Journal of Psychiatric Research 114 (2019) 178–207

Contents lists available at ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/jpsychires

Unravelling the genetic basis of schizophrenia and bipolar disorder with GWAS: A systematic review

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ARTICLE INFO

Keywords:
Genome-wide association study
Single nucleotide polymorphism
Biological psychiatry
Bipolar disorder
Schizophrenia

ABSTRACT

Objectives: To systematically review findings of GWAS in schizophrenia (SZ) and in bipolar disorder (BD); and to interpret findings, with a focus on identifying independent replications.
Method: PubMed search, selection and review of all independent GWAS in SZ or BD, published since March 2011, i.e. studies using non-overlapping samples within each article, between articles, and with those of the previous review (Li et al., 2012).
Results: From the 22 GWAS included in this review, the genetic associations surviving standard GWAS-significance were for genetic markers in the regions of *ACSL3/KCNE4*, *ADCY2*, *AMBRA1*, *ANK3*, *BRP44*, *DTL*, *FBLN1*, *HHAT*, *INTS7*, *LOC392301*, *LOC645434/NMBR*, *LOC729457*, *LRRFIP1*, *LSM1*, *MDM1*, *MHC*, *MIR2113/POU3F2*, *NDST3*, *NKAPL*, *ODZ4*, *PGBD1*, *RENBP*, *TRANK1*, *TSPAN18*, *TWIST2*, *UGT1A1/HJURP*, *WHSC1L1/FGFR1* and *ZKSCAN4*. All genes implicated across both reviews are discussed in terms of their function and implication in neuropsychiatry.
Conclusion: Taking all GWAS to date into account, *AMBRA1*, *ANK3*, *ARNTL*, *CDH13*, *EFHD1* (albeit with different alleles), *MHC*, *PLXNA2* and *UGT1A1* have been implicated in either disorder in at least two reportedly non-overlapping samples. Additionally, evidence for a SZ/BD common genetic basis is most strongly supported by the implication of *ANK3*, *NDST3*, and *PLXNA2*.

Summations

- The present systematic review found that *AMBRA1*, *ANK3*, *ARNTL*, *CDH13*, *EFHD1*, *MHC*, *PLXNA2* and *UGT1A1* have been associated with SZ or BD diagnosis in at least two independent samples (of either SZ or BD studies).
- The abovementioned genes are known to be involved in neurodevelopment, synaptic plasticity, maintenance of circadian rhythms, immune system function, and epigenetic regulation (at the cellular level), as well as impulsivity, seasonality and neurocognitive impairment (at the phenotypical level) – processes that have been previously associated with SZ or BD.

Considerations

- While the identified genes' function fit into current pathophysiological models of psychosis, their concomitant association with other neuropsychiatric disorders suggests shared pathogenetic mechanisms and challenges the diagnostic boundaries between these diseases, to which trans-diagnostic approaches might offer an advantage.
- Even though problems with sample heterogeneity and population stratification in terms of both known and unknown variables emerge in large sample sizes, GWAS are useful, with replication attempts being fundamental, and complementary genome-wide approaches, such as the polygenic score and the GCTA analyses, recommended.

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https://doi.org/10.1016/j.jpsychires.2019.04.007
Received 20 October 2018; Received in revised form 8 April 2019; Accepted 10 April 2019

1. Introduction

Schizophrenia (SZ) and bipolar disorder (BD) are severely debilitating mental disorders, affecting a combined 3% of the general population at least once in a life time (Kessler et al., 2005; Merikangas and Pato, 2009). Given previous twin (Sullivan et al., 2003) and family studies (Lichtenstein et al., 2009) pointing to their high heritability (~60–80%), the implications of elucidating the specific genetic variants that increase risk are vast. Candidate gene studies, i.e. based on *a priori* knowledge of a gene's function, or its position in genomic regions linked with SZ or BD through linkage studies, were the norm until about 2006; since the genome-wide exploration of potential risk gene variants by genome-wide association studies (GWAS) has taken over. The most obvious benefit of GWAS is its ability to detect risk variants regardless of previous hypotheses. This is particularly important in psychiatry, given the unconvincing and inconsistent evidence from candidate gene studies and a genetic architecture for most diseases that seems to be polygenic.

Lee et al. (2012a) conducted a systematic review of all GWAS published in either BD or SZ, since the first study in 2006 until March 2011. They reviewed a total of 14 single nucleotide polymorphism (SNP) and 5 copy-number variation (CNV) studies in BD, and 12 SNP and 23 CNV studies in SZ. They report genome-wide significant associations in ZNF804A, MHC region, NRGN, TCF4 for SZ, and in ANK3, CACNA1C, DGKH, PBRM1 and NCAN for BD. None of the findings of the BD studies were replicated at the time. Among SZ studies, two SNPs were identified twice: rs752016 in the *PLXNA2* gene (coding a neurodevelopmental-mediating receptor) (Mah et al., 2006; Sullivan et al., 2008) and rs1344706 in ZNF804A (coding a neurodevelopmental transcription factor) (O'Donovan et al., 2008; Williams et al., 2011a), although possible sample overlap had not been verified/disclosed in the review, and thus these results are not necessarily true replications. Regarding CNVs, there was replication of significant deletions involving *NRXN1* in 6 studies (SJ et al., 2008; Kirov et al., 2008a; Walsh et al., 2008; Rujescu et al., 2009; Ikeda et al., 2010; Vrijenhoek et al., 2008) and *NRG3*, *RAPGEF6*, *MYO38*, *GST1*, *GSTT2* and *VIPR2* in at least 2 studies (Xu et al., 2008, 2009; McCarthy et al., 2009; Need et al., 2009; Kirov et al., 2009; Rodriguez-Santiago et al., 2010; Vacic et al., 2011; Levinson et al., 2011). Some genes discussed in Lee et al. (2012a) were: 1) associated with both SZ and BD by GWA, giving credence to a long-alleged partially overlapping genetic basis in these disorders (Lee et al., 2012a); and 2) have accumulated early and suggestive evidence of impact on brain structure and function, such as *ANK3*, *CACNA1C*, *NRGN* and *ZNF804A*, plus in *TCF4* and *DGKH,* as we reviewed elsewhere (Gurung and Prata, 2015). However, given that GWAS identify several SNPs of small effect, using different statistical methodology, heterogeneous and overlapping sampling, it can be unclear how meaningful each individual GWAS result is. Hence, their findings should be revised and weighted with caution. The meta- and mega-analyses that more recently ensued from meta-consortia, although most powerful (by collating some of the studies reviewed), focus on the "global" significance; hence SNPs under study, that are associated with the disorders in specific populations, may be missed. There is obviously a trade-off between random hits in small studies and real hits in specific populations that do not reach genome-wide significance in a "global" sample, consistent with a genetic heterogeneity of SCZ and BD. Thus, a critical appraisal of the evidence supporting findings from smaller and more local studies (i.e. more homogeneous from ethnicity to clinical diagnosis practice), and, in particular, evidence of replication between them, is a timely need in the BD and SZ genetics field.

The present systematic review aims to follow on from Lee et al. (2012a)'s review by: 1) identifying and discussing all SNP GWAS findings published since March 2011; and 2) verifying replication consistency for all genetic variations implicated in both reviews, i.e., reported since January 2006. We particularly highlight new support for previously identified genes and replications for novel genes. We do this by thoroughly checking and excluding any sample overlap between any pair of studies across Lee et al. (2012a)'s and our present review – in order to protect this updated review from sampling biases or 'double-dipping' replications. Thus, we only highlight replications from independent samples. Additionally, we contextualize each genetic variant implicated by non-overlapping studies across both Lee et al. (2012a)'s review, and our review, in the current body of knowledge regarding protein function and role in the central nervous system and psychiatric pathologies. Complementarily, we also discuss recent (sample-overlapping) international meta-consortia findings.

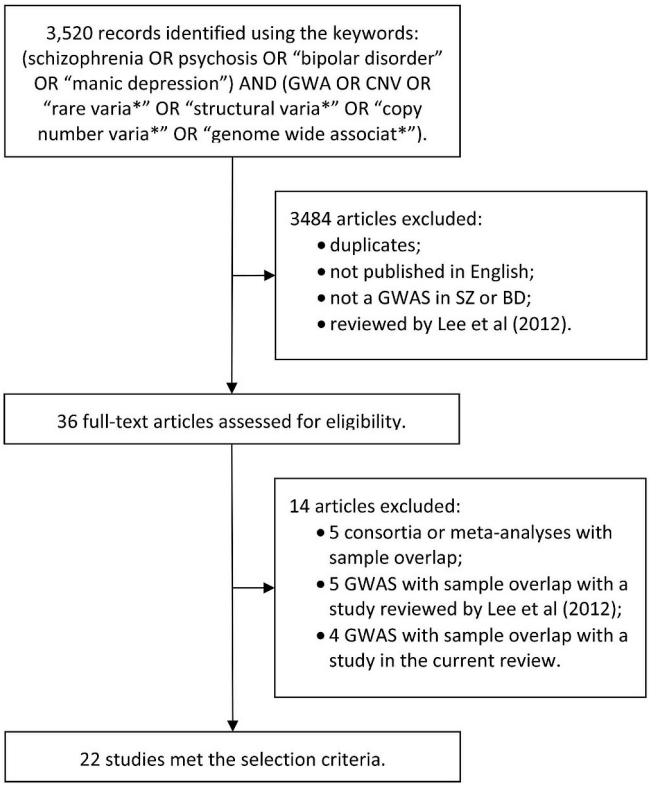

Fig. 1. Selection of studies.

2. Material and methods

We followed the PRISMA (Liberati et al., 2009) guidelines for systematic reviews to identify relevant studies for inclusion. Initially, we performed a PubMed search for all existing GWAS in SZ or BD published in English since the previous review by Lee et al. (2012a) (i.e., from March 2011) to 15th November 2016 (see Fig. 1 for details on search terms). Duplicates were removed using EndNote's native tool. To reduce the bias from duplicate sampling we excluded all: 1) meta-analyses/consortia including samples from studies in this or the previous review (although we address some of these large studies in the discussion); 2) GWAS with overlap in their discovery sample; 3) GWAS findings from replication analyses with sample overlap; and 4) GWAS findings from studies reviewed by Lee et al. (2012a) that were not using independent samples from studies already included.

We extracted all SNPs (and p-values) that survived the statistical significance threshold, defined by the authors, in both the discovery and replication analyses into the results table (Table 1). Thresholds for this method of cross-validation were also added to Table 1 to allow for a more systematic between-studies comparability. Throughout our review, we refer to 'associations' when results were statistically significant after correction for multiple comparisons (according to the threshold defined by each study's authors), and to 'trends' when reported by authors as such. Although trends are discussed in Results,

Table 1
GWAS in SZ and BD following those published in Lee et al. (2012a). Replication results are included in the row just below each study, when applicable. We report the odds ratio (OR) for the risk allele (even when it has been reported for the protective allele in the original paper; for easier comparability between studies). The sample sizes indicated are after quality control.

Author (Year)	Clinical phenotype	Ethnicity	Sample (cases; controls)	SNP	Closest gene	P-value*	Odds Ratio/ß (Risk Allele)	Platform	Threshold Notes
(Alkelai et al., 2011)	SZ	Arab-Israeli	58 families (71 cases)	rs12052937	LRRFIP1	1.2×10^{-11}	3.75 (A)	Illumina HumanCNV- 370	FDR $q < 5 \times 10^{-2}$
				rs4895576	LOC645434/NMBR	7.8×10^{-11}	1.33 (G)		
				rs10498146	ACSL3/KCNE4	7.4×10^{-10}	2.33 (A)		
				rs9751357	TWIST2	2.1×10^{-9}	2.60 (G)		
				rs741160	UGT1A1/HJURP	3.4×10^{-8}	1.17 (G)		
				rs6715815	LRRFIP1	4.0×10^{-8}	2.39 (G)		
				rs4278886	RAB17/LRRFIP1	3.3×10^{-7}	2.30 (A)		
				rs7578760	EFHD1	1.0×10^{-6}	3.33 (A)		
Replication	SZ	German	627; 541	rs7578760	EFHD1	1.0×10^{-2}	1.58 (opposite allele, G)	Illumina 300	Replication attempt if FDR $q < 5 \times 10^{-2}$. Then, $p < 5 \times 10^{-2}$
				rs741160	UGT1A1	3.0×10^{-2}	1.30 (same allele, G)		
(Alkelai et al., 2012)	SZ	Jewish- Israeli	107 families (155 cases)	rs2074127	DOCK4	1.1×10^{-7}	3.00 (N/R)	Illumina 370	Bonferroni correction, FDR $q < 5 \times 10^{-2}$
(Ikeda et al., 2011)	SZ	Japanese	575; 564	None	None	N/A	N/A	Affymetrix 5.0	$p < 7.2 \times 10^{-8}$
Replication	SZ	Japanese, UK	1511; 2451 (Jap.); 479; 2938 (UK)	None	None	N/A	N/A	UK: Affymetrix 500, Jap: Illumina 550, Sequenom	Replication attempt if several conditions were verified, totaling 97 SNPs. Then $p < 5 \times 10^{-3}$
(Shi et al., 2011)	SZ	Han Chinese	3750; 6468	None	None	N/A	N/A	Affymetrix 6.0	$p < 5 \times 10^{-8}$
Replication	SZ	Han Chinese	4383; 4539	rs1488935	WHSC1L1/FGFR1	3.4×10^{-5} (5.1×10^{-9})	1.15 (G) (N/R)	Ligation detection reaction (LDR)	Replication attempted if $p < 5 \times 10^{-6}$ (5 SNPs). Then $p < 5 \times 10^{-2}$
				rs16887244	LSM1	2.2×10^{-8} (1.3×10^{-10})	1.20 (A) (N/R)		
				rs10489202	BRP44	4.8×10^{-5} (9.5×10^{-9})	1.19 (A) (N/R)		
				rs1060041	DCAF6	1.2×10^{-2} (5.3×10^{-7})	1.11 (T) (NR)		
(Wang et al., 2011a)	SZ (onset age)	European-American	1162; 1378	rs7819815	ZFAT	3.1×10^{-7}	N/R	Affymetrix 6.0	$p < 5 \times 10^{-7}$
(Yamada et al., 2011)	SZ	Japanese	120 family trios (360)	None	None	N/A	N/A	Affymetrix 100	$p < 5.1 \times 10^{-7}$
Replication	SZ	Japanese	506; 506	None	None	N/A	N/A	Illumina	Replication attempted if $p < 1 \times 10^{-2}$ (1632 SNPs) + 473 if $p < 0.05$ and previously in linkage regions with SZ. Then $p < 5 \times 10^{-2}$, followed by Bonferroni
(Yosifova et al., 2011)	BD	Bulgarian	188; 376	rs1971058	PGAM1P1	9.9×10^{-8}	2.78 (N/R)	Illumina HumanHap-550	$p < 1 \times 10^{-7}$
Replication	BD	Bulgarian	122; 328	None	None	N/A	N/A	N/R	Attempted replication for 100 top SNPs. Then Bonferroni- corrected $p < 6 \times 10^{-4}$

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Table 1 (*continued*)

Author (Year)	Clinical phenotype	Ethnicity	Sample (cases; controls)	SNP	Closest gene	P-value*	Odds Ratio/ß (Risk Allele)	Platform	Threshold Notes
(Yue et al., 2011)	SZ	Han Chinese	746; 1599	rs10738881	LOC392301	5.3×10^{-9}	1.47 (T)	Illumina 610-Quad	$P < 1.01 \times 10^{-7}$
				rs2652007	LOC729457	2.4×10^{-9}	1.53 (G)		
Replication	SZ	Han Chinese	4027; 5603	rs1233710	*ZKSCAN4*	4.1×10^{-7} (4.8×10^{-11})	1.25 (C) (1.27)	Sequenom Mass-ARRAY system	Attempted replication if $p < 1 \times 10^{-5}$ (46 SNPs). Then, $p < 5 \times 10^{-2}$ No correction for multiple testing
				rs1635	*NKAPL*	5.5×10^{-8} (6.9×10^{-12})	1.27 (G) (1.28)		
				rs2142731	*PGBD1*	9.2×10^{-7} (5.1×10^{-10})	1.25 (G) (1.27)		
				rs11038167	*TSPAN18*	3.3×10^{-6} (1.0×10^{-11})	1.27 (A) (1.29)		
				rs11038172	*TSPAN18*	1.1×10^{-5} (7.2×10^{-10})	1.23 (A) (1.25)		
				rs835784	*TSPAN18*	2.4×10^{-7} (2.7×10^{-11})	1.25 (A) (1.27)		
(Bergen et al., 2012)	SZ & BD	Swedish	1507 (SZ) + 836 (BD); 2093	None	None	N/A	N/A	Affymetrix 6.0 & 5.0	$p < 5 \times 10^{-8}$
(Greenwood et al., 2012)	BD (5 tempera-ments)	European	1566; 1434	rs416350 (hyperthymic)	MDM1	4.1×10^{-8}	1.17 (A)	Affymetrix 6.0	$p < 5 \times 10^{-8}$
				rs1985671 (hyperthymic)	FBLN1	2.1×10^{-8}	1.02 (G)		
				rs739215 (hyperthymic)	FBLN1	4.3×10^{-8}	1.01 (G)		
				rs17018311 (irritable)	INTS7	1.7×10^{-8}	1.10 (T)		
				rs17018426 (irritable)	DTL	4.8×10^{-8}	1.16 (C)		
(Levinson et al., 2012)	SZ	Mostly European (& African, Sephardic Jewish & South Indian)	2461 SZ from 631 families	None	None	N/A	N/A	Illumina 610-Quad	$p < 5 \times 10^{-8}$
Irish Schizophrenia Genomics and the Wellcome Trust Case Control, 2012	SZ	Irish	1606; 1794	rs204999	MHC	2.8×10^{-8} (5.4×10^{-10})	1.33 (T) (N/R)	Affymetrix 6.0	$p < 1 \times 10^{-8}$
(Rietschel et al., 2012) §	SZ	European (Germany, Netherlands)	1169; 3714	None	None	N/A	N/A	Illumina HumanHap-550v3	$p < 5 \times 10^{-8}$ or Bonferroni $p < 1.1 \times 10^{-7}$
Replication 1	SZ	European (Germany, Netherlands, Denmark)	2569; 4088	rs11819869	AMBRA1	5.0×10^{-5} (3.9×10^{-9})	1.20 (T) (1.25)	N/R	Attempted replication of 43 top SNP. Then $p < 5 \times 10^{-2}$
				rs7112229	AMBRA1	5.3×10^{-5} (7.4×10^{-9})	1.21 (T) (1.25)		
				rs7130141	AMBRA1	6.5×10^{-5} (7.0×10^{-9})	1.20 (T) (1.24)		
				rs12574668	AMBRA1	7.2×10^{-5} (1.0×10^{-8})	1.20 (A) (1.24)		
				rs4309482	CCDC68/TCF4	2.9×10^{-4} (9.7×10^{-7})	1.14 (G) (1.15)		
				rs6465845	CUX1	6.4×10^{-3} (4.3×10^{-6})	1.11 (T) (1.17)		
				rs370760	CUX1	8.4×10^{-3} (2.7×10^{-5})	1.12 (A) (1.17)		
				rs404523	CUX1	1.0×10^{-2} (3.8×10^{-5})	1.11 (G) (1.17)		
				rs2717001	VRK2	2.0×10^{-2} (1.0×10^{-4})	1.08 (C) (1.12)		

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Table 1 (*continued*)

