oslo, Norway.; Department of Medical Genetics, Oslo University Hospital, Oslo, Norway.	Norway	srdjan.djurovic@medisin.uio.no
ADHD	Alysa E.	Doyle	Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.	USA	doylea@helix.mgh.harvard.edu
SCZ	Jubao	Duan	Department of Psychiatry and Behavioral Sciences; NorthShore University HealthSystem and University of Chicago; Evanston; Illinois; USA	USA	jduan@northshore.org
BPD, SCZ	Frank	Dudbridge	Department of Non-communicable Disease Epidemiology; London School of Hygiene and Tropical Medicine; London; England; UK	UK	frank.dudbridge@lshtm.ac.uk
BPD	Howard J	Edenberg	Department of Biochemistry and Molecular Biology, Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis, Indiana, USA.; Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis, Indiana, USA.	USA	
BPD	Amanda	Elkin	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.	UK	
BPD	Bruno	Etain	INSERM, U955, Psychiatrie Génétique, Créteil, France.; Université Paris Est, Faculté de Médecine, Créteil, France.; Assistance Publique–Hôpitaux de Paris (AP-HP); Hôpital H. Mondor–A. Chenevier; Département de Psychiatrie; Créteil; France.; ENBREC group; Fondation Fondamental; Créteil; France.	France	
BPD, MDD	Anne E	Farmer	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.	UK	anne.farmer@kcl.ac.uk
BPD	Manuel A	Ferreira	Queensland Institute of Medical Research, Brisbane, Australia.	Australia	
BPD	I Nicol	Ferrier	Division of Neuroscience, Medical Research Institute, University of Dundee, Ninewells Hospital & Medical School, Dundee, UK.	UK	
BPD	Matthew	Flickinger	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.	USA	
BPD	Tatiana	Foroud	Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis, Indiana, USA.	USA	
MDD; BPD	Josef	Frank	Department of Genetic Epidemiology in Psychiatry; Central Institute of Mental Health; University of Heidelberg; Mannheim; Germany	Germany	josef.frank@zi-mannheim.de
ADHD	Barbara	Franke; PhD	Radboud University Medical Centre; Donders Institute for Brain; Cognition and Behaviour; departments of Human Genetics (855) and Psychiatry; Nijmegen; The Netherlands	The Netherlands	b.franke@gen.umcn.nl
BPD	Christine	Fraser	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.	UK	
SCZ	Robert	Freedman	Department of Psychiatry; University of Colorado Denver; Aurora; Colorado; USA	USA	Robert.Freedman@ucdenver.edu
SCZ	Nelson B	Freimer	UCLA Center for Neurobehavioral Genetics; University of California at Los Angeles; Los Angeles; California; USA	USA	nfreimer@mednet.ucla.edu
SCZ	Marion	Friedl	Molecular and Clinical Neurobiology; Department of Psychiatry; Ludwig-Maximilians-University; Munich; Germany	Germany	Marion.Friedl@med.uni-muenchen.de
BPD	Louise	Frisén	Department of Molecular Medicine, Karolinska Institutet, Stockholm, Sweden.	Sweden	
SCZ	Pablo V	Gejman	Department of Psychiatry and Behavioral Sciences; NorthShore University HealthSystem and University of Chicago; Evanston; Illinois; USA	USA	pgejman@northshore.org
SCZ	Lyudmila	Georgieva	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	georgievaln1@cardiff.ac.uk
BPD	Elliot S	Gershon	Institute for Medical Biometry, Informatics and Epidemiology, University of Bonn, Bonn, Germany.	Germany	
SCZ	Ina	Giegling	Molecular and Clinical Neurobiology; Department of Psychiatry; Ludwig-Maximilians-University; Munich; Germany	Germany	Ina.Giegling@med.uni-muenchen.de
ASD, BPD, SCZ	Michael	Gill; MD	Neuropsychiatric Genetics Research Group, Department of Psychiatry and Institute of Molecular Medicine, Trinity College Dublin, Dublin, Ireland.	Ireland	mgill@TCD.IE
MDD	Scott D	Gordon	Queensland Institute of Medical Research	Australia	scott.gordon@qimr.edu.au
BPD	Katherine	Gordon-Smith	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.; Department of Psychiatry, School of Clinical and Experimental Medicine, Birmingham University, Birmingham, UK.	UK	