Author (Year)	Clinical phenotype	Ethnicity	Sample (cases; controls)	SNP	Closest gene	P-value*	Odds Ratio/ß (Risk Allele)	Platform	Threshold Notes
Replication 2	SZ	European	4734; 18,472	rs11819869	AMBRA1	2.9×10^{-3}	1.11 (T)	N/R	Attempted replication for the top GWAS SNP
(Betcheva et al., 2013)	SZ	Bulgarian	188; 376	None	None	N/A	N/A	Illumina Human550v3	Bonferroni corrected $p < 1.0 \times 10^{-7}$
Replication	SZ	Bulgarian	99; 328	rs7527939	HHAT	8.8×10^{-4} (6.5×10^{-9})	2.50 (C) (2.63)	N/R	Attempted replication for 100 top SNPs. Then, Bonferroni corrected $p < 1 \times 10^{-4}$
(Borglum et al., 2013)	SZ	Danish	888; 882	None	None	N/A	N/A	Illumina Human 610-quad	$P < 5 \times 10^{-8}$
				rs7902091	CTNNA3 x maternal CMV	7.3×10^{-7}	5.30 (A)		$P < 1.7 \times 10^{-6}$
Replication 1	SZ	Danish	1396; 1803	rs4757144	ARNTL	5.9×10^{-3} (3.8×10^{-6})	1.16 (G) (1.21)	Sequenom Mass-ARRAY	Attempted replication in 100 top SNPs. Then, Bonferroni corrected $p < 9.6 \times 10^{-7}$
				rs8057927	CDH13	7.1×10^{-3} (1.4×10^{-5})	1.32 (C) (1.44)		
Replication 2	SZ	German, Dutch	1169; 3714	rs4757144	ARNTL	1.0×10^{-1} (5.4×10^{-6})	1.08 (G) (1.15)	Illumina HumanHap-550v3	N/R
				rs8057927	CDH13	1.4×10^{-2} (1.2×10^{-6})	1.24(C) (1.34)		
				rs12922317	RUNDC2A	1.7×10^{-3} (9.0×10^{-7})	1.17 (G) (1.17)		
(Greenwood and Kelsoe, 2013)	BD (2 factor dimensions)	European	117 irritable mania; 843 elated mania; 1033 controls	None	None	N/A	N/A	Affymetrix 6.0	$p < 1 \times 10^{-4}$
Replication	BD (2 factor dimensions)	European	121 irritable mania; 1026 elated mania; 401 controls	None	None	N/A	N/A	Affymetrix 6.0	Attempted replication if $p < 1 \times 10^{-4}$ (62 SNPs). Then, $p < 5 \times 10^{-2}$
(Shibata et al., 2013)	SZ	Japanese	457; 457	None	None	N/A	N/A	Illumina GoldenGate assay	$p < 0.05$ (plus 3 sequential steps of screening using 3 independent sets of pooled samples)
Replication	SZ	Japanese	2224; 2250	rs13404754	SLC23A3	5×10^{-3}	N/R	Illumina GoldenGate assay	Attempted replication for top 31 SNPs. Then $p < 5 \times 10^{-2}$
				rs1043160	CNPPD1	1.1×10^{-2}	N/R		
				rs6436122	FAM134A	3.5×10^{-2}	N/R		
(Kanazawa et al., 2013)	Atypical psychosis	Japanese	47; 882	None	None	N/A	N/A	Affymetrix 6.0	$p < 5 \times 10^{-8}$
(Lencz et al., 2013)	SZ	Ashkenazi Jews	904; 1640	rs11098403	NDST3	6.6×10^{-9} (2.7×10^{-8})	1.41 (G) (1.15)	Illumina Human-Omni1-Quad	$p < 6.6 \times 10^{-8}$
(Muhleisen et al., 2014)	BD	European, German, Polish, Russian, Spanish, US- American	9747; 14278	rs10994415	ANK3	6.9×10^{-11}	1.27 (C)#	Illumina HumanHap-300, Illumina HumanHap-550, Illumina Human610-Quad, Illumina Human660-Quad, Illumina Human-Omni1-Quad	$p < 5 \times 10^{-8}$
				rs10994397	ANK3	2.9×10^{-10}	1.29 (T)#		
				rs9633553	ANK3	3.0×10^{-10}	1.29 (G)#		
				rs2154393	ANK3	3.0×10^{-10}	1.26 (T)#		
				rs1938540	ANK3	8.2×10^{-10}	1.27 (T)#		
				rs10821792	ANK3	8.3×10^{-10}	1.27 (T)#		
				rs1938526	ANK3	8.6×10^{-10}	1.27 (G)#		
				rs12412135	ANK3	3.3×10^{-9}	1.24 (T)#		
				rs10821789	ANK3	3.7×10^{-9}	1.24 (A)#		
				rs10994404	ANK3	4.4×10^{-9}	1.24 (C)#		
				rs10821745	ANK3	1.3×10^{-8}	1.27 (G)#		
				rs10821736	ANK3	1.6×10^{-8}	1.28 (T)#		
				rs10994430	ANK3	2.2×10^{-8}	1.18 (T)#		
				rs10994429	ANK3	2.2×10^{-8}	1.18 (T)#		
				rs10994336	ANK3	2.3×10^{-8}	1.27 (T)#		
				rs16915231	ANK3	2.5×10^{-8}	1.18 (A)#		

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Table 1 (*continued*)

Author (Year)	Clinical phenotype	Ethnicity	Sample (cases; controls)	SNP	Closest gene	P-value*	Odds Ratio/ß (Risk Allele)	Platform	Threshold Notes
				rs1380459	ANK3	2.7×10^{-8}	1.27 (T)#		
				rs4948412	ANK3	3.0×10^{-8}	1.27 (C)#		
				rs16915196	ANK3	3.0×10^{-8}	1.18 (G)#		
				rs10994322	ANK3	3.3×10^{-8}	1.27 (T)#		
				rs4948417	ANK3	3.4×10^{-8}	1.27 (G)#		
				rs10994338	ANK3	3.4×10^{-8}	1.26 (A)#		
				rs10994308	ANK3	3.6×10^{-8}	1.27 (A)#		
				rs3808943	ANK3	3.7×10^{-8}	1.27 (T)#		
				rs12416380	ANK3	3.8×10^{-8}	1.27 (G)#		
				rs10509129	ANK3	4.8×10^{-8}	1.29 (T)#		
				rs12290811	ODZ4	1.1×10^{-9}	1.19 (A)#		
				rs1944449	ODZ4	1.4×10^{-9}	1.19 (T)#		
				rs12576775	ODZ4	4.5×10^{-9}	1.17 (G)#		
				rs17138230	ODZ4	5.9×10^{-9}	1.17 (T)#		
				rs7932890	ODZ4	9.4×10^{-9}	1.17 (G)#		
				rs17138171	ODZ4	1.4×10^{-8}	1.16 (C)#		
				rs12279388	ODZ4	1.8×10^{-8}	1.16 (G)#		
				rs10501439	ODZ4	2.2×10^{-8}	1.17 (G)#		
				rs11237799	ODZ4	2.5×10^{-8}	1.16 (C)#		
				rs11237805	ODZ4	3.0×10^{-8}	1.16 (G)#		
				rs17826816	ADCY2	9.9×10^{-9}	1.14 (G)#		
				rs13166360	ADCY2	1.8×10^{-8}	1.14 (T)#		
				rs12202969	MIR2113/POU3F2	1.1×10^{-8}	1.12 (A)#		
				rs12206087	MIR2113/POU3F2	1.6×10^{-8}	1.12 (A)#		
				rs1906252	MIR2113/POU3F2	3.4×10^{-8}	1.12 (A)#		
				rs1487441	MIR2113/POU3F2	3.6×10^{-8}	1.12 (A)#		
				rs6550435	TRANK1	2.1×10^{-8}	1.13 (G)#		
				rs9882911	TRANK1	2.1×10^{-8}	1.13 (C)#		
				rs4678910	TRANK1	2.4×10^{-8}	1.13 (G)#		
				rs9821223	TRANK1	2.4×10^{-8}	1.13 (C)#		
				rs4234258	TRANK1	2.5×10^{-8}	1.13 (G)#		
				rs4624519	TRANK1	2.6×10^{-8}	1.13 (T)#		
				rs1532965	TRANK1	2.7×10^{-8}	1.12 (G)#		
				rs9811916	TRANK1	2.9×10^{-8}	1.12 (G)#		
				rs3732386	TRANK1	3.1×10^{-8}	1.12 (T)#		
				rs4678909	TRANK1	3.1×10^{-8}	1.12 (G)#		
				rs7652637	TRANK1	3.4×10^{-8}	1.12 (C)#		
				rs17807744	TRANK1	4.1×10^{-8}	1.12 (T)#		
				rs12637912	TRANK1	4.8×10^{-8}	1.12 (A)#		
				rs9834970	TRANK1	4.8×10^{-8}	1.12 (C)#		
(Wong et al., 2014)	SZ	Han Chinese	498; 2025	None	None	N/A	N/A	Illumina Human610-Quad, Illumina Human550	$p < 5 \times 10^{-8}$
Replication	SZ	Han Chinese	1027; 1005	rs2269372	RENBP	4.0×10^{-8}	1.31 (A)	Illumina GoldenGate Assay	Attempted replication of top 130 SNPs plus 254 candidate risk loci. Then $p < 1 \times 10^{-5}$.
(Goes et al., 2015)	SZ or SZA	Ashkenazi Jews	592; 505	None	None	N/A	N/A	Affymetrix 6.0	N/R

FOOTNOTE: *in parenthesis, p-values for the combined sample when provided; # risk allele is assumed to be author's 'allele-of-effect'. Acronyms used: BD – bipolar disorder; FDR – false discovery rate; GWAS – genome wide association study; N/A – not applicable; N/R – not reported; SNP – single nucleotide polymorphism; SZ – schizophrenia.

Table 2
Overview of gene regions associated with SZ or BD, both in Lee et al. (2012a)'s and the current review, with reference to their known role and their implications in the central nervous system and psychiatric or neurological pathophysiology or epidemiology. Genes and/or SNPs implicated (which are ticked) in more than one independent sample (either within or between study, or within or between illness), are highlighted in bold and underlined; with within- or between-study allele-direction consistencies or inconsistencies flagged. Any overlap in findings (markers and genes) between the studies (both from of our review and of Lee et al. (2012a)'s), including with the meta-consortia studies (with the important disclaimer that they, of course, overlap in their samples), is added a symbol whose meaning is made explicit in the footnote. P-values are reported when significant according to the original article. We report the odds ratio (OR) for the risk allele (even when it has been reported for the protective allele in the original paper; for easier comparability between studies).

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
ACSL3/ KCNE4	rs10498146	✓	✗	7.4×10^{-10} (D)	2.33 (D) (A)	(Alkelai et al., 2011)	ACSL3 codes for an enzyme that catalyses the conversion of long-chain fatty acids into fatty acyl-CoA esters (Yao and Ye, 2008). KCNE4 codes for the subunit of a voltage-gated potassium channel (Kv) that maintains its stability and modulates its gating kinetics (Vanoye et al., 2009).	ACSL3 plays a role in lipid biosynthesis and fatty acid degradation, which are required to maintain normal myelin structure (Wood et al., 2013). K^+ channels modulate DAergic neurons electrical excitability in the nigrostriatal and mesolimbic pathways and are potential pharmacological targets for psychosis treatment. Their function has also been related to synaptic plasticity in the hippocampus and cognitive performance (for a review see Imbrici et al. (Imbrici et al., 2013)).
ACSM1	rs433598	✓	✗	3.3×10^{-6} (D + R_1)	N/R (D + R_1)	(Athanasiu et al., 2010)	Enzyme that catalyses the degradation of medium-chain fatty acids for energy production (Fujino et al., 2001).	Polymorphisms in this gene contribute to multiple cardio-metabolic risk factors, which are known to be present in SZ patients (Mitchell et al., 2013).
ADAMTSL3	rs2135551	✓	✗	1.3×10^{-7} (D + R_1)	(N/R) (D + R_1)	(Need et al., 2009)	Member of the ADAMTS superfamily involved in ECM function and vascular homeostasis (Hall et al., 2003).	In the neural ECM the primary substrates of ADAMTS superfamily members are chondroitin sulfate proteoglycans (Hamel et al., 2008), which are overexpressed in the amygdala of SZ patients (Pantazopoulos et al., 2010). Thus, it probably has a role in the development and remodelling of the neuronal architecture of the brain, with a critical role in synaptogenesis and synaptic plasticity (Dow et al., 2011).
ADCY2	rs17826816	✗	✓	$9{,}9 \times 10^{-9}$ (D)	1.14 (D) (G)	(Muhleisen et al., 2014)	Enzyme that catalyses the conversion of ATP into the second messenger cAMP (Ding et al., 2004).	Predominantly expressed in limbic areas of the brain, particularly in AChergic cells of the striatum and GLUergic cells of the hippocampus (Hellevuo et al., 1993; Cote et al., 2001). Plays a role in spatial learning, memory (Mons et al., 2003) and mood (Porteous et al., 2006), and participates in AChergic receptor downstream signalling cascades (Shen et al., 2012). It has been previously implicated in SZ and BD (Kahler et al., 2010), and shown to be downregulated in the cortex of BD and MDD patients (Kwan et al., 2012; Higgs et al., 2006).
	rs13166360	✗	✓	1.8×10^{-8} (R_1)	1.14 (R_1) (T)			
AMBRA1§	**rs11819869**	✓✓	✗	5.0×10^{-5} (R_1) 3.9×10^{-9} (D + R_1) 2.9×10^{-3} (R_2)	1.20 (R_1) (T) 1.25 (D + R_1) (T) 1.11 (R_2) (T)	(Rietschel et al., 2012)	Autophagy and apoptosis regulator (Fimia et al., 2007).	Widely expressed in the brain, including the striatum and midbrain DAergic neurons (Van Humbeeck et al., 2011). Critical for autophagy-mediated clearance of ubiquinated products in the CNS (Fimia et al., 2007). Loss of its function can lead to neural tube defects (Fimia et al., 2007), and may contribute to PD (Van Humbeeck et al., 2011). The SZ-risk variant is also associated with impulsivity at a behavioural and neuroimaging level (Heinrich et al., 2013a).
	rs12574668	✓	✗	7.2×10^{-5} (R_1) 1.0×10^{-8} (D + R_1)	1.20 (R_1) (A) 1.24 (D + R_1) (A)			
	rs7112229	✓	✗	5.3×10^{-5} (R_1) 7.4×10^{-9} (D + R_1)	1.21 (R_1) (T) 1.25 (D + R_1) (T)			
	rs7130141	✓	✗	6.5×10^{-5} (R_1) 7.0×10^{-9} (D + R_1)	1.20 (R_1) (T) 1.24 (D + R_1) (T)			

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
ANK3[1]	**rs10994336**	✗	✓✓	9.1×10^{-9} (D + R_1) 2.3×10^{-8} (D + R_1)	1.45 (D + R_1) (T) 1.27 (D + R_1) (T)	(Ferreira et al., 2008) (Muhleisen et al., 2014)	Membrane-cytoskeleton linker (Maiweilidan et al., 2011).	Maintains the structure at the nodes of Ranvier and axonal initial segments required for action potential generation and propagation (Jenkins and Bennett, 2002). This gene has been implicated in the etiology of BD (Hayashi et al., 2015; Wirgenes et al., 2014; Lim et al., 2014), SZ (Lee et al., 2016), intellectual disability, autism (Bi et al., 2012) and stress-related disorders (Logue et al., 2013). ANK3-deficient mice exhibit manic-like behaviour, and treatment with lithium reverses these changes at a phenotypical and molecular level (Gottschalk et al., 2017). BD patients carrying the risk-variant, show cognitive deficits (Cassidy et al., 2014; Klionsky et al., 2016; Zhang et al., 2014; Ota et al., 2016), brain atrophy (Cassidy et al., 2014; Klionsky et al., 2016; Zhang et al., 2014; Ota et al., 2016), inability to suppress the default mode network (Delvecchio et al., 2015), and decreased connectivity of the facial affect-processing network (Dima et al., 2013). SZ-risk variant carriers also display brain structure changes and neurocognitive dysfunction (Cassidy et al., 2014; Klionsky et al., 2016; Zhang et al., 2014; Ota et al., 2016).
	rs10994415	✗	✓	6.9×10^{-11} (D + R_1)	1.27 (D + R_1) (C)			
	rs10994397	✗	✓	2.9×10^{-10} (D + R_1)	1.29 (D + R_1) (T)			
	rs9633553	✗	✓	3.0×10^{-10} (D + R_1)	1.29 (D + R_1) (G)			
	rs2154393	✗	✓	3.0×10^{-10} (D + R_1)	1.26 (D + R_1) (T)			
	rs1938540	✗	✓	8.2×10^{-10} (D + R_1)	1.27 (D + R_1) (T)			
	rs10821792	✗	✓	8.3×10^{-10} (D + R_1)	1.27 (D + R_1) (T)			
	rs1938526	✗	✓	8.6×10^{-10} (D + R_1)	1.27 (D + R_1) (G)			
	rs12412135	✗	✓	3.3×10^{-9} (D + R_1)	1.24 (D + R_1) (T)			
	rs10821789	✗	✓	3.7×10^{-9} (D + R_1)	1.24 (D + R_1) (A)			
	rs10994404	✗	✓	4.4×10^{-9} (D + R_1)	1.24 (D + R_1) (C)			
	rs10821745	✗	✓	1.3×10^{-8} (D + R_1)	1.27 (D + R_1) (G)			
	rs10821736	✗	✓	1.6×10^{-8} (D + R_1)	1.28 (D + R_1) (T)			
	rs10994430	✗	✓	2.2×10^{-8} (D + R_1)	1.18 (D + R_1) (T)			
	rs10994429	✗	✓	2.2×10^{-8} (D + R_1)	1.18 (D + R_1) (T)			
	rs16915231	✗	✓	2.5×10^{-8} (D + R_1)	1.18 (D + R_1) (A)			
	rs1380459	✗	✓	2.7×10^{-8} (D + R_1)	1.27 (D + R_1) (T)			
	rs4948412	✗	✓	3.0×10^{-8} (D + R_1)	1.27 (D + R_1) (C)			
	rs16915196	✗	✓	3.0×10^{-8} (D + R_1)	1.18 (D + R_1) (G)			
	rs10994322	✗	✓	3.3×10^{-8} (D + R_1)	1.27 (D + R_1) (T)			
	rs4948417	✗	✓	3.4×10^{-8} (D + R_1)	1.27 (D + R_1) (G)			
	rs10994338	✗	✓	3.4×10^{-8} (D + R_1)	1.26 (D + R_1) (A)			
	rs10994308	✗	✓	3.6×10^{-8} (D + R_1)	1.27 (D + R_1) (A)			
	rs3808943	✗	✓	3.7×10^{-8} (D + R_1)	1.27 (D + R_1) (T)			
	rs12416380	✗	✓	3.8×10^{-8} (D + R_1)	1.27 (D + R_1) (G)			
	rs10509129	✗	✓	4.8×10^{-8} (D + R_1)	1.29 (D + R_1) (T)			
	rs10761482	✓	✗	7.7×10^{-6} (D + R_1)	N/R (D + R_1)	(Athanasiu et al., 2010)		
ARNTL	**rs4757144**	✓✓	✗	5.9×10^{-3} (R_1) 3.8×10^{-6} (D + R_1) 1.0×10^{-1} (R_2) 5.4×10^{-6} (D + R_1+R_2)	1.16 (R_1) (G) 1.21 (D + R_1) (G) 1.08 (R_2) (G) 1.15 (D + R_1+R_2) (G)	(Borglum et al., 2013)	Transcriptional activator that is part of the genetic network that maintains circadian rhythms (Rudic et al., 2004).	Circadian disruptions are theorized to play a role in the etiology of SZ (Wulff et al., 2012), BD (Gonzalez, 2014), and neurodegenerative disorders (Videnovic et al., 2014). Polymorphisms in ARNTL have been associated with BD (Gonzalez et al., 2015), prophylactic response to lithium (Rybakowski et al., 2014), seasonal variation in mood and behaviour (Kim et al., 2015), and risk for AD (Chen et al., 2015a) and PD (Gu et al., 2015). ADHD patients show loss of rhythmic expression of ARNTL (Baird et al., 2012).

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
BRP44	rs10489202	✓	✗	4.8×10^{-5} (R_1) 9.5×10^{-9} (D + R_1)	1.19 (R_1) (A) N/R (D + R_1)	(Shi et al., 2011)	Participates in the citric acid cycle by mediating the uptake of pyruvate into mitochondria (Bricker et al., 2012).	Unknown.
CACNA1C	rs1006737	✗	✓	7.0×10^{-8} (D + R_1)	1.18 (D + R_1) (A)	(Ferreira et al., 2008)	α-1 subunit of a voltage-dependent Ca^{2+} channel that mediates the cellular influx of Ca^{2+} for Ca^{2+}-dependent processes, including cell survival, NT release, synaptic plasticity and gene expression (Sinnegger-Brauns et al., 2009).	Variation within CACNA1C may be associated with BD via CNS changes at a molecular (reduced synaptic plasticity (Moosmang et al., 2005) and adult neurogenesis (Lee et al., 2016), disrupted MAPK and CREB signalling (Moosmang et al., 2005), and decreased levels of BDNF in hippocampal neurons (Lee et al., 2016); modulation of the cellular rhythm amplitude response to lithium (McCarthy et al., 2016)), structural (reduced total gray matter volume (Kempton et al., 2009); regional differences in the volume of amygdala (Perrier et al., 2011; Wolf et al., 2014), and hypothalamus (Perrier et al., 2011); reduced frontotemporal gray matter and functional connectivity (Wang et al., 2011b)), neurochemical (decreased cerebrospinal fluid levels of markers of neuroaxonal plasticity (Jakobsson et al., 2016)), cognitive (impaired attention (Thimm et al., 2011), working memory (Zhang et al., 2012), executive function (Soeiro-de-Souza et al., 2013), learning and memory (Moosmang et al., 2005), verbal fluency (Krug et al., 2010), and facial emotion recognition (Soeiro-de-Souza et al., 2012)), and behavioural level (amygdala-mediated fear conditioning (Shinnick-Gallagher et al., 2003); blunted reward responsiveness (Lancaster et al., 2014); affective personality traits (Dao et al., 2010); and schizotipy (Roussos et al., 2013)). Gene association studies have also implicated this gene in SZ (Green et al., 2010; Nyegaard et al., 2010), MDD (Green et al., 2010), autism (Li et al., 2015), epilepsy (Lv et al., 2015a), and AD (Daschil et al., 2013).
CCDC60	rs11064768	✓	✗	1.2×10^{-6} (D)	N/R (D)	(Kirov et al., 2008b)	Unknown.	Unknown.
CDH13	**rs8057927**	✓✓	✗	7.1×10^{-3} (R_1); 1.4×10^{-5} (D + R_1); 1.4×10^{-2} (R_2); 1.2×10^{-6} (D + R_1+R_2)	1.32 (R_1) (C); 1.44 (D + R_1) (C); 1.24 (R_2) (C); 1.34 (D + R_1+R_2) (C)	(Borglum et al., 2013)	Neuronal membrane adhesion protein and signalling molecule (Rivero et al., 2013).	May act as a negative regulator of neurite outgrowth and axon guidance required for development and synaptic plasticity (Rivero et al., 2013). Contributes to deficits in impulse control, as shown by polymorphisms linking violent behaviour (Takeuchi et al., 2000), hyperactivity/impulsivity and impaired working memory in ADHD (Salatino-Oliveira et al., 2015; Arias-Vasquez et al., 2011), as well as alcohol (Treutlein et al., 2009) and (met)amphetamine (Hart et al., 2012; Uhl et al., 2008) addiction.
CNPPD1	rs1043160	✓	✗	1.1×10^{-2} (D + R_1)	N/R (D + R_1)	(Shibata et al., 2013)	Unknown	Unknown.

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
CSF2RA[b]	rs4129148	✓	✗	3.7×10^{-7} (D)	3.23 (D) (CC)	(Lencz et al., 2007)	Cytokin receptor for granulocyte-macrophage colony-stimulating factor (GM-CSF) (Ridwan et al., 2012).	GM-CSF is a haemopoietic growth factor and proinflammatory mediator implicated in several autoimmune and inflammatory diseases, including MS (van Nieuwenhuijze et al., 2013). It induces the proliferation and activation of microglial cells, which secondarily promote oxidative stress and GLU neurotoxicity (Ponomarev et al., 2007). It is also involved in BBB disruption (King et al., 2009). GM-CSF inhibitors have been developed for the treatment of inflammatory conditions (van Nieuwenhuijze et al., 2013). To the authors' knowledge, there are no ongoing SZ clinical trials with GM-CSF inhibitors, but there are trials for drugs that act on related molecular pathways: Natalizumab (NCT03093064), Tocilizumab (NCT01696929) and Siltuximab (NCT02796859).
CTNNA3	rs7902091	✓	✗	7.3×10^{-7} ($D + R_1$)	5.30 ($D + R_1$) (A)	(Borglum et al., 2013)	Mediator of cell-cell adhesion and cytoskeleton integrity (Janssens et al., 2001).	It has been proposed as a risk factor for late-onset AD (Morgan et al., 2008), but this effect may be dependent of APOE-4 (Miyashita et al., 2007) or of a female-specific mechanism (Martin et al., 2005). Increases susceptibility to autism (Bacchelli et al., 2014) and TS (Clarke et al., 2012).
CUX1[§]	rs370760	✓	✗	8.4×10^{-3} (R_1) 2.7×10^{-5} ($D + R_1$)	1.12 (R_1) (A) 1.17 ($D + R_1$) (A)	(Rietschel et al., 2012)	Transcription factor involved in the regulation of cellular proliferation and differentiation (Zhang et al., 2012).	Regulates dendritogenesis and synaptogenesis in upper cortical layers during development (Cubelos et al., 2010). Abnormal function in mice correlates with synaptic dysfunction and cognitive deficits (Cubelos et al., 2010). It has been associated with BD (Glaser et al., 2005), treatment-resistant MDD (Sasayama et al., 2013), and autism (Choi et al., 2012).
	rs404523	✓	✗	1.0×10^{-2} (R_1) 3.8×10^{-5} ($D + R_1$)	1.11 (R_1) (G) 1.17 ($D + R_1$) (G)			
	rs6465845	✓	✗	6.4×10^{-3} (R_1) 4.3×10^{-6} ($D + R_1$)	1.11 (R_1) (T) 1.17 ($D + R_1$) (T)			
DCAF6	rs1060041	✓	✗	1.2×10^{-2} (R_1) 5.3×10^{-7} ($D + R_1$)	1.11 (T) (N/R) ($D + R_1$)	(Shi et al., 2011)	Ligand-dependent coactivator of nuclear receptors (Tsai et al., 2005).	Unknown.
DGKH	rs1012053	✗	✓	1.5×10^{-8} ($D + R_1$)	1.59 ($D + R_1$) (A)	(Baum et al., 2008a)	Enzyme that catalyses the metabolization of diacylglycerol (DAG), which activates protein kinase C (PKC) (Murakami et al., 2003).	Glucocorticoid-inducible and stress-responsive gene highly expressed in the healthy brain (Murakami et al., 2003; Klauck et al., 1996), and overexpressed in BD (Moya et al., 2010). DGKH and lithium modulate PKC signalling (Chen et al., 2000; Kittel-Schneider et al., 2015), and both increase amygdala volume (Kittel-Schneider et al., 2015; Hallahan et al., 2011). PKC signalling is suspected to play a role in the pathophysiology of BD, corroborated by the anti-manic effects of PKC inhibitors in animal models. Together, these findings implicate PKC signalling as the common molecular mechanism mediating the effects of genetic variation and lithium treatment in amygdala structure. DGKH risk variant carriers with positive family history of BD also show differential brain activity within the left medial frontal gyrus, left precuneus, and right parahippocampus gyrus during a verbal fluency task, which may reflect a failure to disengage default-mode regions (Whalley et al., 2012). Gene variation is also associated with MDD and ADHD (Weber et al., 2011).

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
DLEU2	rs1750565 rs1750567 rs1798968	✗ ✗ ✗	✓ ✓ ✓	2.4×10^{-5} (D) 2.2×10^{-5} (D) 2.7×10^{-5} (D)	1.73 (D) (A) 1.75 (D) (A) 1.74 (D) (C)	(Djurovic et al., 2010)	Long non-coding RNA (Corcoran et al., 2004).	Unknown.
DOCK4	rs2074127	✓	✗	1.1×10^{-7} (D)	3.00 (D) (N/R)	(Alkelai et al., 2012)	Regulator of cell-cell adhesion and Wnt/β- catenin pathway signalling (Yajnik et al., 2003).	Plays a role in axonal patterning and neurite differentiation during development (Biersmith et al., 2011; Xiao et al., 2013), with its deficiency leading to reduced dendritic growth and branching in hippocampal neurons (Miyamoto and Yamauchi, 2010). This role is further supported by its association with autism (Liang et al., 2014), TS (Petek et al., 2001) and dyslexia (Pagnamenta et al., 2010).
DTL	rs17018426	✗	✓	4.8×10^{-8} (D)	1.16 (D) (C)	(Greenwood et al., 2012)	Component of a complex that maintains genomic stability after DNA damage (Higa et al., 2006; Sansam et al., 2006; Terai et al., 2010).	Unknown.
EFHD1	**rs7578760**	✓✓	✗	1.0×10^{-6} (D); 1.0×10^{-2} (R)	3.33 (D) (A, arab-israeli); 1.58 (R) (G, german)	(Alkelai et al., 2011)	Ca^{2+}-sensor for mitochondrial flash activation (Hou et al., 2016b).	May participate in neuronal differentiation (Tominaga and Tomooka, 2002).
FAM134A	rs6436122	✓	✗	3.5×10^{-2} (D + R_1)	N/R (D + R_1)	(Shibata et al., 2013)	Unknown.	Unknown.
FBLN1	rs1985671 rs739215	✗ ✗	✓ ✓	2.1×10^{-8} (D) 4.3×10^{-8} (D)	1.02 (D) (T) 1.01 (D) (A)	(Greenwood et al., 2012)	Extracellular glycoprotein that plays a role in cell adhesion and motility along fibers within the ECM (Twal et al., 2001).	Required for morphogenesis of neural crest-derived structures (Cooley et al., 2008). A missense mutation causes a syndrome of delayed motor milestones, mental retardation, brain atrophy, cryptorchidism and syndactyly (Bohlega et al., 2014).
GUCY1B2	rs11617400	✗	✓	3.7×10^{-5} (D)	1.8 (D) (C)	(Djurovic et al., 2010)	Subunit of an enzyme activated by nitric oxide that catalyses the conversion of GTP to the second messenger cGMP (Reierson et al., 2011).	The cGMP signalling cascade is expressed in the brain and is involved in dendrite formation, axon guidance, neuroplasticity and neurogenesis, as well as stress induced disturbance of neuroplasticity, MDD and antidepressant effects (Reierson et al., 2011).
HHAT	rs7527939	✓	✗	8.8×10^{-4} (R_1) 6.5×10^{-9} (D + R_1)	2.50 (R_1) (C) 2.63 (D + R_1) (C)	(Betcheva et al., 2013)	Endoplasmic reticulum enzyme that catalyses the post-transcriptional modification of sonic hedgehog (SHH) (Chamoun et al., 2001).	Controls the activity of SHH, a secreted glycoprotein that promotes the migration of neuronal precursors along the neural tube and their differentiation into DAergic neurons (Bohlega et al., 2014; Perrone-Capano and Di Porzio, 2000). It also resides in a genetic linkage region for SZ (Hovatta et al., 1999).
HIST1H2AG[a]	rs6913660	✓	✗	2.4×10^{-8} (D + R_1) 1.1×10^{-9} (D + R_1)	N/R (D + R_1) 1.15 (D + R_1) (C)	(Shi et al.. 2009) (Stefansson et al., 2009)	Core component of the nucleosome (Zhang et al., 2004).	The nucleosome is a major component of epigenetic regulation, which is hypothesized to mediate variation in gene expression within the CNS (Deutsch et al., 2008; Akbarian and Huang, 2009; Roth et al., 2009). Histone modification, in particular, is an epigenetic mechanism implicated in the pathogenesis of SZ (Gavin and Sharma, 2010) and BD (Ludwig and Dwivedi, 2016).
INTS7	rs17018311	✗	✓	1.7×10^{-8} (D)	1.10 (D) (T)	(Greenwood et al., 2012)	RNA processing protein involved in the DNA damage response (Cotta-Ramusino et al., 2011).	Unknown.
JAM3	rs10791345	✗	✓	1.0×10^{-6} (D + R_1)	1.25 (D + R_1) (G)	(Baum et al., 2008b)	Component of tight junctions involved in cell-cell adhesion and signal transduction (Bazzoni, 2003).	Tight junctions are a core component of the BBB in the cerebrovascular endothelium (Mochida et al., 2010). Functional mutations in JAM3 can result in massive haemorrhagic stroke (Mochida et al., 2010).
JARID2	rs2235258	✓	✗	8.7×10^{-3} (D + R_1)	1.88 (D + R_1) (N/R)	(Liu et al., 2009)	DNA-binding protein that functions as a transcriptional repressor (Takeuchi et al., 1999).	This gene is essential for neural tube formation (Liu et al., 2009), with its variation leading to congenital neural tube defects (Volcik et al., 2004). This SNP lies in the same LD block as *DTNBP1* (dysbindin), also associated with SZ (Riley et al., 2009).

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
LOC392301	rs10738881	✓	✗	5.3×10^{-9} (D)	1.47 (D) (T)	(Yue et al., 2011)	Unknown.	Unknown.
LOC729457	rs2652007	✓	✗	2.4×10^{-9} (D)	1.53 (D) (G)	(Yue et al., 2011)	(Withdrawn gene record.)	Unknown
LRRFIP1	rs12052937	✓	✗	1.2×10^{-11} (D)	3.75 (D) (A)	(Alkelai et al., 2011)	Transcriptional repressor of EGFR, PDGFA, and TNF, Toll-like receptor and Wnt/β-catenin signalling pathways (Khachigian et al., 1999; Suriano et al., 2005).	EGFR and PGDFA are growth factors required for oligodendrogenesis and myelination (Galvez-Contreras et al., 2013; Funa and Sasahara, 2014), which is thought to be disturbed in SZ (Chang et al., 2007). TNF, Toll-like receptor and Wnt signalling pathways are key regulators of immune processes, and play essential functions in the CNS, including neurogenesis, synaptic plasticity and response to neuronal damage (Beattie et al., 2002a, 2002bBeattie et al., 2002a; Pan et al., 1997; Stellwagen and Malenka, 2006Beattie et al., 2002b; Panaccione et al., 2013). These signalling pathways have been implicated in SZ and BD (Panaccione et al., 2013; Lv et al., 2015b; Hoseth et al., 2017; Venkatasubramanian and Debnath, 2013), providing support the hypothesis of neuroimmune dysfunction in SZ and BZ (Whalley et al., 2012).
	rs6715815	✓	✗	4.0×10^{-8} (D)	2.39 (D) (G)			
	rs4278886	✓	✗	3.3×10^{-7} (D)	2.30 (D) (A)			
LSM1	rs16887244	✓	✗	2.2×10^{-8} (R_1) 1.3×10^{-10} (D + R_1)	1.20 (R_1) (A) (N/R) (D + R_1)	(Shi et al., 2011)	RNA-binding protein involved in the degradation of mRNA (Mullen and Marzluff, 2008).	Unknown.
MBOAT1	rs16883399	✗	✓	6.8×10^{-5} (D + R_1)	N/R (D + R_1)	(Belmonte Mahon et al., 2011)	Enzyme involved in arachidonic acid (AA) recycling (Gijon et al., 2008).	AA-derived eicosanoids regulate immune and inflammatory responses and have recently emerged as key players in neuropsychiatric disorders (for a review, see Yui et al. (Yui et al., 2015)).
MDM1	rs416350	✗	✓	4.1×10^{-8} (D)	1.17 (D) (A)	(Greenwood et al., 2012)	Microtubule-binding protein that negatively regulates centriole duplication (Van de Mark et al., 2015).	Unknown.
MHC[#, e]	rs204999	✓	✗	2.8×10^{-8} (D) 5.4×10^{-10} (D + R_1)	1.33 (D) (T) (N/R) (D + R_1)	(Irish Schizophrenia Genomics and the Wellcome Trust Case Control, 2012)	Family of surface proteins that play a central role in the immune system by presenting antigen-derived peptides for recognition by $CD4^+$ T lymphocytes (Shiina et al., 2009).	The MHC region has long been suspected to play a role in SZ, giving way to complex models of immune-CNS interactions (review in (Mokhtari and Lachman, 2016)).
	rs13211507	✓	✗	8.3×10^{-11} (D + R_1)	1.24 (D + R_1) (T)	(Stefansson et al., 2009)		
	rs13219354	✓	✗	1.3×10^{-10} (D + R_1)	1.20 (D + R_1) (T)			
	rs3131296	✓	✗	2.3×10^{-10} (D + R_1)	1.19 (D + R_1) (G)			
	rs6932590	✓	✗	1.4×10^{-12} (D + R_1)	1.16 (D + R_1) (T)			
MIR2113/ POU3F2	rs12202969	✗	✓	1.1×10^{-8} (D + R_1)	1.12 (D + R_1) (A)	(Muhleisen et al., 2014)	*MIR2113* codes for an on-coding RNA involved in post-transcriptional regulation of gene expression (Davies et al., 2015).	*MIR2113* is associated with general cognitive functioning (Davies et al., 2015) and education attainment (Rietveld et al., 2013).
	rs12206087	✗	✓	1.6×10^{-8} (D + R_1)	1.12 (D + R_1) (A)			
	rs1906252	✗	✓	3.4×10^{-8} (D + R_1)	1.12 (D + R_1) (A)		*POU3F2* codes for a member of the POU-III class of neural transcription factors (Atanasoski et al., 1995).	*POU3F2* plays an important role in the development and function of the hypothalamus, and possibly participates in the neuroendocrine control of energy balance and body mass (Kasher et al., 2016). CNV deletions of the 6q16.1 region encompassing *POU3F2* causes a syndrome with developmental delay, intellectual disability, and susceptibility to obesity and hyperphagia (Kasher et al., 2016).
	rs1487441	✗	✓	3.6×10^{-8} (D + R_1)	1.12 (D + R_1) (A)			
MYO18B	rs5761163	✓	✗	3.4×10^{-7} (D)	1.25 (D) (A)	(International Schizophrenia et al., 2009)	Member of a family of unconventional myosins that regulates muscle-specific genes when in the nucleus, while it influences intracellular trafficking when in the cytoplasm (Ajima et al., 2008).	Its specific role in the CNS is unknown, but myosins are known to be involved in axonal transport (Bridgman, 2004). A SNP was associated with mathematical disability and reduced volume of the right intraparietal sulcus in the parietal cortex (a key structure involved in numerical processing) of dyslexic children (Ludwig et al., 2013).

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
MYO5B	rs4939921	✗	✓	1.7×10^{-7} (D)	1.51 (D) (G)	(Sklar et al., 2008)	Member of a family of unconventional myosins involved in vesicular trafficking (Szperl et al., 2011).	Participates in vesicle trafficking in neurons, a mechanism whereby it regulates EGFR cycling (another family of proteins implicated in the pathophysiology of BD). Confers susceptibility for dyslexia (Mueller et al., 2014).
NCAN	rs1064395	✗	✓	3.0×10^{-8} (D + R_1)	1.31 (D + R_1) (A)	(Cichon et al., 2011)	Brain-specific extracellular matrix glycoprotein involved in cell adhesion and migration (Cichon et al., 2011).	Modulates neuronal adhesion and neurite growth and influences cortical folding during development (Rauch et al., 2001; Schultz et al., 2014; Avram et al., 2014). It is expressed in subcortical brain areas involved in emotional processing, including the amygdala, hippocampus, and orbitofrontal cortex (Cichon et al., 2011). Gene variation affects gray matter volume in these structures independent of disease, suggesting it might confer increased risk for BD via neurostructural deficits (Dannlowski et al., 2015). A case-control association study has also implicated this gene in SZ (Muhleisen et al., 2012).
NDST3[b]	rs11098403	✓	✗	6.6×10^{-9} (D) 2.7×10^{-8} (D + R_1)	1.41 (D) (G) 1.15 (D + R_1) (G)	(Lencz et al., 2013)	Enzyme required for the biosynthesis of heparan sulfate (HS) (Aikawa and Esko, 1999).	This gene is highly expressed in the hippocampus (Lein et al., 2007), where it might have a regulatory function (Lencz et al., 2013), and in the cerebellum (Lein et al., 2007). Indeed, structural and functional abnormalities of the hippocampus and the cerebellum have been demonstrated in SZ (Ganzola et al., 2014; Knable et al., 2004; Liu et al., 2011) and BD (Roda et al., 2015; Johnson et al., 2015). Its function in HS metabolism appears to be critical for neurite outgrowth, axon guidance and synaptogenesis (Irie et al., 2008; Inatani et al., 2003; Lucido et al., 2009). The pattern of HS sulfation determines its binding affinity to *NRG1*, which is also implicated in SZ and BD (see below).
NKAPL	rs1635	✓	✗	5.5×10^{-8} (R_1); 6.9×10^{-12} (D + R_1)	1.27 (R_1) (G); 1.28 (D + R_1) (G)	(Yue et al., 2011)	Transcriptional repressor of Notch-mediated signalling located within the MHC region (Okuda et al., 2015).	Abundantly expressed in the mice cortex, hippocampus, ventral lateral nucleus, locus coeruleus (Yue et al., 2011). *NAKPL* knockout causes impaired neuronal migration and synaptic defects in animal models, suggesting a role in neurodevelopment (Yue et al., 2011).
NMBR	rs4895576	✓	✗	7.8×10^{-11} (D)	1.33 (D) (G)	(Alkelai et al., 2011)	Transmembrane G protein-coupled receptor that binds to the bombesin-like peptide family member neuromedin B (NMB) (Minamino et al., 1983).	The NMB/NMBR pathway contributes to behavioural homeostasis by regulating feeding behaviour (Flood and Morley, 1988; McCoy and Avery, 1990) and thermoregulation (He et al., 2006). It is also implicated in spontaneous activity, susceptibility to stress and fear/anxiety (Gonzalez et al., 2008; Yamada et al., 2002), the latter possibility due to changes in serotoninergic transmission in ventral hippocampal neurons (Merali et al., 2006). Moreover, blocking this pathway suppresses dopamine agonist-induced effects in mice (Meller et al., 2004). Bombesin-like peptides have also been associated autism (Ishikawa-Brush et al., 1997), although the pathophysiological mechanism is unclear.