BPD	Elaine K	Green	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.	UK	
BPD	Tiffany A	Greenwood	Neurosciences Centre of Excellence in Drug Discovery, GlaxoSmithKline Research and Development, Verona, Italy.	Italy	
SCZ	The Genetic Risk Outcome of Psychosis (GROUP) Consortium				
MDD	Magdalena	Gross	Dept. of Psychiatry; University of Bonn; Bonn; Germany	Germany	m.gross@uni-bonn.de
BPD	Detelina	Grozeva	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.	UK	

BPD	Weihua	Guan	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.	USA	
BPD, SCZ	Hugh	Gurling	Molecular Psychiatry Laboratory; Research Department of Mental Health Sciences; University College London Medical School; Windeyer Institute of Medical Sciences; London; England; UK	UK	h.gurling@ucl.ac.uk
BPD	Ómar	Gustafsson	deCODE Genetics, Sturlugata 8 101 Reykjavík, Iceland.	Iceland	
ASD	Hakon	Hakonarson	The Center for Applied Genomics, The Children´s Hospital of Philadelphia, Philadelphia, PA Department of Pediatrics, The Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA	USA	hakonarson@email.chop.edu
MDD	Steven P	Hamilton	University of California; San Francisco	USA	steve.hamilton@ucsf.edu
BPD, SCZ	Marian L	Hamshere	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.; Biostatistics and Bioinformatics Unit, Cardiff University School of Medicine, Cardiff, UK.	UK	HamshereML@cardiff.ac.uk
SCZ	Thomas F	Hansen	Institute of Biological Psychiatry; MHC Sct. Hans; Copenhagen University Hospital; Roskilde; Denmark	Denmark	thomas.hansen@regionh.dk
SCZ	Annette M	Hartmann	Molecular and Clinical Neurobiology; Department of Psychiatry; Ludwig-Maximilians-University; Munich; Germany	Germany	Annette.Hartmann@med.uni-muenchen.de
BPD	Martin	Hautzinger	Department of Clinical and Developmental Psychology; Institute of Psychology; University of Tubingen; Tubingen; Germany.	Germany	
MDD	Andrew C	Heath	University of Washington; St Louis	USA	AHeath@wustl.edu
MDD	Anjali K	Henders	Queensland Institute of Medical Research	Australia	anjali.henders@qimr.edu.au
MDD; BPD	Stefan	Herms	Dept. of Genomics; Life & Brain Center; University of Bonn; Bonn; Germany	Germany	stefan.herms@uni-bonn.de
MDD	Ian B	Hickie	University of Sydney; Sydney	Australia	ian.hickie@sydney.edu.au
BPD	Maria	Hipolito	Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine, Washington, DC, USA.	USA	
MDD	Susanne	Hoefels	Dept. of Psychiatry; University of Bonn; Bonn; Germany	Germany	susanne.hoefels@ukb.uni-bonn.de
BPD, SCZ	Peter A	Holmans	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.; Biostatistics and Bioinformatics Unit, Cardiff University School of Medicine, Cardiff, UK.	UK	holmanspa@cardiff.ac.uk
MDD	Florian	Holsboer	Max Planck Institute of Psychiatry	Germany	holsboer@mpipsykl.mpg.de
MDD	Jouke Jan	Hottenga	VU University; Amsterdam	The Netherlands	jj.hottenga@psy.vu.nl;
BPD, SCZ	Christina M	Hultman	Department of Medical Epidemiology and Biostatistics; Karolinska Institutet; Stockholm; Sweden	Sweden	Christina.Hultman@ki.se
SCZ	Andrés	Ingason	Institute of Biological Psychiatry; MHC Sct. Hans; Copenhagen University Hospital; Roskilde; Denmark	Denmark	andres.ingason@regionh.dk
MDD	Marcus	Ising	Max Planck Institute of Psychiatry; Munich	Germany	ising@atgu.mgh.harvard.edu
BPD	Stéphane	Jamain	INSERM, U955, Psychiatrie Génétique, Créteil, France.; Université Paris Est, Faculté de Médecine, Créteil, France.; Assistance Publique–Hôpitaux de Paris (AP-HP); Hôpital H. Mondor–A. Chenevier; Département de Psychiatrie; Créteil; France.; ENBREC group; Fondation Fondamental; Créteil; France.	France	
BPD	Edward G	Jones	Department of Psychiatry and Behavioral Sciences, Center for Neuroscience, University of California, Davis, California, USA.	USA	
BPD, MDD	Lisa	Jones	Department of Psychiatry, School of Clinical and Experimental Medicine, Birmingham University, Birmingham, UK.	UK	l.a.jones@bham.ac.uk
BPD, MDD	Ian	Jones	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.	UK	JonesIR1@cf.ac.uk