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
NRG1	rs221533	✓	✗	9.1×10^{-4} (D + R_1)	N/R (D + R_1)	(Sullivan et al., 2008)	Member of epithelial growth factor family that mediates cell-cell signalling via binding to ErbB tyrosine kinase receptors (Falls, 2003).	Regulates developmental neuronal survival, synaptogenesis, myelin formation, astrocytic differentiation, and microglial activation (Falls, 2003; Britsch, 2007; Basak et al., 2015). Plays a protective role in the injured CNS (Tokita et al., 2001), particularly in DAergic neurons (Carlsson et al., 2011). When neuregulin-1 is peripherally administered in neonatal mice, it activates ErbB4 and leads to a persistent hyperdopaminergic state (Carlsson et al., 2011). In adult rats, improves functional recovery when given before or immediately after ischemic brain injury (Xu et al., 2006). It is implicated in AD (Chaudhury et al., 2003), PD (Carlsson et al., 2011), TLE (Zhu et al., 2016), cognitive performance in BD (Rolstad et al., 2015) and SZ (Cho et al., 2015), and in the OFC sulcogyral patterns of SZ patients (Yoshimi et al., 2016).
NRGN[#]	rs12807809	✓	✗	2.4×10^{-9} (D + R_1)	1.15 (D + R_1) (T)	(Stefansson et al., 2009)	Postsynaptic substrate of protein kinase C-mediated molecular cascades (Prichard et al., 1999).	*NRGN* is expressed in brain areas associated with cognitive functioning, and plays a role in cortical development, with its expression being reduced in the ACC and DLPFC of SZ patients (Walton et al., 2013). Affects signalling cascades downstream of glutamatergic NMDA receptors, which are postulated to be hypofunctioning in SZ and related to cognitive deficits (Tsai and Coyle, 2002). Biological fluid levels of neurogranin are candidate biomarkers for neuronal damage in TBI (Yang et al., 2015) and AD (Hellwig et al., 2015). This gene may also be a mediator of thyroid hormone effects in the brain (Martinez de Arrieta et al., 1999).
ODZ4[!]	rs12290811	✗	✓	1.1×10^{-9} (D + R_1)	1.19 (D + R_1) (A)	(Muhleisen et al., 2014)	Transmembrane protein involved in cell surface signalling and neuronal pathfinding (Hor et al., 2015).	Expressed predominantly in neurons, particularly in the white matter of the cerebellum (Hor et al., 2015). Plays a key role in oligodendrocytogenesis and axonal guidance (Hor et al., 2015). The risk variant influences reward processing in the amygdala (Heinrich et al., 2013b).
	rs1944449	✗	✓	1.4×10^{-9} (D + R_1)	1.19 (D + R_1) (T)			
	rs12576775	✗	✓	4.5×10^{-9} (D + R_1)	1.17 (D + R_1) (G)			
	rs17138230	✗	✓	5.9×10^{-9} (D + R_1)	1.17 (D + R_1) (T)			
	rs7932890	✗	✓	9.4×10^{-9} (D + R_1)	1.17 (D + R_1) (G)			
	rs17138171	✗	✓	1.4×10^{-8} (D + R_1)	1.16 (D + R_1) (C)			
	rs12279388	✗	✓	1.8×10^{-8} (D + R_1)	1.16 (D + R_1) (G)			
	rs10501439	✗	✓	2.2×10^{-8} (D + R_1)	1.17 (D + R_1) (G)			
	rs11237799	✗	✓	2.5×10^{-8} (D + R_1)	1.16 (D + R_1) (C)			
	rs11237805	✗	✓	3.0×10^{-8} (D + R_1)	1.16 (D + R_1) (G)			
PALB2	rs420259	✗	✓	6.3×10^{-8} (D)	N/R (D)	(Wellcome Trust Case Control, 2007)	Protein involved in DNA-damage response (Zhang et al., 2009b).	Unknown.
PBRM1	rs2251219	✗	✓	1.1×10^{-8} (D); 1.7×10^{-9} (D + R_1)	1.15 (D) (A); N/R (D + R_1)	(McMahon et al., 2010)	Subunit of ATP-dependent chromatin-remodelling complexes (Xue et al., 2000).	Unknown.
PGAM1P1	rs1971058	✗	✓	9.9×10^{-8} (D)	2.78 (N/R)	(Yosifova et al., 2011)	Unknown.	Unknown.

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
PGBD1	rs2142731	✓	✗	9.2×10^{-7} (R_1); 5.1×10^{-10} (D + R_1)	1.25 (R_1) (G); 1.27 (D + R_1) (G)	(Yue et al., 2011)	Member of a family of transposases related to transposons (Rolland et al., 2014).	Specifically expressed in the brain. Transposons related to human endogenous retroviruses might be involved in the pathogenesis of SZ, possibly through an epigenetic mechanism (Yao et al., 2008; Yolken et al., 2000; Bundo et al., 2014).
PLAA	rs7045881	✓	✗	2.0×10^{-4} (R_1); 2.1×10^{-6} (D + R_1)	1.16 (R_1) (T); N/R (D + R_1)	(Athanasiu et al., 2010)	Activates phospholipase A2, an enzyme that catalyses the release and reincorporation arachidonic acid into cellular membranes (Zhang et al., 2008).	PLAA participates in inflammatory responses, which is consistent with current neuroinflammation models of SZ (Pasternak et al., 2016) and BD (Haarman et al., 2014). In fact, SZ patients have higher levels of phospholipase A2, leading to increased membrane phospholipid breakdown in the frontal cortex (Peet et al., 1998).
PLXNA2	rs6540451	✗	✓	7.5×10^{-3} (D)	N/R (D)	(Hattori et al., 2009)	Semaphorin receptor involved in signal transduction cascades (Tamagnone et al., 1999).	Facilitates axonal guidance during embryogenesis (Belyk et al., 2015). Gene polymorphisms alter the post-natal developmental trajectory of corpus callosum microstructure (Belyk et al., 2015), and are associated with autism (Suda et al., 2011), generalized anxiety disorder (Coric et al., 2010), AD (Jun et al., 2014), and PD (Schulte et al., 2013).
	rs752016	✓	✗	6.0×10^{-3} (D)	1.49 (D) (T)	(Mah et al., 2006)		
RELN	rs7341475	✓	✗	8.8×10^{-7} (only females) (D + R_1)	1.58 (D + R_1) (GG)	(Shifman et al., 2008)	Extracellular matrix serine protease that acts as a signalling molecule mediating cell-cell interactions (Martinez-Cerdeno et al., 2002).	Expressed in GABAergic interneurons of the cortex and hippocampus (Martinez-Cerdeno et al., 2002), as well as in nerve cells of the enteric nervous system (Bottner et al., 2014). In the embryonic brain, it guides neuronal migration and lamination, while in the adult brain it affects synaptic function and hippocampal neurogenesis (Franco et al., 2011; Folsom and Fatemi, 2013; Teixeira et al., 2012). Altered expression may impair neuronal connectivity and synaptic plasticity, leading to the development of neuropsychiatric disorders, such as SZ (Pisante et al., 2009), BD (Goes et al., 2010), autism (Shen et al., 2016), MDD (Caruncho et al., 2016), AD (Seripa et al., 2008), temporal lobe epilepsy (Dazzo et al., 2015).
RENBP	rs2269372	✓	✗	4.0×10^{-8} (D + R_1)	1.31 (D + R_1) (A)	(Wong et al., 2014)	Renin binding protein plays a role in the regulation of renin activity. The RENBP gene is associated with either increased or decreased risk of developing essential hypertension (Dong et al., 2013).	Unknown.
RUNDC2A	rs12922317	✓	✗	9.0×10^{-7} (D + R_1+R_2)	1.17 (D + R_1+R_2) (G)	(Borglum et al., 2013)	Member of a family of phosphoinositide-binding proteins that orchestrate membrane trafficking events throughout the endocytic network (Seet and Hong, 2006).	Unknown.
SLC23A3	rs13404754	✓	✗	5.0×10^{-3} (D + R_1)	N/R (D + R_1)	(Shibata et al., 2013)	Unknown	Unknown.
SLC39A3	rs4806874	✗	✓	9.0×10^{-6} (D + R_1)	N/R (D + R_1)	(Baum et al., 2008a)	Zinc-influx transporter (Gaither and Eide, 2000).	Zinc is an essential nutrient for brain function and its deficiency may be associated with depression and neurodegeneration (Gronli et al., 2013).

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
TCF4[c,#,§]	rs4309482	✓	✗	2.9×10^{-4} (R_1); 9.7×10^{-7} (D + R_1)	1.14 (R_1) (G); 1.15 (D + R_1) (G)	(Rietschel et al., 2012)	*TCF4* codes for a transcription factor (Flora et al., 2007).	Interacts with a proneural factor to initiate neuronal differentiation of the hindbrain during development (Flora et al., 2007). SZ patients that carry the risk-variant have neurocognitive deficits, reduced sensorimotor gating (Albanna et al., 2014; Lennertz et al., 2011; Quednow et al., 2011), and earlier onset disease (Chow et al., 2016). Gene deletions cause Pitt-Hopkins syndrome, characterized by intellectual disability, developmental delay, epilepsy and craniofacial dysmorphism (Brockschmidt et al., 2007). TCF4 is also involved in a reciprocal transcriptional regulation of the Wnt/β-catenin pathway (Sanchez-Tillo et al., 2015), which is known to be involved in SZ and BD (Panaccione et al., 2013; Lv et al., 2015b; Hoseth et al., 2017; Venkatasubramanian and Debnath, 2013). Exposure to the neurotropic virus EBV affects *TCF4*'s transcription rate, which may be an immunogenetic mechanism mediating psychosis risk (Homa et al., 2013; Arias et al., 2012) (albeit the unclear association between EBV and SZ (de Witte et al., 2015; Bolu et al., 2016)). Valproate treatment increases its expression in a dose- and time-dependent manner (Muhleisen et al., 2014).
	rs9960767	✓	✗	4.1×10^{-9} (D + R_1)	1.23 (D + R_1) (C)	(Stefansson et al., 2009)		
TRANK1	rs6550435	✗	✓	2.1×10^{-8} (D + R_1)	1.13 (D + R_1) (G)	(Muhleisen et al., 2014)	Nucleoside triphosphate hydrolase associated with DNA/ATP binding or DNA repair (Kent et al., 2002).	
	rs9882911	✗	✓	2.1×10^{-8} (D + R_1)	1.13 (D + R_1) (C)			
	rs4678910	✗	✓	2.4×10^{-8} (D + R_1)	1.13 (D + R_1) (G)			
	rs9821223	✗	✓	2.4×10^{-8} (D + R_1)	1.13 (D + R_1) (C)			
	rs4234258	✗	✓	2.5×10^{-8} (D + R_1)	1.13 (D + R_1) (G)			
	rs4624519	✗	✓	2.6×10^{-8} (D + R_1)	1.13 (D + R_1) (T)			
	rs1532965	✗	✓	2.7×10^{-8} (D + R_1)	1.12 (D + R_1) (G)			
	rs9811916	✗	✓	2.9×10^{-8} (D + R_1)	1.12 (D + R_1) (G)			
	rs3732386	✗	✓	3.1×10^{-8} (D + R_1)	1.12 (D + R_1) (T)			
	rs4678909	✗	✓	3.1×10^{-8} (D + R_1)	1.12 (D + R_1) (G)			
	rs7652637	✗	✓	3.4×10^{-8} (D + R_1)	1.12 (D + R_1) (C)			
	rs17807744	✗	✓	4.1×10^{-8} (D + R_1)	1.12 (D + R_1) (T)			
	rs12637912	✗	✓	4.8×10^{-8} (D + R_1)	1.12 (D + R_1) (A)			
	rs9834970	✗	✓	4.8×10^{-8} (D + R_1)	1.12 (D + R_1) (C)			
TSPAN8	rs1705236	✗	✓	6.1×10^{-7} (D)	1.72 (D) (A)	(Sklar et al., 2008)	Member of a family of transmembrane proteins involved in signal transduction events that regulate cell adhesion, motility, activation, and proliferation (Garcia-Frigola et al., 2001).	Tetraspanins have been implicated in myelination (Bronstein, 2000). Most of them form complexes with integrins, which, in turn, are involved in PKC signalling (Berditchevski et al., 1997).
TSPAN18	rs11038167	✓	✗	3.3×10^{-6} (R_1); 1.1×10^{-11} (D + R_1)	1.27 (R_1) (A); 1.29 (D + R_1) (A)	(Yue et al., 2011)	(Same as above.) (Garcia-Frigola et al., 2001)	A tetraspanin that may participate in Ca^{2+}-dependent apoptosis of DAergic neurons in SZ (Zhang et al., 2011a, 2015bZhang et al., 2015bZhang et al., 2011a).
	rs11038172	✓	✗	1.1×10^{-5} (R_1); 7.2×10^{-10} (D + R_1)	1.23 (R_1) (A); 1.25 (D + R_1) (A)			
	rs835784	✓	✗	2.4×10^{-7} (R_1); 2.7×10^{-11} (D + R_1)	1.25 (R_1) (A); 1.27 (D + R_1) (A)			

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Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
TWIST2	rs9751357	✓	✗	2.1×10^{-9} (D)	2.60 (D) (G)	(Alkelai et al., 2011)	Transcription factor (Lee et al., 2000).	Unknown.
UGT1A1	**rs741160**	✓	✗	3.4×10^{-8} (D); 3.0×10^{-2} (R_1)	1.17 (D) (G); 1.30 (R_1) (G)	(Alkelai et al., 2011)	*UGT1A1* codes for a detoxification enzyme involved in the elimination of exogenous and endogenous compounds (Kutsuno et al., 2015).	Highly expressed in the brain, where it regulates the local concentration of thyroid hormone, estradiol and bilirrubin (Kutsuno et al., 2015). Inactivating polymorphisms cause bilirrubin-induced neurotoxicity via activation of TLR-2-mediated inflammatory signals (Yueh et al., 2014). Inflammation itself downregulates *UGT1A1* expression via NF-κB activation (Shiu et al., 2013). The UGT1 complex locus is also involved in the metabolism of 5-HT, DA, antidepressants, antipsychotics, mood stabilizers and BZD (de Leon, 2003).
VRK2[§]	rs2717001	✓	✗	2.0×10^{-2} (R_1); 1.0×10^{-4} (D + R_1)	1.08 (R_1) (C); 1.12 (D + R_1) (C)	(Rietschel et al., 2012)	Serine/threonine protein kinase that maintains nuclear architecture and regulates signalling pathways involved in cell growth, apoptosis, stress response to hypoxia and IL-1β transcriptional response (Blanco et al., 2006, 2007, 2008Blanco et al., 2006Blanco et al., 2007Blanco et al., 2008).	Participates in a signalling pathway that protects against stress-induced neuronal death and contributes to axonal development (Li et al., 2006; Dong et al., 2005). It also interacts with a gene product of EBV (Li et al., 2006), a virus whose early-life exposure increases the risk of psychosis (Khandaker et al., 2014). Genetic variation was associated with altered brain structure in healthy subjects (Li et al., 2012) and SZ patients (Sohn et al., 2014), as well as genetic forms of epilepsy (Consortium et al., 2012) and neurodevelopmental syndromes (Chabchoub et al., 2008; Prontera et al., 2011; Rajcan-Separovic et al., 2007).
WHSC1L1/ FGFR1	rs1488935	✓	✗	3.4×10^{-5} (R_1) 5.1×10^{-9} (D + R_1)	1.15 (R_1) (G) (N/R) (D + R_1)	(Shi et al., 2011)	WHSC1L1 codes for a histone methyltransferase involved in epigenetic control of gene expression (Kim et al., 2006). FGFR1 codes for a tyrosine-protein kinase that acts as cell-surface receptor for fibroblast growth factors (FGFs), thereby regulating mitogenesis and differentiation (Mohammadi et al., 1996).	Epigenetic mechanisms have been implicated in the pathogenesis of SZ (Gavin and Sharma, 2010) and BD (Ludwig and Dwivedi, 2016), but the specific contribution of WHSC1L1 is unknown. FGFs play key physiological roles during development, including gastrulation, organogenesis, neurogenesis and neuronal differentiation (Eswarakumar et al., 2005; Lonic et al., 2013), and their effects are mediated by interaction with TGFβ, Wnt, MAPK, IP3 and Notch signalling pathways (among others) (Dailey et al., 2005). More specifically, FGFR1 controls the terminal differentiation, maturation, and maintenance of midbrain DAergic neurons (Baron et al., 2012), with its inhibition resulting in a SZ-like phenotype in mice (Klejbor et al., 2006). Additionally, it forms heteroreceptor complexes with 5-HT1A in midbrain raphe and hippocampal neurons, where it exerts neurotrophic and antidepressant effects (Borroto-Escuela et al., 2015). Likewise, inhibition of FGFR blocks antidepressant-induced glial cell line-derived neurotrophic factor production (Hisaoka et al., 2011). Its role in SZ and MDD is also supported by its increased expression in these disorders (Gaughran et al., 2006).
ZFAT	rs7819815	✓	✗	3.1×10^{-7} (D)	N/R (D)	(Wang et al., 2011a)	Zinc finger protein that acts as a transcriptional regulator of immune-cell survival and vascular remodelling (Yoshida et al., 2010; Fujimoto et al., 2009).	Implicated in the regulation of immune responses and susceptibility for immune-mediated diseases, specifically autoimmune thyroid disease (Shirasawa et al., 2004) and multiple sclerosis (Bourguiba-Hachemi et al., 2016). It is expressed in the brain and placenta, and it is down-regulated in placentas from complicated pregnancies, which is a well-known SZ risk factor (Barbaux et al., 2012). We hypothesize that variation in this gene might be implicated in maternal transmission of SZ.

(*continued on next page*)

Table 2 (*continued*)

Gene (region)	SNP	SZ	BD	P-value*	Odds Ratio (risk allele)	Reference	Gene product function	Implication in the CNS
ZNF804A[d]	rs1344706	✓	✗	2.5×10^{-11} (D + R_1) 7.1×10^{-7} (D) 1.6×10^{-7} (D + R_1+R_2)	1.10 (D + R_1) (T) 1.38 (D) (T) 1.12 (D + R_1) (T)	(Williams et al., 2011a) (O'Donovan et al., 2008)	Zinc finger protein that plays a role in DNA binding and transcriptional control (Girgenti et al., 2012).	Regulates the expression of genes implicated in DAergic transmission (Girgenti et al., 2012), and it is regulated by GLUergic transmission (Chang et al., 2015). Affects neurons' response to inflammatory cytokines, thereby supporting a role for immuno-inflammatory processes in psychosis (Chen et al., 2015a). The association with SZ has been replicated in gene association studies (Zhang et al., 2011b; Chen et al., 2012), and correlates with abnormal functional connectivity in working memory and theory of mind networks (Esslinger et al., 2011; Zhang et al., 2016b; Mohnke et al., 2014). SZ-risk variants also increase the risk of heroin addiction possibly secondary to changes in decision-making and gray matter volume (Sun et al., 2016). ZNF804 CNVs have been implicated in autism (Griswold et al., 2012) and neurodevelopmental impairment (Blake et al., 2014).
ZKSCAN4	rs1233710	✓	✗	4.1×10^{-7} (R_1); 4.8×10^{-11} (D + R_1)	1.25 (R_1) (C); 1.27 (D + R_1) (C)	(Yue et al., 2011)	Zinc finger protein that acts as a transcriptional regulator of MDM2 (a proto-oncogene) and EP300 (a histone acetyltransferase) (Li et al., 2007).	Unknown.

Abbreviations: ACC, anterior cingulate cortex; ACh, Acetylcholine; AD, Alzheimer's disease; ADHD, attention-deficit/hyperactivity disorder; ATP, adenosine triphosphate; BBB, blood brain barrier; BDNF, brain-derived neurotrophic factor; BDZ, benzodiazepines; cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; CNS, central nervous system; DA, dopamine; DLPFC, dorsolateral prefrontal cortex; EBV, Epstein-Barr virus; ECM, extracellular matrix; EGFR, epidermal growth factor receptor; GLU, glutamate; GTP, guanosine triphosphate; IP3, inositol triphosphate; LD, linkage disequilibrium; MAPK, mitogen-activated protein kinase; MDD, major depressive disorder; MS, multiple sclerosis; NT, neurotransmitter; OFC, orbitofrontal cortex; PDGFA, platelet-derived growth factor subunit A; PD, Parkinson's disease; PKC, protein kinase C; TBI, traumatic brain injury; TGFβ, transforming growth factor beta; TLE, temporal lobe epilepsy; TNF – tumour necrosis factor; TS, Tourette's syndrome.

*D = discovery sample; $R_{(n)}$ = replication sample(s); D + R = combined samples. [!]Found in the Psychiatric GWAS Consortium Bipolar Disorder Working Group (2011) (PsychiatricConsorti, 2011). [#]Found in the Schizophrenia Psychiatric Genome-Wide Association Study Consortium (2011) (The Schizophrenia Psychia, 2011). [§]Correction in legend of Table 2 of Rietschel et al. (2012): "major/minor allele" should read "minor/major allele".