MDD	Tzeng	Jung-Ying	North Carolina State University	USA	jytzeng@stat.ncsu.edu
SCZ	Anna	Kahler, PhD	Karolinska Institutet	Sweden	anna.kahler@ki.se
BPD	Radhika	Kandaswamy	Molecular Psychiatry Laboratory, Mental Health Sciences Unit, University College London, London, UK.	UK	
SCZ	Matthew C.	Keller	Department of Psychology; University of Colorado; Boulder; Boulder; Colorado; USA	USA	matthew.c.keller@gmail.com
BPD	John R	Kelsoe	Department of Psychiatry, University of California San Diego, La Jolla, California, USA.; Department of Psychiatry; Special Treatment and Evaluation Program (STEP); Veterans Affairs San Diego Healthcare System; San Diego; California; USA.	USA	
BPD	James L	Kennedy	Psychiatric Neurogenetics Section, Centre for Addiction and Mental Health, Toronto, Ontario, Canada.	Canada	
SCZ	Elaine	Kenny	Neuropsychiatric Genetics Research Group, Dept of Psychiatry and Institute of Molecular Medicine, Trinity College Dublin, Dublin, Ireland	Ireland	kennyel@tcd.ie
ADHD	Lindsey	Kent; MD	University of St Andrews	UK	lsk8@st-andrews.ac.uk
SCZ	Yunjung	Kim	Department of Genetics; University of North Carolina at Chapel Hill; Chapel Hill; North Carolina; USA	USA	yunjungk@email.unc.edu
BPD, SCZ	George K	Kirov	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	kirov@cardiff.ac.uk
MDD	James A	Knowles	University of Southern California	USA	knowles@med.usc.edu
MDD	Martin A.	Kohli	Max Planck Institute of Psychiatry; Munich	Germany	MKohli@med.miami.edu
BPD	Daniel L	Koller	Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis, Indiana, USA.	USA	
SCZ	Bettina	Konte	Molecular and Clinical Neurobiology; Department of Psychiatry; Ludwig-Maximilians-University; Munich; Germany	Germany	Bettina.Konte@med.uni-muenchen.de
MDD	Ania	Korszun	Queen Mary University of London	UK	a.korszun@qmul.ac.uk
SCZ	Robert	Krasucki, MD	University College London	UK	robert@krasucki.freeserve.co.uk
ADHD	Jonna	Kuntsi PhD	King's College London, Institute of Psychiatry	UK	jonna.kuntsi@kcl.ac.uk

BPD	Phoenix	Kwan	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.	USA	
BPD	Mikael	Landén	Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden.; Institute of Neuroscience and Physiology, University of Gothenburg, Gothenburg, Sweden.	Sweden	
BPD	Niklas	Langstrom	Institute of Neuroscience and Physiology, University of Gothenburg, Gothenburg, Sweden.	Sweden	
BPD	Mark	Lathrop	Centre National de Génotypage; Evry; France.	France	
BPD, SCZ	Jacob	Lawrence	Molecular Psychiatry Laboratory; Research Department of Mental Health Sciences; University College London Medical School; Windeyer Institute of Medical Sciences; London; England; UK	UK	jacob.lawrence@nelmht.nhs.uk
BPD, MDD	William B	Lawson	Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine, Washington, DC, USA.	USA	wblawson@howard.edu
BPD	Marion	Leboyer	INSERM, U955, Psychiatrie Génétique, Créteil, France.; Université Paris Est, Faculté de Médecine, Créteil, France.; Assistance Publique–Hôpitaux de Paris (AP-HP); Hôpital H. Mondor–A. Chenevier; Département de Psychiatrie; Créteil; France.; ENBREC group; Fondation Fondamental; Créteil; France.	France	
SCZ	Todd	Lencz	Department of Psychiatry; Division of Research; The Zucker Hillside Hospital Division of the North Shore - Long Island Jewish Health System; Glen Oaks; New York; USA; Center for Psychiatric Neuroscience; The Feinstein Institute for Medical Research; Manhasset; New York; USA; Department of Psychiatry and Behavioral Science; Albert Einstein College of Medicine of Yeshiva University; Bronx; New York; USA	USA	lencz@lij.edu
ADHD	Klaus-Peter	Lesch; MD	Division of Molecular Psychiatry, ADHD Clinical Research Unit, Department of Psychiatry, Psychosomatics and Psychotherapy, University of Wuerzburg, and Department of Neuroscience, School for Mental Health and Neuroscience (MHENS), Maastricht University, The Netherlands	Germany	kplesch@mail.uni-wuerzburg.de
MDD	Cathryn M	Lewis	Institute of Psychiatry; King's College London	UK	cathryn.lewis@kcl.ac.uk
BPD	Jun	Li	Department of Human Genetics, University of Michigan, Ann Arbor, Michigan, USA	USA	
BPD	Paul	Lichtenstein	Department of Medical Epidemiology and Biostatistics; Karolinska Institutet; Stockholm; Sweden	Sweden	paul.lichtenstein@ki.se
SCZ	Jeffrey A	Lieberman	Department of Psychiatry; Columbia University; New York; New York; USA	USA	jlieberman@pi.cpmc.columbia.edu
BPD, MDD, SCZ	Danyu	Lin	Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, USA.	USA	lin@bios.unc.edu
BPD	Chunyu	Liu	Department of Psychiatry, University of Chicago, Chicago, Illinois, USA.	USA	
BPD	Falk W	Lohoff	Department of Psychiatry, University of Pennsylvania, Philadelphia, Pennsylvania, USA.	USA	lohoff@mail.med.upenn.edu
ADHD	Sandra K.	Loo; Ph.D.	University of California Los Angeles	USA	sloo@mednet.ucla.edu
BPD, MDD	Susanne	Lucae	Max Planck Institute of Psychiatry	Germany	lucae@mpipsykl.mpg.de
MDD	Donald	MacIntyre	University of Edinburgh	UK	donald.macintyre@lpct.scot.nhs.uk
MDD	Pamela AF	Madden	University of Washington; St Louis	USA	pmadden@wustl.edu
SCZ	Patrik	Magnusson, PhD	Karolinska Institutet	Sweden	patrik.magnusson@ki.se
BPD	Pamela B	Mahon	Department of Mental Health, Johns Hopkins University and Hospital, Baltimore, Maryland, USA.	USA	
MDD; BPD; SCZ	Wolfgang	Maier	Dept. of Psychiatry; University of Bonn; Bonn; Germany	Germany	wolfgang.maier@ukb.uni-bonn.de
SCZ	Anil K	Malhotra	Department of Psychiatry; Division of Research; The Zucker Hillside Hospital Division of the North Shore - Long Island Jewish Health System; Glen Oaks; New York; USA; Center for Psychiatric Neuroscience; The Feinstein Institute for Medical Research; Manhasset; New York; USA; Department of Psychiatry and Behavioral Science; Albert Einstein College of Medicine of Yeshiva University; Bronx; New York; USA	USA	malhotra@lij.edu
BPD, MDD	Nicholas G	Martin	Queensland Institute of Medical Research, Brisbane, Australia.	Australia	nick.martin@qimr.edu.au
MDD; BPD; SCZ	Manuel	Mattheisen	(1) Dept. of Genomics; Life & Brain Center; University of Bonn; Bonn; Germany; (2) Department of Biostatistics; Harvard School of Public Health; Boston; MA; USA; (3) Institute for Genomic Mathematics; University of Bonn; Bonn; Germany	Germany	mmattheisen@uni-bonn.de
BPD	Keith	Matthews	Division of Neuroscience, Medical Research Institute, University of Dundee, Ninewells Hospital & Medical School, Dundee, UK.	UK	
BPD, SCZ	Morten	Mattingsdal	KG Jebsen Centre for Psychosis Research, Institute of Clinical Medicine, University of Oslo, Oslo, Norway.; Sorlandet Hospital HF, Kristiansand, Norway.	Norway	morten.mattingsdal@gmail.com
SCZ	Steve	McCarroll, PhD	Harvard/Broad Institute	USA	mccarroll@genetics.med.harvard.edu
BPD	Kevin A	McGhee	Division of Psychiatry; University of Edinburgh; Royal Edinburgh Hospital; Edinburgh; Scotland; UK	UK	kmcghee@ed.ac.uk
ADHD	James J.	McGough	University of California, Los Angeles, CA	USA	jmcgough@mednet.ucla.edu
MDD	Patrick J	McGrath	Columbia University; NYSPI	USA	mcgrath@nyspi.columbia.edu
BPD, MDD	Peter	McGuffin	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.	UK	peter.mcguffin@kcl.ac.uk
BPD	Melvin G	McInnis	Department of Psychiatry, University of Michigan, Ann Arbor, Michigan, USA.	USA	
BPD, MDD, SCZ	Andrew	McIntosh	Division of Psychiatry, University of Edinburgh, Royal Edinburgh Hospital, Edinburgh, UK.; Medical Genetics Section, University of Edinburgh Molecular Medicine Centre, Western General Hospital, Edinburgh, UK.	UK	andrew.mcintosh@ed.ac.uk
BPD	Rebecca	McKinney	Neurosciences Centre of Excellence in Drug Discovery, GlaxoSmithKline Research and Development, Verona, Italy.	Italy	

BPD, MDD	Alan W	McLean	Division of Psychiatry, University of Edinburgh, Royal Edinburgh Hospital, Edinburgh, UK.; Medical Genetics Section, University of Edinburgh Molecular Medicine Centre, Western General Hospital, Edinburgh, UK.	UK	a.w.maclean@ed.ac.uk