[a] Although Shi et al. (2009) and Stefansson et al. (2009) each reported an association between SZ and *HIST1H2AG* with rs6913660's allele C, we have only considered one independent association, due to sample overlap between these studies.

[b] In Lencz et al. (2013), the discovery sample included SZ patients, and one replication sample comprised SZ and another comprised BD subjects.

[c] Although Rietschel et al. (2012) found an association with *TCF4*, their sample overlaps with the replication sample from Stefansson et al. (2009) reviewed by Lee et al. (2012a).

[d] (O'Donovan et al., 2008) and Williams et al. (2011a) reported an association between SZ and *ZNF804A*; however, due to sample overlap, we only considered one positive association. Moreover, Williams et al. (2011a)'s discovery sample contained SZ and BD patients, but the replication sample comprised only SZ subjects; thus, it is unclear if there is an association with BD.

[e] None of the SNPs that are reported to be within or near the MHC region have been implicated more than once (which is why there is no SNP in bold/underlined). Yet this gene region (which is a large region comprised of several different genes) has been implicated in the two cited (independent) studies – which is why the MHC is in bold/underlined.

they are not included in Table 1. To facilitate interpretation of the GWAS results retrieved either in this review or in the Lee et al. (2012a) review, each implicated gene is contextualized in terms of its association with SZ or BD, and its contribution to cellular-level function and to central nervous system (CNS) function in Table 2.

Finally, to appraise the quality of the reviewed studies, we considered the consensually recommended guidelines (Spencer et al., 2009) of: 1) a minimum of 2000 cases and 2000 controls (in either the discovery or, for full replication, the replication sample); 2) power calculation to detect a genome-wide significant association; 3) the inclusion of imputed SNPs; 4) a follow-up replication of the most significant associations in an initial sample (based on, e.g. the 'N top SNPs' or 'all SNPs above threshold') to preclude false positives; 5) a statistical significance threshold, corrected for multiple comparisons, of $p < 5 \times 10^{-8}$; 6) the reporting of effect sizes; 7) an account for ethnic differences in the sample, e.g. via stratification using principal component analysis (PCA), as different ethnicities carry different linkage disequilibrium (LD) patterns and allelic frequencies; and 8) an account of age and gender differences across cases and controls.

3. Results

3.1. Overview

Our initial database search identified 3520 records. After removal of duplicates and abstract screening for relevant studies, we retrieved 36 articles for full-text reading. From these, 5 meta-consortia or meta-analyses (PsychiatricConsorti, 2011; Green et al., 2012a; The Schizophrenia Psychia, 2011; Schizophrenia Working Gro, 2014; Williams et al., 2011b) and 9 GWAS (Nassan et al., 2017; Steinberg et al., 2012; Smith et al., 2011; Fanous et al., 2012; Hou et al., 2016a; Ripke et al., 2013; Sleiman et al., 2013; Xu et al., 2014; Meier et al.,

2012) were excluded due to sample overlap with a study from the current or the previous review with the exception of 2 GWAS. In one (Rietschel et al., 2012) the authors took into account the issue of sample overlap in their analysis: as the study includes two replication samples, and only part of the first replication sample overlaps with the sample from a study (Stefansson et al., 2009) reviewed by Lee et al.(2012)[5], in their discussion, the authors explain that the finding of rs4309482, located near CCDC68/TCF4, cannot be regarded as an independent replication (this information is included as a footnote in our Table 2), and tested the other SNPs in the non-overlapping portion of the sample. The second study's sample (Muhleisen et al., 2014), albeit overlapping with a replication sample from a study reviewed by Lee et al. (2012a), had never been analysed in a discovery step for BD before. A total of 22 studies were eligible for inclusion, from which we additionally omitted 6 replication analyses, which had overlapping samples (Wang et al., 2011a; Irish Schizophrenia Genomics and the Wellcome Trust Case Control, 2012; Goes et al., 2015; Kanazawa et al., 2013; Lencz et al., 2013; Alkelai et al., 2012). Excluded studies' abstracts and reasons for exclusion can be found in Supplementary Results 1.

3.2. GWAS in SZ

In the Arab-Israeli population, Alkelai et al. (2011) (Alkelai et al., 2011) found associations between 8 SNPs located within 6 genes (*LRRFIP1*, *LOC*645434/NMBR, *ACSL3*, *TWIST2*, *UGT1A1* and *EFHD1*) and SZ. The *UGT1A1* and *EFHD1* genes were, in fact, also associated in a German case-control replication sample but, surprisingly, the latter SNP, in the opposite allelic direction which could be a spurious effect or, since two different ethnic populations are concerned, a flip-flop effect (Lin et al., 2007). *LRRFIP1,* a transcription factor putatively regulating neurogenesis (Alkelai et al., 2011), and *EGFR* (Rikiyama et al., 2003) (implicated in epidermal growth), have been previously associated with SZ (Morar et al., 2007; Benzel et al., 2007). *UGTA1* has a role in the solubility and excretion of drugs, toxins, hormones, and neurotransmitters (de Leon, 2003); *EFHD1* in neuronal differentiation (Tominaga and Tomooka, 2002); and *NMBR* in fear/anxiety, thermoregulation and susceptibility stress in mice (Gonzalez et al., 2008; Yamada et al., 2002). Another study by the same group (Alkelai et al., 2012), with a Jewish- Israeli sample, found an association with an intronic SNP in *DOCK4,* implicated in neurodevelopment (Ueda et al., 2008) (including the Wnt/beta-catenin pathway previously associated with SZ (Freyberg et al., 2010)); and dyslexia (Pagnamenta et al., 2010) and autism (Pagnamenta et al., 2010), which share common genetic factors with SZ (Horrobin et al., 1995; Stefansson et al., 2014; Malhotra and Sebat, 2012). In terms of genetic associations with age of SZ onset in European-Americans, Wang et al. (2011) (Wang et al., 2011a) found *ZFAT,* likely involved in immune-cell survival and autoimmune diseases, such as thyroid disease (Shirasawa et al., 2004) and multiple sclerosis (Comabella et al., 2009) in a linkage region previously associated with SZ and BD (Park et al., 2004). Bergen et al. (2012), who also investigated BD, failed, in their Swedish sample, to find any SNPs, but when meta-analytically combining their samples with previous ones, found support for the MHC region.

The *MHC* finding supports previous GWAS reports (Stefansson et al., 2009; International Schizophrenia et al., 2009; Shi et al., 2009) and the growing evidence of its role in neurodevelopment (Shatz, 2009) and brain connectivity (Needleman et al., 2010). It got further support from the Irish SZ Genomics Consortium (2012) (Consortium, 2012). From 11 SNPs selected in a discovery sample, they replicated 3SNPs, in a 10x larger sample: 1 in *MHC,* 1 in *CACNA1I* and the intergenic rs7618341. *CACNAC1I* codes for a subunit of a calcium channel family, similarly to the abovementioned *CACNA1C* (Lee et al., 2012a) (GWA-implicated in SZ (Green et al., 2010) and BD (Ferreira et al., 2008)), and to CACNB2[30] and CACNB3[27] - also found to be associated with SZ and BD respectively in below-mentioned consortia. Thus, there are three lines of evidence implicating calcium channels in psychosis.

Using several mixed European samples, Rietschel et al. (2012) (Rietschel et al., 2012) highlighted 9 SNPs across *AMBRA1*, *CUX1, VRK2* and *CCDC68/TCF4.* The latter SNP was identified in an overlapping sample with a previous positive report (Stefansson et al., 2009), and hence, was not regarded as an independent replication. *VRK2*, on the other hand, has been replicated with SZ in the Han Chinese (Zhang et al., 2015a). *AMBRA1* with a major role in neurodevelopment (Behrends et al., 2010; Fimia et al., 2007; Cecconi et al., 2007), resides in a high LD region with genes regulating: autophagy (Behrends et al., 2010); neurotransmission (Nobili et al., 2018); signal transduction (Moskvina et al., 2009), neurodevelopment and plasticity (Hozumi et al., 2009), working memory, anxiety (Dere et al., 2014) and prepulse inhibition in mice (Nakamura et al., 1998; Ohgake et al., 2009). Betcheva et al. (2013) (Betcheva et al., 2013) found, in a Bulgarian sample, an intronic SNP for SZ in *HHAT*, which is involved in carcinogenesis (Katoh and Katoh, 2005) and neurodevelopment (Chamoun et al., 2001), and is in a candidate region for SZ as suggested by previous genetic linkage studies and cytogenetic findings (Hovatta et al., 1999). In a population cohort, Borglum et al. (2013) (Borglum et al., 2013) found an interaction between the maternal cytomegalovirus infection (suspected as a precursor of SZ) and an intronic *CTNNA3* SNP on SZ risk, with a Danish (as well as German and Dutch) sample. *CTNNA3* mediates brain cell–cell adhesion and cytoskeletal structure (Yap et al., 1997), which may be disrupted by the cytomegalovirus during gestation (Scholz et al., 1999), as was found in transgenic Drosophila (Steinberg et al., 2008). Such findings strengthen the immunological hypothesis of SZ stemming from previous associations with: 1) prenatal infections with viral or bacterial pathogens (albeit not consistently) (Brown, 2006), 2) auto-immune processes (Strous and Shoenfeld, 2006) and 3) the strong signal in the MHC locus (Stefansson et al., 2009). Moreover, *CTNNA3* and its nested gene *LRRTM3* have both been associated with Alzheimer's disease (Edwards et al., 2009; Morgan et al., 2008; Liang et al., 2007) and autism (Sousa et al., 2010; Wang et al., 2009).

In Ashkenazi Jews, Lencz et al. (2013) (Lencz et al., 2013) identified a novel susceptibility locus for SZ near *NDST3,* replicated in six SZ and five BD independent cohorts (excluded due to overlap). This gene is abundantly expressed in the cerebellum and hippocampus (Lein et al., 2007) and is involved in neurite outgrowth (Irie et al., 2008), axon guidance (Inatani et al., 2003) synaptogenesis (Lucido et al., 2009), and binding affinity to *NRG1* (neuroregulin 1), a gene previously implicated in SZ (Cho et al., 2015) and BD (Rolstad et al., 2015). This association was later replicated in a Han Chinese population (Zhang et al., 2016a). Goes et al. (2015) (Goes et al., 2015) found no GWAs in Ashkenazi Jews, but their strongest markers were in the 22q11.2 deletion syndrome region, one of the most strongly associated CNVs with SZ (Jonas et al., 2014) which contains the genes *TBXI*, *GLN1* and *COMT*.

Regarding the East Asian population, Ikeda et al. (2011) (Ikeda et al., 2011) failed to find significant associations in Japanese, but obtained support for a previous GWAS finding of *NOTCH4* and new trends for *OAT* and *SULT6B1*. Shi et al. (2011) (Shi et al., 2011) found an association of SZ with 3 SNPs in a replication (but not the discovery; both Han Chinese) sample within *WHSC1L1/FGFR1, LSM1, BRP44,* and *DCAF6*. *FGFR1* is involved in neurodevelopment and upregulated in SZ and major depression (Gaughran et al., 2006), with its manipulation triggering a SZ-like phenotype in mice (Klejbor et al., 2006). Moreover, fibroblast growth factors (FGFs) have been implicated in SZ (Terwisscha van Scheltinga et al., 2010; O'Donovan et al., 2009). The intronic SNP for *DCAF6* is located downstream of *MPZL1,* a previous SZ-risk candidate (He et al., 2006), coding for a myelin protein upregulated in SZ, possibly as a compensatory mechanism (Tkachev et al., 2003). Yamada et al. (2011) (Yamada et al., 2011) failed to find any GWA-significant SNPs in Japanese, but reported a trend in *ELAVL2*. Yue et al. (2011) (Yue et al., 2011) found 2 SNPs in Han Chinese, *LOC392301* and *LOC729457*, and replicated 6 SNP associations across the neurodevelopmental ZKSCAN4 and NKAPL genes, the epigenetic PGBD1 gene, and

the apoptosis regulator *TSPAN18*. The PGBD1 gene has been associated with SZ in Europeans (Stefansson et al., 2009), but not Asians (Zhang et al., 2015b; Kitazawa et al., 2012); and the NKAPL has been validated in other studies (Wang et al., 2015; Chen et al., 2014). Kanazawa et al. (2013) (Kanazawa et al., 2013) attempted to trace genetic susceptibility to atypical psychosis and its overlap with SZ and BD, also in the Japanese population. Although no SNPs fulfilled GWA significance, there were trends in *CHN2/CPVL*, *COL21A1*, *PYGL/TRIM9* and *MHC* regions. Wong et al. (2014) (Wong et al., 2014) failed to find any GWA significant association in Han Chinese, but after analysing their combined sample, found a significant SNP near *RENBP* gene, involved in regulating blood pressure and sodium homeostasis, which are commonly disrupted in SZ and BD and influenced by antipsychotic treatment (for a review, see Correll et al. (2015)). An explanation for this relationship may be the central effects of angiotensin-II on dopaminergic activity (Jenkins et al., 1996). Uncommonly using microsatellites (N = 28,601), instead of SNPs, Shibata et al. (2013) (Shibata et al., 2013) marked regions of susceptibility, with 3 sequential steps of pooled Japanese DNA analyses to consecutively select the best-associated microsatellite markers, implicating *SLC23A3*, *CNPPD1*, and *FAM134A*, which are expressed in the brain but of yet unclear function and not previously associated with SZ.

3.3. GWAS in BD

Using a highly mixed sample (across USA, West and Eastern Europe, and Russia), (Muhleisen et al., 2014) identified 56 genome-wide significant SNPs in 3 previously known risk genes (*ANK3, ODZ4* and *TRANK1*) and in 2 new regions (*ADCY2* and *MIR2113/POU3F2*). *ADCY2* mediates dopamine signalling (Beaulieu and Gainetdinov, 2011), is implicated in learning, memory and mood (Porteous et al., 2006), and is associated with SZ and BD (Kahler et al., 2010). *ODZ4* is implicated in neuronal plasticity and signalling (PsychiatricConsorti, 2011); *POU3F2* in neurodevelopment (Kwan et al., 2012), and *MIR2113* is a microRNA gene with unknown function. In a Bulgarian sample, Yosifova et al. (2011) (Yosifova et al., 2011) found one non-replicated association near *LOC100130514/LOC728103* genes.

With an uncommon quantitative trait loci GWAS approach, Greenwood et al. (2012) (Greenwood et al., 2012) performed in Europeans a search for genetic associations with five clinical subtypes of BD defined by temperament: hyperthymic, dysthymic, cyclothymic, irritable and anxious. Hyperthymia was associated with 3 SNPs within or near *MDM1* (of unknown function) and the neurodevelopmental FBLN1 gene, while the irritable subtype yielded 2 SNPs for the INTS7 and the DTL genes of unknown function. In an independent study, Greenwood et al. (2013) (Greenwood and Kelsoe, 2013) also looked at two clinical dimensions in Europeans: “irritable” vs “elated mania”. Albeit none were GW significant, 3 trends ($p < 1 \times 10^{-4}$) within *SLITRK1/6, GRIA3* and *GABRG1* were found after permutation analysis, with support from nearby SNPs also associated with a $p < 1 \times 10^{-4}$.

3.4. Appraisal overview

From the 22 studies we reviewed, only 4 (18,2%) used the minimum recommended 2000 + cases and 2000 + controls (in either the discovery or, for full replication, replication sample) (Spencer et al., 2009). Approximately half (45,4%) of the studies included imputed SNPs to increase power (Spencer et al., 2009), and the same percentage used the widely consensual significance threshold of $p < 5 \times 10^{-8}$. However, most of the studies that failed to do this were published earlier, indicating that this is now becoming the norm. Slightly more than half (54,5%) reported a power calculation to detect a GWA. In 72,7%, there was a reported replication attempt in an independent sample. Effect sizes were reported, when the effect was statistically significant, in 87,5% of studies. Of all the studies including subjects of different ethnicities, only 68,2% corrected their analyses for population stratification, with multidimensional scaling analysis or PCA. Finally, only half of the studies reported having controlled for age and gender differences across cases and controls. Appraisal of each of the studies reviewed, for each of the appraisal criteria, are available in Supplementary Table 1.

4. Discussion

4.1. Results' overview

The advent of GWAS over the last decade has accelerated the discovery of novel genetic markers associated with SZ and BD. As most psychiatric disorders are polygenic and associated SNPs have small effect size, the Psychiatric Genomics Consortium (PGC) (PsychiatricConsorti, 2011; The Schizophrenia Psychia, 2011; Schizophrenia Working Gro, 2014) as well as other consortia (Stefansson et al., 2009; Irish Schizophrenia Genomics and the Wellcome Trust Case Control, 2012; International Schizophrenia et al., 2009) were formed to conduct meta- and mega-analyses of available genome-wide data; hence these powerful studies constitute useful summaries of the progress in the field. However, evaluation of independent studies, such as undertaken herein, is useful. This allows the critical appraisal of the evidence supporting findings from smaller studies, in particular, evidence of replication, which is a useful adjunctive to the “blanket” approach of the threshold criterion of genome-wide significance in a meta-analysis. In addition, such systematic review of the smaller studies can potentially help entice and design pathophysiological hypotheses, especially in specific populations, for further genetic, transcriptomics, proteomics and drug studies (with animal model included). They can help make sense of recent and future meta- and mega-analyses reports, and help the research community's reflection on the heterogeneity and inconsistencies in samples, methods and findings across SZ and BD association studies.

Out of the 22 GWAS reviewed, and according to p-value thresholds authors chose: 9 found at least a significant association in the discovery stage, 7 found at least one in the replication stage, 2 found at least one in both stages, and 7 failed to find any at all. It is evident that GWAS have become more exploratory regarding statistical methodology and design, since the review by Lee et al. (2012a). Two reviewed (Stefansson et al., 2009; Greenwood et al., 2012) (and two excluded (Fanous et al., 2012; Meier et al., 2012)) GWAS split SZ or BD into phenotypic subtypes, thus making steps towards validating genome-wide links between genotypes and clinical phenotypes. Shibata et al. (2013) (Shibata et al., 2013) used microsatellite markers and a three-step design, carrying only the top SNPs identified to the next step, with independent samples of pooled DNA being used at each stage, and a relaxed significance threshold of $p < 0.05$. As microsatellites have multiple alleles, they can detect higher LD than SNPs as well as higher levels of heterozygosity. However, given its novelty, it is still difficult to compare this GWAS design with a standard SNP-based one, especially as it is unclear how well it does correct for multiple testing. Finally, the use of family-based approach (in 4 studies (Alkelai et al., 2012; Alkelai et al., 2011; Yamada et al., 2011; Levinson et al., 2012)) was beneficial for its ability to control for population stratification which can be a problem in standard case-control GWAS.

We found that whilst 104 SNPs associations were reported as significant, only 83 met the standard stringent threshold at $p < 5 \times 10^{-8}$ (either in the discovery, the replication or the combined analysis). The latter SNPs were located within 28 gene regions: *ACSL3/KCNE4, ADCY2, AMBRA1, ANK3, BRP44, DTL, FBLN1, HHAT, INTS7, LOC*645434/NMBR, *LOC729457, LRRFIP1, LSM1, MDM1, MHC, MIR2113/POU3F2, NDST3, NKAPL, ODZ4, PGBD1, RENBP, LOC392301, TRANK1, TSPAN18, TWIST2, UGT1A1/HJURP, WHSC1L1/FGFR1,* and *ZKSCAN4*. Other genes implicated in the reviewed studies (using authors' significance thresholds) even though not reaching the standard $p < 5 \times 10^{-8}$ were: *ARNTL, CCDC68/TCF4,*

CDH13, CNPPD1, CTNNA3, CUX1, DCAF6, DOCK4, EFHD1, FAM134A, LOC100130514/LOC728103, RAB17/LRRFIP1, RUNDC2A, SLC23A3, UGT1A1, VRK2, and *ZFAT*. Finally, *AMBRA1*, *ARNTL*, *CDH13*, *EFHD1* and *UGT1A1* have been implicated in two independent samples reviewed in the present review. Moreover, the ANK3 gene and MHC region have been once implicated in one study from the set reviewed by Lee et al. (2012a) and also in one independent study from our set of reviewed studies.

In summary, to this date, taking all studies discussed in Lee et al. (2012a) and in the current one into account, as in Table 2, we found that the MHC region, and the *AMBRA1*, *ANK3, ARNT*, *CDH13*, *EFHD*, *PLXNA2* and *UGT1A1* genes have been found to be associated (with the same SNP) with either SZ or BD, in at least two reportedly independent (non-overlapping) samples; and with the same risk allele (except for *EFHD1)*. This lends substantial confidence in their association with psychosis. No further evidence for a shared genetic basis between SZ and BD since the review by Lee et al. (2012a) and the appraisal by Williams et al. (2011b), which had implicated *ANK3* and *PLXNA2* in both diseases. As a side note, if we had not excluded non-overlapping samples, the findings for *CACNA1C*, *NDST3*, and *ZNF408A* would have come out as new supportive evidence for a shared genetic influence between BD and SZ.