BPD	Francis J	McMahon	National Institute of Mental Health, US National Institutes of Health, Bethesda, Maryland, USA.	USA	
BPD, SCZ	Andrew	McQuillin	Molecular Psychiatry Laboratory; Research Department of Mental Health Sciences; University College London Medical School; Windeyer Institute of Medical Sciences; London; England; UK	UK	a.mcquillin@ucl.ac.uk
SCZ	Helena	Medeiros, MSW	Keck School of Medicine, University of Southern California, Los Angeles, California, USA	USA	helena.medeiros@keck.usc.edu
Analysis	Sarah E.	Medland	Queensland Institute of Medical Research, Brisbane	Australia	medlandse@gmail.com
BPD	Sandra	Meier	Department of Genetic Epidemiology in Psychiatry, Central Institute of Mental Health Mannheim, University of Heidelberg, Mannheim, Germany.	Germany	
BPD, SCZ	Ingrid	Melle	KG Jebsen Centre for Psychosis Research; Institute of Clinical Medicine; University of Oslo; Division of Mental Health and Addiction, Oslo University Hospital, Oslo, Norway	Norway	ingrid.melle@medisin.uio.no
BPD	Fan	Meng	Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, Michigan, USA.	USA	
MDD	Christel M	Middeldorp	VU University; Amsterdam	The Netherlands	cm.middeldorp@psy.vu.nl
MDD	Lefkos	Middleton	Imperial College	UK	l.middleton@imperial.ac.uk
SCZ	Vihra	Milanova	Department of Psychiatry; First Psychiatric Clinic; Alexander University Hospital; Sofia; Bulgaria	Bulgaria	vihmil@yahoo.com
BPD	Philip B	Mitchell	School of Psychiatry; University of New South Wales and Black Dog Institute; Sydney; New South Wales; Australia.	Austrailia	
BPD, MDD	Grant W	Montgomery	Queensland Institute of Medical Research, Brisbane, Australia.	Austrailia	grant.montgomery@qimr.edu.au
BPD	Jennifer	Moran	Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	jlmoran@broadinstitute.org
BPD	Gunnar	Morken	Department of Psychiatry, St. Olavs Hospital, Trondheim, Norway.; Department of Neuroscience, Norwegian University of Science and Technology, Trondheim, Norway.	Norway	gunnar.morken@ntnu.no
BPD, SCZ	Derek W	Morris	Neuropsychiatric Genetics Research Group; Department of Psychiatry and Institute of Molecular Medicine; Trinity College Dublin; Dublin; Ireland	Ireland	morrisdw@tcd.ie
BPD, SCZ	Valentina	Moskvina	Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Henry Wellcome Building, School of Medicine, Cardiff University, Heath Park, Cardiff, UK.; Biostatistics and Bioinformatics Unit, Cardiff University School of Medicine, Cardiff, UK.	UK	
SCZ	Bryan J	Mowry	Queensland Brain Institute and Queensland Centre for Mental Health Research; University of Queensland; Brisbane; Queensland; Australia	Australia	b.mowry@uq.edu.au
BPD, MDD	Pierandrea	Muglia	Neurosciences Centre of Excellence in Drug Discovery, GlaxoSmithKline Research and Development, Verona, Italy.	Italy	pierandrea.muglia@utoronto.ca
BPD	Thomas W	Mühleisen	Institute of Human Genetics, University of Bonn, Bonn, Germany.; German Center for Neurodegenerative Disorders (DZNE), Bonn, Germany.	Germany	
BPD	Walter J	Muir	Division of Psychiatry, University of Edinburgh, Royal Edinburgh Hospital, Edinburgh, UK.; Medical Genetics Section, University of Edinburgh Molecular Medicine Centre, Western General Hospital, Edinburgh, UK.	UK	
BPD, MDD	Bertram	Müller-Myhsok	Max Planck Institute of Psychiatry; Munich	Germany	bmm@mpipsykl.mpg.de
BPD	Richard M	Myers	HudsonAlpha Institute for Biotechnology, Huntsville, Alabama, USA.	USA	
ADHD	Stan F.	Nelson	University of California Los Angeles	USA	snelson@ucla.edu
BPD	Caroline M	Nievergelt	Neurosciences Centre of Excellence in Drug Discovery, GlaxoSmithKline Research and Development, Verona, Italy.	Italy	
BPD	Ivan	Nikolov	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	ivan.nikolov@mac.com
BPD	Vishwajit	Nimgaonkar	Department of Human Genetics, University of Pittsburgh, Pittsburgh, Pennsylvania, USA.		