4.2. Replication findings and meta-consortia

The creation of large international meta-consortia has been a successful story in psychiatric genetics, as the collection and meta-analysis of very large samples has led to the discovery of several markers associated with the disorders. However, even with tens of thousands of cases and controls, we cannot eliminate the possibility of type I error as, in some cases, genome-wide significant findings from one consortium study have not always been replicated with the expansion of the total sample (The Schizophrenia Psychiatric Genome-Wide Association Study (GWAS) Consortium, 2011; Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014). One criticism that we deem important is that meta-analyses focus only on markers with a "universal" effect on the phenotype and may miss SNPs with effect in specific populations. Of course it is difficult to be confident that the later are not false findings (type I error) in small samples. One way of overcoming the possibility of type I error is to focus on SNPs that were replicated independently in a different sample, as was the focus of our review. Another handicap the earliest GWAs studies, in our review, presented, relates to the *combined* p-value (i.e. the one emerging from a discovery and a replication sample combined) of a given SNP, being considered by some authors as one that provides a '*validation*' of that marker's original association in the discovery sample. What truly validates a finding is it being replicated in a completely independent sample. Other weaknesses are the omission, or explicitness, of the significance threshold in the replication sample or the selection of an arbitrary number of top SNPs to replicate, e.g. the "top 43 SNPs after ranking their p-values in descending order". These weaknesses arose as a need to compensate for lack of power, and available data, but been surpassed in the most recent, and powerful, GWAS.

Accepting replications at a nominal significance level (i.e. $p < 0.05$) of markers that do not reach genome-wide significance in the discovery sample also increases the possibility of type I error. On the other hand the selection of an arbitrary p-value threshold to determine SNPs to replicate may exclude truly associated SNPs (as indicated from the increase of polygenic prediction when lowering the p-value threshold and including thousands of SNPs), increasing the possibility for type II error.

As we mentioned above, in order to avoid overestimated conclusions or false replications we have excluded studies that used samples overlapping with those from studies we or Lee et al. (2012a) already review [e.g. meta-consortia, meta-analyses, etc.]. Nevertheless, we next discuss meta-consortia results (PsychiatricConsorti, 2011; The Schizophrenia Psychia, 2011; Irish Schizophrenia Genomics and the Wellcome Trust Case Control, 2012), including the latest Genomics consortium study (Schizophrenia Working Gro, 2014). On this note, we bring awareness to the fact that the studies we herein review, besides using smaller samples, have a slightly larger number of non-Caucasian-samples, compared to the meta-consortia studies which typically use Caucasian samples.

The Psychiatric Genomics Consortium BD Working Group (2011) (PsychiatricConsorti, 2011) found 4 significant associations of BD with SNPs in *ANK3*, *SYNE1* and *ODZ4,* with the first two not further replicated. From the 34 SNPs selected for replication (46,918 European cases and controls), associations were found across: *CACNA1C*, *ODZ4, ZDHHC24*, *RND1*, *TXNDC9, SPHK2, CACNB3, TUBA1B*, *LOC731779, C15orf53, MAD1L1*, *LBA1*, *FSTL5*, *LMAN2L*, *WDR82* and *ZZZ3,* among others in the same LD blocks. Combination of the two samples made a SNP for *CACNA1C* and one for *ODZ4* reach an association ($p < 5 \times 10^{-8}$). The *CACNA1C* finding further supports its above-mentioned previous associations with BD (Ferreira et al., 2008) and SZ (Green et al., 2010). Furthermore, the implication of *CACNA1C,* albeit via another SNP, was confirmed in a joint analysis with a SZ sample. The latter also highlighted a SNP for *ANK3* and a multi-gene region *ITIH3-ITIH4* tagged by rs2239547, which encodes plasma serine protease inhibitors with functions in extracellular matrix stabilization and suggestive involvement in suicidal behaviour in SZ and BD (Finseth et al., 2014).

Green et al. (2012) confirmed findings in *ODZ4* and *CACNA1C* (but not of *ANK3* or *SYNE1*) of the above BD consortia study (PsychiatricConsorti, 2011), after testing the 3106 SNPs identified at $p < 5 \times 10^{-3}$ in that consortium. Moreover, a combined analysis showed two novel statistically significant associations: rs7296288 between *RHEBL1* and *DHH*, and rs3818253 tagging the *TRPC4AP/GSS/MYH7B* region. Confirmation for *SYNE1* was obtained in another replication sample (Green et al., 2012b), at $p < 0.05$, with BD, and also unipolar depression - it encodes an outer-membrane protein connecting nuclei to cytoskeletons, implicated in muscle formation, weight regulation and growth in mice (Zhang et al., 2009a, 2010). Steinberg et al. (2011) (Steinberg et al., 2011) also extended an earlier SZ study (itself containing replication for SNPs at $p < 5 \times 10^{-5}$), to replicate findings at $p < 5 \times 10^{-4}$ significance. In addition to confirming *MHC*, *NRGN* and *TCF4* associations, they found one within *CCDC68/TCF4*, and another upstream of *VRK2* – however, this sample, and thus these findings, overlap with one we review above (Rietschel et al., 2012).

Also using some of the samples of studies we review, the SZ Psychiatric Genomics Consortium (The Schizophrenia Psychia, 2011) found 136 $p < 5 \times 10^{-8}$ significant SNP associations at discovery-level, the majority of which (N = 129) mapped to the *MHC*, *TCF4* and *NRGN* regions, among other new regions in 10q24.33 and 8q21.3. Among the 81 SNPs ($p < 5 \times 10^{-5}$) where replication was attempted, rs1625579, within a *MIR137* intron, was the strongest, followed by other loci targets of this gene, suggesting its dysregulation may be a newly found etiologic mechanism in SZ. For example, *MIR137* regulates adult neurogenesis (Szulwach et al., 2010) and neuronal maturation (Smrt et al., 2010). The next best target loci were tagged by rs7914558 and rs11191580, implicating multiple genes, then rs7004633 near *MMP16*, which encodes an endopeptidase involved in a range of cellular behaviours; lastly, rs10503253 in *CSMD1,* involved in neuronal growth (Kraus et al., 2006).

In an attempt to further increase power, the 2014 SZ Working Group of the Psychiatric Genomics Consortium aimed to include all existing SZ samples (Schizophrenia Working Gro, 2014). They found significant GWAs spanning 108 conservatively defined loci, 83 of which not previously reported. These provided support for: 1) the dopamine hypothesis of SZ (dopamine receptor type 2, *DRD2* was found); 2) the glutamatergic hypothesis of SZ (*GRM3*, *GRIN2A*, *SRR*, *GRIA1* were found), and 3) immunological hypothesis of SZ (B-lymphocyte lineages involved in acquired immunity such as CD19 and CD20 lines were also found). In addition, associations at *CACNA1C*, *CACNB2* and *CACNA1I*

continue to be strengthened. A more comprehensive portrait of the biological pathways and plausibility inherent to this gene set, for SZ, was provided subsequently via a new framework for interpretation of genetic association studies (DEPICT) (Pers et al., 2015) and showed that its genes are highly expressed in the brain cortex, enriched for ion channel pathways, functionally related to each other, and enriched for previously SZ-associated rare disruptive variants and *de novo* variants, and for genes encoding members of postsynaptic density proteomes.

In summary, findings from the above meta-consortia studies implicate *ANK3, CACNA1C, CACNA1I, CACNB2, CACNB3, DRD2, GRIA1, GRIN2A, GRM3, ITIH1, ITIH3/ITIH4,* MIR137 (and its target loci), *ODZ4, SRR, SYNE1, TCF4, VRK2, ZNF804A,* and, again, the *MHC* region in psychosis.

4.3. Limitations and other GWA-based or GWA-complementary approaches

Given the quality appraisal we retrieved, we call attention to the following three statistical methodological shortcomings found in more than half of the studies: recommending that GWAs studies follow a 2000 + cases and 2000 + controls in either discovery or replication samples, perform imputation, and follow the consensual statistical significance threshold of $p < 5 \times 10^{-8}$; and, although followed in more than half (but not all) of studies, we highlight the recommendation that ethnical stratification correction, replication and effect sizes are performed/reported. An additional limitation noticed in most previous GWAs studies is the absence of a transcriptomic, proteomic or pharmacological experiment in vitro, animal models or humans, with the goal to validate the (epidemiologically) identified genes. This would protect the field against false positive findings, allow an assessment of the gene's functional significance (which often times is unknown for the CNS), and improve the patho-physiological and therapeutic models for these illnesses.

Beyond detecting simultaneously multiple SNPs of small effect, the next challenge is to detect gene-gene interactions. Due to GWAS looking for individual SNP-based associations, any susceptibility factors arising from multiple variations (in the same or multiple genes) interacting with one another may be missed. Oh et al. (2012) (Oh et al., 2012) proposed a new GWA method (involving dimensionality reduction) for detecting gene-gene interactions and applied them to the WTCCC data. Interactions between gene-level effects (which are calculated via combining that of multiple SNPs within each gene) are estimated. For example, their top result, an interaction between *NEBL (*coding for a cardiac muscle protein) and the oncogene *ERG,* had not been identified in GWAS thus far. Hence, complementary to a standard GWAS is the investigation of epistatic interactions between candidate genes: e.g. the insulin-induced *INSIG1* and *INSIG2* genes interact to predict metabolic syndrome onset in SZ patients as a response to atypical antipsychotics therapies (Liou et al., 2010).

An important, and still unaccounted for, consideration in GWAS of psychosis is that gene-environment interactions probably contribute to a large part of the susceptibility (Thomas, 2010; Modinos et al., 2013; European Network of National Networks studying Gene-Environment Interactions in 2014; van Os et al., 2008), even though they remain hidden in current studies (Marigorta and Gibson, 2014). Although SZ and BD are highly heritable conditions, sporadic cases have been calculated to be ~60% (Kendler and Diehl, 1993; GottesmanErlenmeyer-Kimling, 2001). Therefore, GWAS must be viewed within this limitation when formulating models of genetic susceptibility to psychosis (Liou et al., 2010).

Coming from the assurance that a lot of the heritability of SZ derives from common SNPs of small effect, another statistical method attempts to capture the *combined* effect of several SNPs by using a polygenic risk score (International Schizophrenia et al., 2009; Dudbridge, 2013). This score is constructed for each individual as the weighted (OR-based) sum of alleles that were associated (at various p-value cut-offs) with the outcome in a large training sample. In an independent sample, this score is then regressed against the diagnostic status (case or control) and the variance explained is estimated. Although the predictive power and the clinical utility of such method is still low for population screening, it is by far more powerful than single SNP association analyses. It is so far used to provide evidence for the involvement of several markers that are not significant on their own in a typical GWAS sample size and analysis. This method was first applied in SZ (International Schizophrenia et al., 2009), confirming a polygenic component to disease risk, but has also been found in, for example, breast and prostate cancers which may be caused by a much smaller set of genes (Dudbridge, 2013). Also, a polygenic score from a SZ GWAS is likely to be associated with BD, and vice versa, establishing a common polygenic basis. The approach has now been followed by other studies, some of which herein reviewed (Nassan et al., 2017; Bergen et al., 2012; Ikeda et al., 2011; Levinson et al., 2012).

The “genome-wide complex trait analysis (GCTA)” method (Yang, Lee, Goddard, Visscher) has appeared to partially resolve the “missing heritability” problem: that the sum of GWA-identified SNPs explain only a small fraction of heritability. It estimates the variance explained by a constellation of common SNPs from the whole genome for a complex trait, rather than testing the association of any particular SNP to the trait. Using the PGC sample, it was estimated that SNPs account for 23% and 25% of variation in liability to SZ (Lee et al., 2012b) and BD (Cross-Disorder Group of the Psychiatric Genomics, 2013), respectively. They also estimate that 1) this is mainly due to common causal alleles, 2) they must be evenly spread across chromosomes since the variance explained by each chromosome is linearly related to its length, 3) the genetic basis of SZ is the same in males and females and 4) as expected, a disproportionate amount of variation in liability is attributable to a set of 2725 genes expressed in the CNS. Furthermore, using only unrelated subjects and the same SNP genotypes, a 68% genetic *correlation* between these disorders was found. Although most of the SNPs responsible for the variance explained are not yet identified, the rationale is that they will be, as GWAS sample sizes increase and more accurate estimation of the effect size of each SNP is achieved.

A final limitation to keep in mind is the clinical heterogeneity in the samples used, both at the level of the clinical diagnosis of patients, as well as of their socio-demographic characterization. Clinical diagnosis heterogeneity is most probably hampered by the DSM and ICD disease classification systems still having very little correspondence with their biological causes, and by the joining of multiple samples in large studies, whereby each sample is recruited and diagnoses in by different clinicians in different sites, clinical practice cultures, healthcare systems (even in the same country), using almost inevitably, different criteria. This poses concerns in relation to the validity of GWAS results, either in standard analyses or in PRS of GCTA approaches (abovementioned). In specific, the specificity of the results in each disorder, as well as the shared heritability between disorders cannot be reliable ascertained. It is possible that this heterogeneity in samples underlies the share heritability found among disorders. Attempts should be made to improve the clinical and socio-economic-demographic characterization of the samples, including socio-economic-demographic factors as covariates, in new studies/samples and even in published ones.

5. Conclusion

In conclusion, this review has found further support for the strongest gene regions identified in Lee et al. (2012a) review (Lee et al., 2012a): *ANK3* and *MHC*. Most importantly, taking all previously and currently reviewed studies into account, we found that *AMBRA1*, *ANK3*, *ARNTL*, *CDH13*, *EFHD1, MHC*, *PLXNA2* and *UGT1A1* have been implicated in at least two reportedly non-overlapping samples of either SZ or BD, which gives credence to their implication (and the SNP region their respective markers tag) in psychosis, except in *EFHD1*'s case where the allele direction has not been consistent. No further evidence for a shared genetic basis for SZ and BD was found in this review, with *ANK3*

and *PLXNA2* remaining the only GWA-implicated genes in both disorders since the last review. Overall, we also found, when taking the most powerful meta-consortia findings into account, that *ANK3, CACNA1C, CACNA1I, CACNB2, CACNB3, DRD2, GRIA1, GRIN2A, GRM3, ITIH1, ITIH3/ITIH4, MHC, MIR137, ODZ4, SRR, SYNE1, TCF4, VRK2* and *ZNF804A* have emerged as front-runners in terms of susceptibility genes for psychosis.

Even though problems with sample heterogeneity and population stratification exist in terms of both known and unknown variables emerge in large sample sizes, GWAS are useful in elucidating the genetic underpinnings of complex diseases – with replication attempts being fundamental. The difficulty in detecting gene-gene and gene-environment (GottesmanErlenmeyer-Kimling, 2001), as well as the missing-heritability problem, remain as limitations – but are now being tentatively tackled. Novel analytical methods, emerging from genome-wide technologies, such as the polygenic score and the GCTA analyses are being applied to GWAS data in the hope to capture the full degree of genetic influence in psychosis.

Declaration of interest

The authors declare to have no conflicts of interest.

Acknowledgments

We very much thank Bárbara Oliveira, Rita Lóios, Alexandra Cabrita, Senita Rani-Robinson, Salwador Cyranowski and Mateusz Gielata for their contribution to the literature search and manuscript proofreading. We are additionally very grateful to the Reviewers' comments which greatly improved the readability and interest of this manuscript. DP was supported by a National Institute for Health Research fellowship (UK; NIHR-PDF-2010-03-047), an Investigator FCT grant by Fundação para a Ciência e Tecnologia (Portugal; IF/00787/2014) and a Marie Curie Integration Grant by European Commission (EU; 631952-FP7-PEOPLE-2013-CIG). EV was supported by a Guy's & St Thomas Charity Grant (UK). These supported some of the researchers' salary during their research and preparation of the article and had no role in study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the article for publication.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2019.04.007.

References

Aikawa, J., Esko, J.D., 1999. Molecular cloning and expression of a third member of the heparan sulfate/heparin GlcNAc N-deacetylase/N-sulfotransferase family. J. Biol. Chem. 274 (5), 2690–2695.

Ajima, R., Akazawa, H., Kodama, M., et al., 2008. Deficiency of Myo18B in mice results in embryonic lethality with cardiac myofibrillar aberrations. Genes CellsMolecular Cell. Mech. 13 (10), 987–999.

Akbarian, S., Huang, H.S., 2009. Epigenetic regulation in human brain-focus on histone lysine methylation. Biol. Psychiatry 65 (3), 198–203.

Albanna, A., Choudhry, Z., Harvey, P.O., et al., 2014. TCF4 gene polymorphism and cognitive performance in patients with first episode psychosis. Schizophr. Res. 152 (1), 124–129.

Alkelai, A., Lupoli, S., Greenbaum, L., et al., 2011. Identification of new schizophrenia susceptibility loci in an ethnically homogeneous, family-based, Arab-Israeli sample. FASEB J. Off. Pub. Federation Am. Soc. Exp. Biol. 25 (11), 4011–4023.

Alkelai, A., Lupoli, S., Greenbaum, L., et al., 2012. DOCK4 and CEACAM21 as novel schizophrenia candidate genes in the Jewish population. Int. J. Neuropsychopharmacol. Off. Scientific J. Collegium Int. Neuropsychopharmacol. (CINP) 15 (4), 459–469.

Arias, I., Sorlozano, A., Villegas, E., et al., 2012. Infectious agents associated with schizophrenia: a meta-analysis. Schizophr. Res. 136 (1–3), 128–136.

Arias-Vasquez, A., Altink, M.E., Rommelse, N.N., et al., 2011. CDH13 is associated with working memory performance in attention deficit/hyperactivity disorder. Genes Brain Behav. 10 (8), 844–851.

Atanasoski, S., Toldo, S.S., Malipiero, U., Schreiber, E., Fries, R., Fontana, A., 1995. Isolation of the human genomic brain-2/N-Oct 3 gene (POUF3) and assignment to chromosome 6q16. Genomics 26 (2), 272–280.

Athanasiu, L., Mattingsdal, M., Kahler, A.K., et al., 2010. Gene variants associated with schizophrenia in a Norwegian genome-wide study are replicated in a large European cohort. J. Psychiatr. Res. 44 (12), 748–753.

Avram, S., Shaposhnikov, S., Buiu, C., Mernea, M., 2014. Chondroitin sulfate proteoglycans: structure-function relationship with implication in neural development and brain disorders. BioMed Res. Int. 2014, 642798.

Bacchelli, E., Ceroni, F., Pinto, D., et al., 2014. A CTNNA3 compound heterozygous deletion implicates a role for alphaT-catenin in susceptibility to autism spectrum disorder. J. Neurodev. Disord. 6 (1), 17.

Baird, A.L., Coogan, A.N., Siddiqui, A., Donev, R.M., Thome, J., 2012. Adult attention-deficit hyperactivity disorder is associated with alterations in circadian rhythms at the behavioural, endocrine and molecular levels. Mol. Psychiatr. 17 (10), 988–995.

Barbaux, S., Gascoin-Lachambre, G., Buffat, C., et al., 2012. A genome-wide approach reveals novel imprinted genes expressed in the human placenta. Epigenetics 7 (9), 1079–1090.

Baron, O., Forthmann, B., Lee, Y.W., et al., 2012. Cooperation of nuclear fibroblast growth factor receptor 1 and Nurr1 offers new interactive mechanism in postmitotic development of mesencephalic dopaminergic neurons. J. Biol. Chem. 287 (24), 19827–19840.

Basak, S., Desai, D.J., Rho, E.H., Ramos, R., Maurel, P., Kim, H.A., 2015. E-cadherin enhances neuregulin signaling and promotes Schwann cell myelination. Glia 63 (9), 1522–1536.

Baum, A.E., Akula, N., Cabanero, M., et al., 2008a. A genome-wide association study implicates diacylglycerol kinase eta (DGKH) and several other genes in the etiology of bipolar disorder. Mol. Psychiatr. 13 (2), 197–207.

Baum, A.E., Hamshere, M., Green, E., et al., 2008b. Meta-analysis of two genome-wide association studies of bipolar disorder reveals important points of agreement. Mol. Psychiatr. 13, 466.

Bazzoni, G., 2003. The JAM family of junctional adhesion molecules. Curr. Opin. Cell Biol. 15 (5), 525–530.

Beattie, M.S., Hermann, G.E., Rogers, R.C., Bresnahan, J.C., 2002a. Cell death in models of spinal cord injury. Prog. Brain Res. 137, 37–47.

Beattie, E.C., Stellwagen, D., Morishita, W., et al., 2002b. Control of synaptic strength by glial TNFalpha. Science (New York, NY) 295 (5563), 2282–2285.

Beaulieu, J.M., Gainetdinov, R.R., 2011. The physiology, signaling, and pharmacology of dopamine receptors. Pharmacol. Rev. 63 (1), 182–217.

Behrends, C., Sowa, M.E., Gygi, S.P., Harper, J.W., 2010. Network organization of the human autophagy system. Nature 466 (7302), 68–76.

Belmonte Mahon, P., Pirooznia, M., Goes, F.S., et al., 2011. Genome-wide association analysis of age at onset and psychotic symptoms in bipolar disorder. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 156B (3), 370–378.

Belyk, M., Kraft, S.J., Brown, S., 2015. Pediatric Imaging N, Genetics S. PlexinA polymorphisms mediate the developmental trajectory of human corpus callosum microstructure. J. Hum. Genet. 60 (3), 147–150.

Benzel, I., Bansal, A., Browning, B.L., et al., 2007. Interactions among genes in the ErbB-Neuregulin signalling network are associated with increased susceptibility to schizophrenia. Behav. Brain Funct. BBF 3, 31.

Berditchevski, F., Tolias, K.F., Wong, K., Carpenter, C.L., Hemler, M.E., 1997. A novel link between integrins, transmembrane-4 superfamily proteins (CD63 and CD81), and phosphatidylinositol 4-kinase. J. Biol. Chem. 272 (5), 2595–2598.

Bergen, S.E., O'Dushlaine, C.T., Ripke, S., et al., 2012. Genome-wide association study in a Swedish population yields support for greater CNV and MHC involvement in schizophrenia compared with bipolar disorder. Mol. Psychiatr. 17 (9), 880–886.

Betcheva, E.T., Yosifova, A.G., Mushiroda, T., et al., 2013. Whole-genome-wide association study in the Bulgarian population reveals HHAT as schizophrenia susceptibility gene. Psychiatr. Genet. 23 (1), 11–19.

Bi, C., Wu, J., Jiang, T., et al., 2012. Mutations of ANK3 identified by exome sequencing are associated with autism susceptibility. Hum. Mutat. 33 (12), 1635–1638.

Biersmith, B., Liu, Z.C., Bauman, K., Geisbrecht, E.R., 2011. The DOCK protein sponge binds to ELMO and functions in Drosophila embryonic CNS development. PLoS One 6 (1), e16120.

Blake, J., Riddell, A., Theiss, S., et al., 2014. Sequencing of a patient with balanced chromosome abnormalities and neurodevelopmental disease identifies disruption of multiple high risk loci by structural variation. PLoS One 9 (3), e90894.

Blanco, S., Klimcakova, L., Vega, F.M., Lazo, P.A., 2006. The subcellular localization of vaccinia-related kinase-2 (VRK2) isoforms determines their different effect on p53 stability in tumour cell lines. FEBS J. 273 (11), 2487–2504.

Blanco, S., Santos, C., Lazo, P.A., 2007. Vaccinia-related kinase 2 modulates the stress response to hypoxia mediated by TAK1. Mol. Cell Biol. 27 (20), 7273–7283.

Blanco, S., Sanz-Garcia, M., Santos, C.R., Lazo, P.A., 2008. Modulation of interleukin-1 transcriptional response by the interaction between VRK2 and the JIP1 scaffold protein. PLoS One 3 (2), e1660.

Bohlega, S., Al-Ajlan, H., Al-Saif, A., 2014. Mutation of fibulin-1 causes a novel syndrome involving the central nervous system and connective tissues. Eur. J. Hum. Genet. 22 (5), 640–643.

Bolu, A., Oznur, T., Tok, D., et al., 2016. Seropositivity of neurotropic infectious agents in first-episode schizophrenia patients and the relationship with positive and negative symptoms. Psychiatr. Danub. 28 (2), 132–138.

Borglum, A.D., Demontis, D., Grove, J., et al., 2013. Genome-wide study of association and interaction with maternal cytomegalovirus infection suggests new schizophrenia loci. Mol. Psychiatr. 19 (3), 325–333.

Borroto-Escuela, D.O., Narvaez, M., Perez-Alea, M., et al., 2015. Evidence for the

existence of FGFR1-5-HT1A heteroreceptor complexes in the midbrain raphe 5-HT system. Biochem. Biophys. Res. Commun. 456 (1), 489–493.
Bottner, M., Ghorbani, P., Harde, J., et al., 2014. Expression and regulation of reelin and its receptors in the enteric nervous system. Mol. Cell. Neurosci. 61, 23–33.
Bourguiba-Hachemi, S., Ashkanani, T.K., Kadhem, F.J., Almawi, W.Y., Alroughani, R., Fathallah, M.D., 2016. ZFAT gene variant association with multiple sclerosis in the Arabian Gulf population: a genetic basis for gender-associated susceptibility. Mol. Med. Rep. 14 (4), 3543–3550.
Bricker, D.K., Taylor, E.B., Schell, J.C., et al., 2012. A mitochondrial pyruvate carrier required for pyruvate uptake in yeast, Drosophila, and humans. Science (New York, NY) 337 (6090), 96–100.
Bridgman, P.C., 2004. Myosin-dependent transport in neurons. J. Neurobiol. 58 (2), 164–174.
Britsch, S., 2007. The neuregulin-I/ErbB signaling system in development and disease. Adv. Anat. Embryol. Cell Biol. 190, 1–65.
Brockschmidt, A., Todt, U., Ryu, S., et al., 2007. Severe mental retardation with breathing abnormalities (Pitt-Hopkins syndrome) is caused by haploinsufficiency of the neuronal bHLH transcription factor TCF4. Hum. Mol. Genet. 16 (12), 1488–1494.
Bronstein, J.M., 2000. Function of tetraspan proteins in the myelin sheath. Curr. Opin. Neurobiol. 10 (5), 552–557.
Brown, A.S., 2006. Prenatal infection as a risk factor for schizophrenia. Schizophr. Bull. 32 (2), 200–202.
Bundo, M., Toyoshima, M., Okada, Y., et al., 2014. Increased l1 retrotransposition in the neuronal genome in schizophrenia. Neuron 81 (2), 306–313.
Carlsson, T., Schindler, F.R., Hollerhage, M., et al., 2011. Systemic administration of neuregulin-1beta1 protects dopaminergic neurons in a mouse model of Parkinson's disease. J. Neurochem. 117 (6), 1066–1074.
Caruncho, H.J., Brymer, K., Romay-Tallon, R., et al., 2016. Reelin-related disturbances in depression: implications for translational studies. Front. Cell. Neurosci. 10, 48.
Cassidy, C., Buchy, L., Bodnar, M., et al., 2014. Association of a risk allele of ANK3 with cognitive performance and cortical thickness in patients with first-episode psychosis. J. Psychiatry Neurosci. 39 (1), 31–39.
Cecconi, F., Di Bartolomeo, S., Nardacci, R., et al., 2007. A novel role for autophagy in neurodevelopment. Autophagy 3 (5), 506–508.
Chabchoub, E., Vermeesch, J.R., de Ravel, T., de Cock, P., Fryns, J.P., 2008. The facial dysmorphy in the newly recognised microdeletion 2p15-p16.1 refined to a 570 kb region in 2p15. J. Med. Genet. 45 (3), 189–192.
Chamoun, Z., Mann, R.K., Nellen, D., et al., 2001. Skinny hedgehog, an acyltransferase required for palmitoylation and activity of the hedgehog signal. Science (New York, NY) 293 (5537), 2080–2084.
Chang, L., Friedman, J., Ernst, T., Zhong, K., Tsopelas, N.D., Davis, K., 2007. Brain metabolite abnormalities in the white matter of elderly schizophrenic subjects: implication for glial dysfunction. Biol. Psychiatry 62 (12), 1396–1404.
Chang, E.H., Kirtley, A., Chandon, T.S., et al., 2015. Postnatal neurodevelopmental expression and glutamate-dependent regulation of the ZNF804A rodent homologue. Schizophr. Res. 168 (1–2), 402–410.
Chaudhury, A.R., Gerecke, K.M., Wyss, J.M., Morgan, D.G., Gordon, M.N., Carroll, S.L., 2003. Neuregulin-1 and erbB4 immunoreactivity is associated with neuritic plaques in Alzheimer disease brain and in a transgenic model of Alzheimer disease. J. Neuropathol. Exp. Neurol. 62 (1), 42–54.
Chen, G., Masana, M.I., Manji, H.K., 2000. Lithium regulates PKC-mediated intracellular cross-talk and gene expression in the CNS in vivo. Bipolar Disord. 2 (3 Pt 2), 217–236.
Chen, M., Xu, Z., Zhai, J., et al., 2012. Evidence of IQ-modulated association between ZNF804A gene polymorphism and cognitive function in schizophrenia patients. Neuropsychopharmacology 37 (7), 1572–1578.
Chen, S.F., Chao, Y.L., Shen, Y.C., Chen, C.H., Weng, C.F., 2014. Resequencing and association study of the NFKB activating protein-like gene (NKAPL) in schizophrenia. Schizophr. Res. 157 (1–3), 169–174.
Chen, J., Lin, M., Hrabovsky, A., et al., 2015a. ZNF804A transcriptional networks in differentiating neurons derived from induced pluripotent stem cells of human origin. PLoS One 10 (4), e0124597.
Chen, Q., Peng, X.D., Huang, C.Q., Hu, X.Y., Zhang, X.M., 2015b. Association between ARNTL (BMAL1) rs2278749 polymorphism T > C and susceptibility to Alzheimer disease in a Chinese population. Genet Mol Res 14 (4), 18515–18522.
Cho, Y., Ryu, S., Huh, I., et al., 2015. Effects of genetic variations in NRG1 on cognitive domains in patients with schizophrenia and healthy individuals. Psychiatr. Genet. 25 (4), 147–154.
Choi, J., Ababon, M.R., Matteson, P.G., Millonig, J.H., 2012. Cut-like homeobox 1 and nuclear factor I/B mediate ENGRAILED2 autism spectrum disorder-associated haplotype function. Hum. Mol. Genet. 21 (7), 1566–1580.
Chow, T.J., Tee, S.F., Yong, H.S., Tang, P.Y., 2016. Genetic association of TCF4 and AKT1 gene variants with the age at onset of schizophrenia. Neuropsychobiology 73 (4), 233–240.
Cichon, S., Muhleisen, T.W., Degenhardt, F.A., et al., 2011. Genome-wide association study identifies genetic variation in neurocan as a susceptibility factor for bipolar disorder. Am. J. Hum. Genet. 88 (3), 372–381.
Clarke, R.A., Lee, S., Eapen, V., 2012. Pathogenetic model for Tourette syndrome delineates overlap with related neurodevelopmental disorders including Autism. Transl. Psychiatry 2, e158.
Comabella, M., Craig, D.W., Morcillo-Suarez, C., et al., 2009. Genome-wide scan of 500,000 single-nucleotide polymorphisms among responders and nonresponders to interferon beta therapy in multiple sclerosis. Arch. Neurol. 66 (8), 972–978.
Consortium, I.S.G., 2012. Genome-wide association study implicates HLA-C*01:02 as a risk factor at the major histocompatibility complex locus in schizophrenia. Biol. Psychiatry 72 (8), 620–628.
Consortium, E., Consortium, E.M., Steffens, M., et al., 2012. Genome-wide association analysis of genetic generalized epilepsies implicates susceptibility loci at 1q43, 2p16.1, 2q22.3 and 17q21.32. Hum. Mol. Genet. 21 (24), 5359–5372.
Cooley, M.A., Kern, C.B., Fresco, V.M., et al., 2008. Fibulin-1 is required for morphogenesis of neural crest-derived structures. Dev. Biol. 319 (2), 336–345.
Corcoran, M.M., Hammarsund, M., Zhu, C., et al., 2004. DLEU2 encodes an antisense RNA for the putative bicistronic RFP2/LEU5 gene in humans and mouse. Genes Chromosomes Cancer 40 (4), 285–297.
Coric, V., Feldman, H.H., Oren, D.A., et al., 2010. Multicenter, randomized, double-blind, active comparator and placebo-controlled trial of a corticotropin-releasing factor receptor-1 antagonist in generalized anxiety disorder. Depress. Anxiety 27 (5), 417–425.
Correll, C.U., Detraux, J., De Lepeleire, J., De Hert, M., 2015. Effects of antipsychotics, antidepressants and mood stabilizers on risk for physical diseases in people with schizophrenia, depression and bipolar disorder. World Psychiatr. 14 (2), 119–136.
Cote, M., Guillon, G., Payet, M.D., Gallo-Payet, N., 2001. Expression and regulation of adenylyl cyclase isoforms in the human adrenal gland. J. Clin. Endocrinol. Metab. 86 (9), 4495–4503.
Cotta-Ramusino, C., McDonald 3rd, E.R., Hurov, K., Sowa, M.E., Harper, J.W., Elledge, S.J., 2011. A DNA damage response screen identifies RHINO, a 9-1-1 and TopBP1 interacting protein required for ATR signaling. Science (New York, NY) 332 (6035), 1313–1317.
Cross-Disorder Group of the Psychiatric Genomics, C., 2013. Genetic relationship between five psychiatric disorders estimated from genome-wide SNPs. Nat. Genet. 45 (9), 984–994.
Cubelos, B., Sebastian-Serrano, A., Beccari, L., et al., 2010. Cux1 and Cux2 regulate dendritic branching, spine morphology, and synapses of the upper layer neurons of the cortex. Neuron 66 (4), 523–535.
Dailey, L., Ambrosetti, D., Mansukhani, A., Basilico, C., 2005. Mechanisms underlying differential responses to FGF signaling. Cytokine Growth Factor Rev. 16 (2), 233–247.
Dannlowski, U., Kugel, H., Grotegerd, D., et al., 2015. NCAN cross-disorder risk variant is associated with limbic gray matter deficits in healthy subjects and major depression. Neuropsychopharmacology 40 (11), 2510–2516.
Dao, D.T., Mahon, P.B., Cai, X., et al., 2010. Mood disorder susceptibility gene CACNA1C modifies mood-related behaviors in mice and interacts with sex to influence behavior in mice and diagnosis in humans. Biol. Psychiatry 68 (9), 801–810.
Daschil, N., Obermair, G.J., Flucher, B.E., et al., 2013. CaV1.2 calcium channel expression in reactive astrocytes is associated with the formation of amyloid-beta plaques in an Alzheimer's disease mouse model. J. Alzheimer's Dis. 37 (2), 439–451.
Davies, G., Armstrong, N., Bis, J.C., et al., 2015. Genetic contributions to variation in general cognitive function: a meta-analysis of genome-wide association studies in the CHARGE consortium (N = 53949). Mol. Psychiatr. 20 (2), 183–192.
Dazzo, E., Fanciulli, M., Serioli, E., et al., 2015. Heterozygous reelin mutations cause autosomal-dominant lateral temporal epilepsy. Am. J. Hum. Genet. 96 (6), 992–1000.
Delvecchio, G., Dima, D., Frangou, S., 2015. The effect of ANK3 bipolar-risk polymorphisms on the working memory circuitry differs between loci and according to risk-status for bipolar disorder. Am. J. Med. Genet. Part B, Neuropsychiatric genetics : the official publication of the International Society of Psychiatric Genetics 168B (3), 188–196.
Dere, E., Dahm, L., Lu, D., et al., 2014. Heterozygous ambra1 deficiency in mice: a genetic trait with autism-like behavior restricted to the female gender. Front. Behav. Neurosci. 8 181-181.
Deutsch, S.I., Rosse, R.B., Mastropaolo, J., Long, K.D., Gaskins, B.L., 2008. Epigenetic therapeutic strategies for the treatment of neuropsychiatric disorders: ready for prime time? Clin. Neuropharmacol. 31 (2), 104–119.
Dima, D., Jogia, J., Collier, D., Vassos, E., Burdick, K.E., Frangou, S., 2013. Independent modulation of engagement and connectivity of the facial network during affect processing by CACNA1C and ANK3 risk genes for bipolar disorder. JAMA Psychiatry 70 (12), 1303–1311.
Ding, Q., Gros, R., Gray, I.D., Taussig, R., Ferguson, S.S., Feldman, R.D., 2004. Raf kinase activation of adenylyl cyclases: isoform-selective regulation. Mol. Pharmacol. 66 (4), 921–928.
Djurovic, S., Gustafsson, O., Mattingsdal, M., et al., 2010. A genome-wide association study of bipolar disorder in Norwegian individuals, followed by replication in Icelandic sample. J. Affect. Disord. 126 (1–2), 312–316.
Dong, Z., Zhou, L., Del Villar, K., Ghanevati, M., Tashjian, V., Miller, C.A., 2005. JIP1 regulates neuronal apoptosis in response to stress. Brain Res Mol Brain Res 134 (2), 282–293.
Dong, Y., Ding, Y., Cun, Y., Xiao, C., 2013. Association of renin binding protein (RnBP) gene polymorphisms with essential hypertension in the hani minority of southwestern China. J. Genet. Genomics 40 (8), 433–436.
Dow, D.J., Huxley-Jones, J., Hall, J.M., et al., 2011. ADAMTSL3 as a candidate gene for schizophrenia: gene sequencing and ultra-high density association analysis by imputation. Schizophr. Res. 127 (1–3), 28–34.
Dudbridge, F., 2013. Power and predictive accuracy of polygenic risk scores. PLoS Genet. 9 (3), e1003348.
Edwards, T.L., Pericak-Vance, M., Gilbert, J.R., Haines, J.L., Martin, E.R., Ritchie, M.D., 2009. An association analysis of Alzheimer disease candidate genes detects an ancestral risk haplotype clade in ACE and putative multilocus association between ACE, A2M, and LRRTM3. Am. J. Med. Genet. Part B, Neuropsychiatric genetics : the official publication of the International Society of Psychiatric Genetics 150B (5), 721–735.
Esslinger, C., Kirsch, P., Haddad, L., et al., 2011. Cognitive state and connectivity effects of the genome-wide significant psychosis variant in ZNF804A. Neuroimage 54 (3), 2514–2523.
Eswarakumar, V.P., Lax, I., Schlessinger, J., 2005. Cellular signaling by fibroblast growth factor receptors. Cytokine Growth Factor Rev. 16 (2), 139–149.