MDD	Willem A	Nolen	Groningen University Medical Center; Groningen	The Netherlands	w.a.nolen@psy.umcg.nl
MDD; BPD; SCZ	Markus M	Nöthen	(1) Dept. of Genomics; Life & Brain Center; University of Bonn; Bonn; (2) Institute of Human Genetics; Unviersity of Bonn; Bonn; Germany; (3) German Center for Neurodegenerative Disorders (DZNE); Bonn; Germany	Germany	markus.noethen@uni-bonn.de
BPD	Evaristus A	Nwulia	Department of Psychiatry and Behavioral Sciences, Howard University College of Medicine, Washington, DC, USA.	USA	
MDD	Dale R	Nyholt	Queensland Institute of Medical Research	Australia	dale.nyholt@qimr.edu.au
BPD, SCZ, Coordinating Committee	Michael C	O'Donovan	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	odonovanmc@cardiff.ac.uk
BPD, SCZ	Colm	O'Dushlaine	Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	odushlain@tcd.ie
ADHD	Robert D.	Oades; PhD	University of Duisburg-Essen	Germany	robert.oades@uni-due.de
SCZ	Ann	Olincy	Department of Psychiatry; University of Colorado Denver; Aurora; Colorado; USA	USA	Ann.Olincy@ucdenver.edu
SCZ	Line	Olsen	Institute of Biological Psychiatry; MHC Sct. Hans; Copenhagen University Hospital; Roskilde; Denmark	Denmark	line.olsen@regionh.dk

SCZ	Roel A	Ophoff	Department of Medical Genetics and Rudolf Magnus Institute of Neuroscience; University Medical Center Utrecht; Utrecht; The Netherlands; UCLA Center for Neurobehavioral Genetics; University of California at Los Angeles; Department of Human Genetics; University of California at Los Angeles; Los Angeles; California; USA	The Netherlands; USA	ROphoff@mednet.ucla.edu
BPD	Urban	Osby	Department of Molecular Medicine, Karolinska Institutet, Stockholm, Sweden.	Sweden	
BPD	Högni	Óskarsson	Therapeia; Reykjavik; Iceland.	Iceland	
BPD, SCZ	Michael J	Owen	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	owenmj@cardiff.ac.uk
ASD	Aarno	Palotie; MD; PhD	Sanger Institute	UK	ap8@SANGER.AC.UK
SCZ	Michele T	Pato	Keck School of Medicine; University of Southern California; Los Angeles; California; USA	USA	mpato@usc.edu
SCZ	Carlos N	Pato	Keck School of Medicine; University of Southern California; Los Angeles; California; USA	USA	pato@usc.edu
MDD; CDG	Brenda P	Penninx	VU University Medical Center; Amsterdam	The Netherlands	b.penninx@vumc.nl;
MDD	Michele L	Pergadia	University of Washington; St Louis	USA	michelep@wustl.edu
BPD	Hannes	Petursson	Department of Psychiatry; National University Hospital; Reykjavik; Iceland	Iceland	hannesp@lsh.is
BPD, SCZ	Benjamin S	Pickard	Division of Psychiatry, University of Edinburgh, Royal Edinburgh Hospital, Edinburgh, UK.; Medical Genetics Section, University of Edinburgh Molecular Medicine Centre, Western General Hospital, Edinburgh, UK.	UK	benjamin.pickard@strath.ac.uk
SCZ	Jonathan	Pimm, MD	University College London	UK	Jonathan.Pimm@eastlondon.nhs.uk
ASD	Joseph	Piven; MD	University of North Carolina	USA	jpiven@MED.UNC.EDU
BPD	Porgeir	Porgeirsson	deCODE Genetics, Sturlugata 8 101 Reykjavík, Iceland.	Iceland	
SCZ	Danielle	Posthuma	VU University; Center for Neurogenomics and Cognitive Research (CNCR); Department of Functional Genomics; VU Medical Centre; Department of Medical Genomics; Amsterdam; The Netherlands	The Netherlands	danielle.posthuma@cncr.vu.nl
BPD, MDD	James B	Potash	University of Iowa	USA	james-potash@uiowa.edu
BPD	Peter	Propping	Institute of Human Genetics, University of Bonn, Bonn, Germany.	Germany	
SCZ	Vinay	Puri, PhD	University College London	UK	puri.vinay@gmail.com
SCZ	Digby	Quested, MD	University of Oxford	UK	digby.quested@psych.ox.ac.uk
BPD	Emma M	Quinn	Neuropsychiatric Genetics Research Group; Department of Psychiatry and Institute of Molecular Medicine; Trinity College Dublin; Dublin; Ireland	Ireland	quinne5@tcd.ie
SCZ	Henrik B.	Rasmussen, DVM PhD	Institute of Biological Psychiatry, Sct. Hans Mental Health Center (MHS); Department of Clinical Medicine, University of Copenhagen; The Lundbeck Initiative for Integrative Psychiatric Research, iPSYCH; Denmark	DK	henrik.berg.rasmussen@regionh.dk
BPD	Soumya	Raychaudhuri	Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA.; Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	
ASD	Karola	Rehnström; PhD	Sanger Institute	UK	kr7@SANGER.AC.UK
ADHD, BPD	Andreas	Reif	Department of Psychiatry; Psychosomatics and Psychotherapy; University of Wuerzburg	Germany	reif_a@klinik.uni-wuerzburg.de
BPD	John	Rice	Washington University School of Medicine, St. Louis, Missouri, USA.	USA	
SCZ	Lizzy	Rossin			
ADHD	Aribert	Rothenberger, MD	University Medicine Goettingen/Child and Adolescent Psychiatry	Germany	arothen@gwdg.de
ASD	Guy	Rouleau; MD; PhD	University of Montreal	Canada	guy.rouleau@UMONTREAL.CA
BPD, SCZ	Douglas	Ruderfer	Division of Psychiatric Genomics, Department of Psychiatry, Mount Sinai School of Medicine, New York, New York, USA.	USA	
SCZ	Dan	Rujescu	Molecular and Clinical Neurobiology; Department of Psychiatry; Ludwig-Maximilians-University; Munich; Germany	Germany	Dan.Rujescu@med.uni-muenchen.de
SCZ	Alan R	Sanders	Department of Psychiatry and Behavioral Sciences; NorthShore University HealthSystem and University of Chicago; Evanston; Illinois; USA	USA	asanders@northshore.org
BPD	Martin	Schalling	Department of Molecular Medicine, Karolinska Institutet, Stockholm, Sweden.	Sweden	
BPD	Alan F	Schatzberg	Psychiatry and Behavioral Science, Stanford University School of Medicine, Palo Alto, California, USA.	USA	
BPD, MDD	William A	Scheftner	Rush University Medical Center	USA	william_a_scheftner@rush.edu
ASD	Gerard	Schellenberg; PhD	Perelman School of Medicine; University of Pennsylvania	USA	gerardsc@MAIL.MED.UPENN.EDU
BPD	Peter R	Schofield	Prince of Wales Medical Institute; Sydney; Australia.; University of New South Wales; Sydney; Australia.	Austrailia	
BPD	Nicholas J	Schork	The Scripps Translational Science Institute and The Scripps Research Institute, La Jolla, California, USA.	USA	
BPD	Johannes	Schumacher	Institute of Human Genetics, University of Bonn, Bonn, Germany.	Germany	
BPD	Markus M	Schwarz	Psychiatric Center Nordbaden; Wiesloch; Germany.	Germany	
BPD	Edward	Scolnick	Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA.	USA	scolnick@broadinstitute.org
BPD	Laura J	Scott	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.	USA	
MDD	Jianxin	Shi	National Cancer Institute	USA	jianxin.shi@nih.gov
SCZ	Jianxin	Shi	Division of Cancer Epidemiology and Genetics; National Cancer Institute; Bethesda; Maryland; USA	USA	jianxins@gmail.com
BPD	Paul D	Shilling	Neurosciences Centre of Excellence in Drug Discovery, GlaxoSmithKline Research and Development, Verona, Italy.	Italy	
MDD	Stanley I	Shyn	University of Washington	USA	stanshyn@uw.edu

BPD	Engilbert	Sigurdsson	Department of Psychiatry; National University Hospital; Department of Psychiatry; University of Iceland; Reykjavik; Iceland	Iceland	engilbs@landspitali.is
SCZ	Jeremy M.	Silverman, PhD	Mount Sinai School of Medicine / Bronx Veterans Affairs Medical Center	USA	jeremy.silverman@mssm.edu
BPD, SCZ	Pamela	Sklar	Division of Psychiatric Genomics, Department of Psychiatry, Mount Sinai School of Medicine, New York, New York, USA.	USA	
MDD	Susan L	Slager	Mayo Clinic	USA	slager.susan@mayo.edu
ADHD	Susan L.	Smalley; Ph.D.	University of California Los Angeles	USA	sue@suesmalley.com
MDD	Johannes H	Smit	VU University Medical Center; Amsterdam	The Netherlands	jh.smit@ggzingeest.nl
BPD	Erin N	Smith	The Scripps Translational Science Institute and Scripps Health, La Jolla, California, USA.	USA	
ADHD	Edmund	Sonuga-Barke PhD	Developmental Brain & Behaviour Laboratory, Psychology University of Southampton; Department of Experimental Clinical & Health Psychology, Ghent University	UK	ejb3@soton.ac.uk
BPD, SCZ	David	St. Clair	Institute of Medical Sciences; University of Aberdeen; Foresterhill; Aberdeen; Scotland; UK	UK	d.stclair@abdn.ac.uk
ASD	Matthew	State; MD; PhD	Yale University	USA	matthew.state@YALE.EDU
BPD	Kari	Stefansson	deCODE Genetics, Sturlugata 8 101 Reykjavík, Iceland.	Iceland	
BPD	Hreinn	Stefansson	deCODE genetics; Reykjavik; Iceland	Iceland	hreinn@decode.is
MDD; BPD	Michael	Steffens	Institute of Medical Biometry; Informatics and Epidemiology (IMBIE); University of Bonn; Bonn; Germany	Germany	michael.steffens@imbie.meb.uni-bonn.de
BPD	Stacy	Steinberg	deCODE genetics; Reykjavik; Iceland	Iceland	stacy.steinberg@decode.is
ADHD	Hans-Christoph	Steinhausen; MD; PhD; DMSc	REGION NORDJYLLAND, Aalborg University Hospital	Denmark	www.psykiatri.rn.dk
BPD	John	Strauss	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.	UK	
BPD	Jana	Strohmaier	Department of Genetic Epidemiology in Psychiatry, Central Institute of Mental Health Mannheim, University of Heidelberg, Mannheim, Germany.	Germany	
SCZ	T Scott	Stroup	Department of Psychiatry; Columbia University; New York; New York; USA	USA	stroups@pi.cpmc.columbia.edu
ASD	James	Sutcliffe; PhD	Vanderbilt University	USA	jim.sutcliffe@VANDERBILT.EDU
ASD	Peter	Szatmari; MD	McMaster University	Canada	szatmar@MCMASTER.CA
BPD	Szabocls	Szelinger	The Translational Genomics Research Institute, Phoenix, Arizona, USA.	USA	
SCZ	Srinivasa	Thirumalai, MD	West Berkshire NHS Trust/University College London	UK	docthiru@hotmail.com
BPD	Robert C	Thompson	Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, Michigan, USA.	USA	
BPD, MDD	Federica	Tozzi	GlaxoSmithKline	Italy	federica.tozzi1@tin.it
MDD; BPD	Jens	Treutlein	Department of Genetic Epidemiology in Psychiatry; Central Institute of Mental Health; University of Heidelberg; Mannheim; Germany	Germany	jens.treutlein@zi-mannheim.de
MDD	Manfred	Uhr	Max Planck Institute of Psychiatry; Munich	Germany	uhr@mpipsykl.mpg.de
MDD	Edwin JCG	van den Oord	Virginia Commonwealth University	USA	ejvandenoord@vcu.edu
MDD	Gerard	Van Grootheest	VU University Medical Center; Amsterdam	The Netherlands	g.vangrootheest@ggzingeest.nl
ASD	Veronica	Vieland; PhD	Nationwide Children's Hospital	USA	veronica.vieland@NATIONWIDECHILDRENS.ORG
BPD	John B	Vincent	Molecular Neuropsychiatry and Development Laboratory, Centre for Addiction and Mental Health, Toronto, Ontario, Canada.	Canada	
SCZ	Peter M.	Visscher	The University of Queensland, Queensland Brain Institute & UnivDiamantina Institute, Brisbane, Australia	Australia	peter.visscher@uq.edu.au
BPD	Stanley J	Watson	Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, Michigan, USA.	USA	
MDD	Myrna M	Weissman	Columbia University	USA	mmw3@columbia.edu
SCZ	Thomas	Werge	Institute of Biological Psychiatry; MHC Sct. Hans; Copenhagen University Hospital; Roskilde; Denmark	Denmark	thomas.werge@regionh.dk
BPD	Thomas F	Wienker	Institute for Medical Biometry, Informatics and Epidemiology, University of Bonn, Bonn, Germany.	Germany	
MDD	Gonneke	Willemsen	VU University	The Netherlands	ahm.willemsen@psy.vu.nl
BPD	Richard	Williamson	Social, Genetic and Developmental Psychiatry (SGDP) Centre, The Institute of Psychiatry, King's College London, De Crespigny Park Denmark Hill, London, UK.	UK	
BPD	Stephanie H	Witt	Department of Genetic Epidemiology in Psychiatry, Central Institute of Mental Health Mannheim, University of Heidelberg, Mannheim, Germany.	Germany	
MDD	Naomi R	Wray	The University of Queensland, Queensland Brain Institute, Brisbane, Australia	Australia	naomi.wray@uq.ed.au
BPD	Adam	Wright	School of Psychiatry; University of New South Wales and Black Dog Institute; Sydney; New South Wales; Australia.	Austrailia	
BPD	Wei	Xu	Dalla Lana School of Public Health, University of Toronto, Toronto, Canada.	Canada	
BPD	Allan H	Young	Division of Neuroscience, Medical Research Institute, University of Dundee, Ninewells Hospital & Medical School, Dundee, UK.; University of British Columbia (UBC) Institute of Mental Health, Vancouver, British Columbia, Canada.	UK: Canada	
SCZ	Stan	Zammit	MRC Centre for Neuropsychiatric Genetics and Genomics; Department of Psychological Medicine and Neurology; School of Medicine; Cardiff University; Cardiff; Wales; UK	UK	zammits@cardiff.ac.uk
BPD	Peter P	Zandi	Department of Mental Health, Johns Hopkins University and Hospital, Baltimore, Maryland, USA.	USA	

BPD	Peng	Zhang	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.; Department of Psychiatry, University of Michigan, Ann Arbor, Michigan, USA.	USA	
MDD	Frans G	Zitman	Leiden University Medical Center; Leiden	The Netherlands	f.g.zitman@lumc.nl
BPD	Sebastian	Zöllner	Department of Biostatistics and Center for Statistical Genetics, School of Public Health, University of Michigan, Ann Arbor, Michigan, USA.; Department of Psychiatry, University of Michigan, Ann Arbor, Michigan, USA.	USA	
CDG; BPD; SCZ	Nicholas	Craddock; MD#	Cardiff University School of Medicine	UK	craddockn@Cardiff.ac.uk
CDG; SCZ	Kenneth	Kendler; MD#	Virginia Institute for Psychiatric and Behavioral Genetics; Departments of Psychiatry and Human and Molecular Genetics; Virginia Commonwealth University School of Medicine; Richmond; Virginia; USA	USA	kendler@vcu.edu

* these individuals contributed equally

#these individuals contributed equally