European Network of National Networks studying Gene-Environment Interactions in, S., van Os, J., Rutten, B.P., et al., 2014. Identifying gene-environment interactions in schizophrenia: contemporary challenges for integrated, large-scale investigations. Schizophr. Bull. 40 (4), 729–736.
Falls, D.L., 2003. Neuregulins: functions, forms, and signaling strategies. Exp. Cell Res. 284 (1), 14–30.
Fanous, A.H., Zhou, B., Aggen, S.H., et al., 2012. Genome-wide association study of clinical dimensions of schizophrenia: polygenic effect on disorganized symptoms. Am. J. Psychiatry 169 (12), 1309–1317.
Ferreira, M.A., O'Donovan, M.C., Meng, Y.A., et al., 2008. Collaborative genome-wide association analysis supports a role for ANK3 and CACNA1C in bipolar disorder. Nat. Genet. 40 (9), 1056–1058.
Fimia, G.M., Stoykova, A., Romagnoli, A., et al., 2007. Ambra1 regulates autophagy and development of the nervous system. Nature 447 (7148), 1121–1125.
Finseth, P.I., Sonderby, I.E., Djurovic, S., et al., 2014. Association analysis between suicidal behaviour and candidate genes of bipolar disorder and schizophrenia. J. Affect. Disord. 163, 110–114.
Flood, J.F., Morley, J.E., 1988. Effects of bombesin and gastrin-releasing peptide on memory processing. Brain Res. 460 (2), 314–322.
Flora, A., Garcia, J.J., Thaller, C., Zoghbi, H.Y., 2007. The E-protein Tcf4 interacts with Math1 to regulate differentiation of a specific subset of neuronal progenitors. Proc. Natl. Acad. Sci. U.S.A. 104 (39), 15382–15387.
Folsom, T.D., Fatemi, S.H., 2013. The involvement of Reelin in neurodevelopmental disorders. Neuropharmacology 68, 122–135.
Franco, S.J., Martinez-Garay, I., Gil-Sanz, C., Harkins-Perry, S.R., Muller, U., 2011. Reelin regulates cadherin function via Dab1/Rap1 to control neuronal migration and lamination in the neocortex. Neuron 69 (3), 482–497.
Freyberg, Z., Ferrando, S.J., Javitch, J.A., 2010. Roles of the Akt/GSK-3 and Wnt signaling pathways in schizophrenia and antipsychotic drug action. Am. J. Psychiatry 167 (4), 388–396.
Fujimoto, T., Doi, K., Koyanagi, M., et al., 2009. ZFAT is an antiapoptotic molecule and critical for cell survival in MOLT-4 cells. FEBS Lett. 583 (3), 568–572.
Fujino, T., Takei, Y.A., Sone, H., et al., 2001. Molecular identification and characterization of two medium-chain acyl-CoA synthetases, MACS1 and the Sa gene product. J. Biol. Chem. 276 (38), 35961–35966.
Funa, K., Sasahara, M., 2014. The roles of PDGF in development and during neurogenesis in the normal and diseased nervous system. J. Neuroimmune Pharmacol. 9 (2), 168–181.
Gaither, L.A., Eide, D.J., 2000. Functional expression of the human hZIP2 zinc transporter. J. Biol. Chem. 275 (8), 5560–5564.
Galvez-Contreras, A.Y., Quinones-Hinojosa, A., Gonzalez-Perez, O., 2013. The role of EGFR and ErbB family related proteins in the oligodendrocyte specification in germinal niches of the adult mammalian brain. Front. Cell. Neurosci. 7, 258.
Ganzola, R., Maziade, M., Duchesne, S., 2014. Hippocampus and amygdala volumes in children and young adults at high-risk of schizophrenia: research synthesis. Schizophr. Res. 156 (1), 76–86.
Garcia-Frigola, C., Burgaya, F., de Lecea, L., Soriano, E., 2001. Pattern of expression of the tetraspanin Tspan-5 during brain development in the mouse. Mech. Dev. 106 (1–2), 207–212.
Gaughran, F., Payne, J., Sedgwick, P.M., Cotter, D., Berry, M., 2006. Hippocampal FGF-2 and FGFR1 mRNA expression in major depression, schizophrenia and bipolar disorder. Brain Res. Bull. 70 (3), 221–227.
Gavin, D.P., Sharma, R.P., 2010. Histone modifications, DNA methylation, and schizophrenia. Neurosci. Biobehav. Rev. 34 (6), 882–888.
Gijon, M.A., Riekhof, W.R., Zarini, S., Murphy, R.C., Voelker, D.R., 2008. Lysophospholipid acyltransferases and arachidonate recycling in human neutrophils. J. Biol. Chem. 283 (44), 30235–30245.
Girgenti, M.J., LoTurco, J.J., Maher, B.J., 2012. ZNF804a regulates expression of the schizophrenia-associated genes PRSS16, COMT, PDE4B, and DRD2. PLoS One 7 (2), e32404.
Glaser, B., Kirov, G., Green, E., Craddock, N., Owen, M.J., 2005. Linkage disequilibrium mapping of bipolar affective disorder at 12q23-q24 provides evidence for association at CUX2 and FLJ32356. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 132B (1), 38–45.
Goes, F.S., Willour, V.L., Zandi, P.P., et al., 2010. Sex-specific association of the Reelin gene with bipolar disorder. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 153B (2), 549–553.
Goes, F.S., McGrath, J., Avramopoulos, D., et al., 2015. Genome-wide association study of schizophrenia in Ashkenazi Jews. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 168 (8), 649–659.
Gonzalez, R., 2014. The relationship between bipolar disorder and biological rhythms. J. Clin. Psychiatry 75 (4), e323–331.
Gonzalez, N., Moody, T.W., Igarashi, H., Ito, T., Jensen, R.T., 2008. Bombesin-related peptides and their receptors: recent advances in their role in physiology and disease states. Curr. Opin. Endocrinol. Diabetes Obes. 15 (1), 58–64.
Gonzalez, R., Gonzalez, S., Villa, E., et al., 2015. Identification of circadian gene variants in bipolar disorder in Latino populations. J. Affect. Disord. 186, 367–375.
Gottesman II, , Erlenmeyer-Kimling, L., 2001. Family and twin strategies as a head start in defining prodromes and endophenotypes for hypothetical early-interventions in schizophrenia. Schizophr. Res. 51 (1), 93–102.
Gottschalk, M.G., Leussis, M.P., Ruland, T., Gjeluci, K., Petryshen, T.L., Bahn, S., 2017. Lithium reverses behavioral and axonal transport-related changes associated with ANK3 bipolar disorder gene disruption. Eur. Neuropsychopharmacol. 27 (3), 274–288.
Green, E.K., Grozeva, D., Jones, I., et al., 2010. The bipolar disorder risk allele at CACNA1C also confers risk of recurrent major depression and of schizophrenia. Mol. Psychiatr. 15 (10), 1016–1022.
Green, E.K., Hamshere, M., Forty, L., et al., 2012a. Replication of bipolar disorder susceptibility alleles and identification of two novel genome-wide significant associations in a new bipolar disorder case-control sample. Mol. Psychiatr. 18 (12), 1302–1307.
Green, E.K., Grozeva, D., Forty, L., et al., 2012b. Association at SYNE1 in both bipolar disorder and recurrent major depression. Mol. Psychiatr. 18, 614–617.
Greenwood, T.A., Kelsoe, J.R., 2013. Genome-wide association study of irritable vs. elated mania suggests genetic differences between clinical subtypes of bipolar disorder. PLoS One 8 (1), e53804.
Greenwood, T.A., Akiskal, H.S., Akiskal, K.K., Kelsoe, J.R., 2012. Genome-wide association study of temperament in bipolar disorder reveals significant associations with three novel Loci. Biol. Psychiatry 72 (4), 303–310.
Griswold, A.J., Ma, D., Cukier, H.N., et al., 2012. Evaluation of copy number variations reveals novel candidate genes in autism spectrum disorder-associated pathways. Hum. Mol. Genet. 21 (15), 3513–3523.
Gronli, O., Kvamme, J.M., Friborg, O., Wynn, R., 2013. Zinc deficiency is common in several psychiatric disorders. PLoS One 8 (12), e82793.
Gu, Z., Wang, B., Zhang, Y.B., et al., 2015. Association of ARNTL and PER1 genes with Parkinson's disease: a case-control study of Han Chinese. Sci. Rep. 5, 15891.
Gurung, R., Prata, D.P., 2015. What is the impact of genome-wide supported risk variants for schizophrenia and bipolar disorder on brain structure and function? A systematic review. Psychol. Med. 45 (12), 2461–2480.
Haarman, B.C., Riemersma-Van der Lek, R.F., de Groot, J.C., et al., 2014. Neuroinflammation in bipolar disorder - a [(11)C]-(R)-PK11195 positron emission tomography study. Brain Behav. Immun. 40, 219–225.
Hall, N.G., Klenotic, P., Anand-Apte, B., Apte, S.S., 2003. ADAMTSL-3/punctin-2, a novel glycoprotein in extracellular matrix related to the ADAMTS family of metalloproteases. Matrix Biol. 22 (6), 501–510.
Hallahan, B., Newell, J., Soares, J.C., et al., 2011. Structural magnetic resonance imaging in bipolar disorder: an international collaborative mega-analysis of individual adult patient data. Biol. Psychiatry 69 (4), 326–335.
Hamel, M.G., Ajmo, J.M., Leonardo, C.C., Zuo, F., Sandy, J.D., Gottschall, P.E., 2008. Multimodal signaling by the ADAMTSs (a disintegrin and metalloproteinase with thrombospondin motifs) promotes neurite extension. Exp. Neurol. 210 (2), 428–440.
Hart, A.B., Engelhardt, B.E., Wardle, M.C., et al., 2012. Genome-wide association study of d-amphetamine response in healthy volunteers identifies putative associations, including cadherin 13 (CDH13). PLoS One 7 (8), e42646.
Hattori, E., Toyota, T., Ishitsuka, Y., et al., 2009. Preliminary genome-wide association study of bipolar disorder in the Japanese population. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 150B (8), 1110–1117.
Hayashi, A., Le Gal, K., Sodersten, K., Vizlin-Hodzic, D., Agren, H., Funa, K., 2015. Calcium-dependent intracellular signal pathways in primary cultured adipocytes and ANK3 gene variation in patients with bipolar disorder and healthy controls. Mol. Psychiatr. 20 (8), 931–940.
He, G., Liu, X., Qin, W., et al., 2006. MPZL1/PZR, a novel candidate predisposing schizophrenia in Han Chinese. Mol. Psychiatr. 11 (8), 748–751.
Heinrich, A., Nees, F., Lourdusamy, A., et al., 2013a. From gene to brain to behavior: schizophrenia-associated variation in AMBRA1 alters impulsivity-related traits. Eur. J. Neurosci. 38 (6), 2941–2945.
Heinrich, A., Lourdusamy, A., Tzschoppe, J., et al., 2013b. The risk variant in ODZ4 for bipolar disorder impacts on amygdala activation during reward processing. Bipolar Disord. 15 (4), 440–445.
Hellevuo, K., Yoshimura, M., Kao, M., Hoffman, P.L., Cooper, D.M., Tabakoff, B., 1993. A novel adenylyl cyclase sequence cloned from the human erythroleukemia cell line. Biochem. Biophys. Res. Commun. 192 (1), 311–318.
Hellwig, K., Kvartsberg, H., Portelius, E., et al., 2015. Neurogranin and YKL-40: independent markers of synaptic degeneration and neuroinflammation in Alzheimer's disease. Alzheimer's Res. Ther. 7, 74.
Higa, L.A., Banks, D., Wu, M., Kobayashi, R., Sun, H., Zhang, H., 2006. L2DTL/CDT2 interacts with the CUL4/DDB1 complex and PCNA and regulates CDT1 proteolysis in response to DNA damage. Cell Cycle 5 (15), 1675–1680.
Higgs, B.W., Elashoff, M., Richman, S., Barci, B., 2006. An online database for brain disease research. BMC Genomics 7, 70.
Hisaoka, K., Tsuchioka, M., Yano, R., et al., 2011. Tricyclic antidepressant amitriptyline activates fibroblast growth factor receptor signaling in glial cells: involvement in glial cell line-derived neurotrophic factor production. J. Biol. Chem. 286 (24), 21118–21128.
Homa, N.J., Salinas, R., Forte, E., Robinson, T.J., Garcia-Blanco, M.A., Luftig, M.A., 2013. Epstein-Barr virus induces global changes in cellular mRNA isoform usage that are important for the maintenance of latency. J. Virol. 87 (22), 12291–12301.
Hor, H., Francescatto, L., Bartesaghi, L., et al., 2015. Missense mutations in TENM4, a regulator of axon guidance and central myelination, cause essential tremor. Hum. Mol. Genet. 24 (20), 5677–5686.
Horrobin, D.F., Glen, A.I., Hudson, C.J., 1995. Possible relevance of phospholipid abnormalities and genetic interactions in psychiatric disorders: the relationship between dyslexia and schizophrenia. Med. Hypotheses 45 (6), 605–613.
Hoseth, E.Z., Ueland, T., Dieset, I., et al., 2017, 43(4):881–890. A study of TNF pathway activation in schizophrenia and bipolar disorder in plasma and brain tissue. Schizophr. Bull. 43 (4), 881–890.
Hou, L., Bergen, S.E., Akula, N., et al., 2016a. Genome-wide association study of 40,000 individuals identifies two novel loci associated with bipolar disorder. Hum. Mol. Genet. 25 (15), 3383–3394.
Hou, T., Jian, C., Xu, J., et al., 2016b. Identification of EFHD1 as a novel Ca(2+) sensor for mitoflash activation. Cell Calcium 59 (5), 262–270.
Hovatta, I., Varilo, T., Suvisaari, J., et al., 1999. A genomewide screen for schizophrenia

genes in an isolated Finnish subpopulation, suggesting multiple susceptibility loci. Am. J. Hum. Genet. 65 (4), 1114–1124.
Hozumi, Y., Watanabe, M., Otani, K., Goto, K., 2009. Diacylglycerol kinase beta promotes dendritic outgrowth and spine maturation in developing hippocampal neurons. BMC Neurosci. 10, 99.
Van Humbeeck, C., Cornelissen, T., Hofkens, H., et al., 2011. Parkin interacts with Ambra1 to induce mitophagy. J. Neurosci. 31 (28), 10249–10261.
Ikeda, M., Aleksic, B., Kirov, G., et al., 2010. Copy number variation in schizophrenia in the Japanese population. Biol. Psychiatry 67 (3), 283–286.
Ikeda, M., Aleksic, B., Kinoshita, Y., et al., 2011. Genome-wide association study of schizophrenia in a Japanese population. Biol. Psychiatry 69 (5), 472–478.
Imbrici, P., Camerino, D.C., Tricarico, D., 2013. Major channels involved in neuropsychiatric disorders and therapeutic perspectives. Front. Genet. 4, 76.
Inatani, M., Irie, F., Plump, A.S., Tessier-Lavigne, M., Yamaguchi, Y., 2003. Mammalian brain morphogenesis and midline axon guidance require heparan sulfate. Science (New York, NY) 302 (5647), 1044–1046.
International Schizophrenia, C., Purcell, S.M., Wray, N.R., et al., 2009. Common polygenic variation contributes to risk of schizophrenia and bipolar disorder. Nature 460 (7256), 748–752.
Irie, F., Okuno, M., Matsumoto, K., Pasquale, E.B., Yamaguchi, Y., 2008. Heparan sulfate regulates ephrin-A3/EphA receptor signaling. Proc. Natl. Acad. Sci. U.S.A. 105 (34), 12307–12312.
Irish Schizophrenia Genomics, C., the Wellcome Trust Case Control, C., 2012. Genome-wide association study implicates HLA-C*01:02 as a risk factor at the major histocompatibility complex locus in schizophrenia. Biol. Psychiatry 72 (8), 620–628.
Ishikawa-Brush, Y., Powell, J.F., Bolton, P., et al., 1997. Autism and multiple exostoses associated with an X;8 translocation occurring within the GRPR gene and 3' to the SDC2 gene. Hum. Mol. Genet. 6 (8), 1241–1250.
Jakobsson, J., Palsson, E., Sellgren, C., et al., 2016. CACNA1C polymorphism and altered phosphorylation of tau in bipolar disorder. Br. J. Psychiatry 208 (2), 195–196.
Janssens, B., Goossens, S., Staes, K., et al., 2001. alphaT-catenin: a novel tissue-specific beta-catenin-binding protein mediating strong cell-cell adhesion. J. Cell Sci. 114 (Pt 17), 3177–3188.
Jenkins, S.M., Bennett, V., 2002. Developing nodes of Ranvier are defined by ankyrin-G clustering and are independent of paranodal axoglial adhesion. Proc. Natl. Acad. Sci. U.S.A. 99 (4), 2303–2308.
Jenkins, T.A., Allen, A.M., Chai, S.Y., MacGregor, D.P., Paxinos, G., Mendelsohn, F.A., 1996. Interactions of angiotensin II with central dopamine. Adv. Exp. Med. Biol. 396, 93–103.
Johnson, C.P., Follmer, R.L., Oguz, I., et al., 2015. Brain abnormalities in bipolar disorder detected by quantitative T1rho mapping. Mol. Psychiatr. 20 (2), 201–206.
Jonas, R.K., Montojo, C.A., Bearden, C.E., 2014. The 22q11.2 deletion syndrome as a window into complex neuropsychiatric disorders over the lifespan. Biol. Psychiatry 75 (5), 351–360.
Jun, G., Asai, H., Zeldich, E., et al., 2014. PLXNA4 is associated with Alzheimer disease and modulates tau phosphorylation. Ann. Neurol. 76 (3), 379–392.
Kahler, A.K., Otnaess, M.K., Wirgenes, K.V., et al., 2010. Association study of PDE4B gene variants in Scandinavian schizophrenia and bipolar disorder multicenter case-control samples. Am. J. Med. Genet. Part B, Neuropsychiatric genetics : the official publication of the International Society of Psychiatric Genetics 153B (1), 86–96.
Kanazawa, T., Ikeda, M., Glatt, S.J., et al., 2013. Genome-wide association study of atypical psychosis. Am. J. Med. Genet. Part B, Neuropsychiatric genetics : the official publication of the International Society of Psychiatric Genetics 162B (7), 679–686.
Kasher, P.R., Schertz, K.E., Thomas, M., et al., 2016. Small 6q16.1 deletions encompassing POU3F2 cause susceptibility to obesity and variable developmental delay with intellectual disability. Am. J. Hum. Genet. 98 (2), 363–372.
Katoh, Y., Katoh, M., 2005. Hedgehog signaling pathway and gastric cancer. Cancer Biol. Ther. 4 (10), 1050–1054.
Kempton, M.J., Ruberto, G., Vassos, E., et al., 2009. Effects of the CACNA1C risk allele for bipolar disorder on cerebral gray matter volume in healthy individuals. Am. J. Psychiatry 166 (12), 1413–1414.
Kendler, K.S., Diehl, S.R., 1993. The genetics of schizophrenia: a current, genetic-epidemiologic perspective. Schizophr. Bull. 19 (2), 261–285.
Kent, W.J., Sugnet, C.W., Furey, T.S., et al., 2002. The human genome browser at UCSC. Genome Res. 12 (6), 996–1006.
Kessler, R.C., Chiu, W., Demler, O., Walters, E.E., 2005. PRevalence, severity, and comorbidity of 12-month dsm-iv disorders in the national comorbidity survey replication. Arch. Gen. Psychiatr. 62 (6), 617–627.
Khachigian, L.M., Santiago, F.S., Rafty, L.A., et al., 1999. GC factor 2 represses platelet-derived growth factor A-chain gene transcription and is itself induced by arterial injury. Circ. Res. 84 (11), 1258–1267.
Khandaker, G.M., Stochl, J., Zammit, S., Lewis, G., Jones, P.B., 2014. Childhood Epstein-Barr Virus infection and subsequent risk of psychotic experiences in adolescence: a population-based prospective serological study. Schizophr. Res. 158 (1–3), 19–24.
Kim, S.M., Kee, H.J., Eom, G.H., et al., 2006. Characterization of a novel WHSC1-associated SET domain protein with H3K4 and H3K27 methyltransferase activity. Biochem. Biophys. Res. Commun. 345 (1), 318–323.
Kim, H.I., Lee, H.J., Cho, C.H., et al., 2015. Association of CLOCK, ARNTL, and NPAS2 gene polymorphisms and seasonal variations in mood and behavior. Chronobiol. Int. 32 (6), 785–791.
King, I.L., Dickendesher, T.L., Segal, B.M., 2009. Circulating Ly-6C+ myeloid precursors migrate to the CNS and play a pathogenic role during autoimmune demyelinating disease. Blood 113 (14), 3190–3197.
Kirov, G., Gumus, D., Chen, W., et al., 2008a. Comparative genome hybridization suggests a role for NRXN1 and APBA2 in schizophrenia. Hum. Mol. Genet. 17 (3), 458–465.
Kirov, G., Zaharieva, I., Georgieva, L., et al., 2008b. A genome-wide association study in 574 schizophrenia trios using DNA pooling. Mol. Psychiatr. 14, 796.
Kirov, G., Grozeva, D., Norton, N., et al., 2009. Support for the involvement of large copy number variants in the pathogenesis of schizophrenia. Hum. Mol. Genet. 18 (8), 1497–1503.
Kitazawa, M., Ohnuma, T., Takebayashi, Y., et al., 2012. No associations found between the genes situated at 6p22.1, HIST1H2BJ, PRSS16, and PGBD1 in Japanese patients diagnosed with schizophrenia. Am. J. Med. Genet. Part B Neuropsych. Genetics Off. Pub. Int. Soc. Psychiatric Genetics 159B (4), 456–464.
Kittel-Schneider, S., Wobrock, T., Scherk, H., et al., 2015. Influence of DGKH variants on amygdala volume in patients with bipolar affective disorder and schizophrenia. Eur. Arch. Psychiatry Clin. Neurosci. 265 (2), 127–136.
Klauck, T.M., Xu, X., Mousseau, B., Jaken, S., 1996. Cloning and characterization of a glucocorticoid-induced diacylglycerol kinase. J. Biol. Chem. 271 (33), 19781–19788.
Klejbor, I., Myers, J.M., Hausknecht, K., et al., 2006. Fibroblast growth factor receptor signaling affects development and function of dopamine neurons - inhibition results in a schizophrenia-like syndrome in transgenic mice. J. Neurochem. 97 (5), 1243–1258.
Klionsky, D.J., Abdelmohsen, K., Abe, A., et al., 2016. Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy 12 (1), 1–222.
Knable, M.B., Barci, B.M., Webster, M.J., Meador-Woodruff, J., Torrey, E.F., Stanley Neuropathology, C., 2004. Molecular abnormalities of the hippocampus in severe psychiatric illness: postmortem findings from the Stanley Neuropathology Consortium. Mol. Psychiatr. 9 (6), 609–620 544.
Kraus, D.M., Elliott, G.S., Chute, H., et al., 2006. CSMD1 is a novel multiple domain complement-regulatory protein highly expressed in the central nervous system and epithelial tissues. J. Immunol. 176 (7), 4419–4430.
Krug, A., Nieratschker, V., Markov, V., et al., 2010. Effect of CACNA1C rs1006737 on neural correlates of verbal fluency in healthy individuals. Neuroimage 49 (2), 1831–1836.
Kutsuno, Y., Hirashima, R., Sakamoto, M., et al., 2015. Expression of UDP-glucuronosyltransferase 1 (UGT1) and glucuronidation activity toward endogenous substances in humanized UGT1 mouse brain. Drug Metab. Dispos. 43 (7), 1071–1076.
Kwan, K.Y., Sestan, N., Anton, E.S., 2012. Transcriptional co-regulation of neuronal migration and laminar identity in the neocortex. Development (Camb.) 139 (9), 1535–1546.
Lancaster, T.M., Heerey, E.A., Mantripragada, K., Linden, D.E., 2014. CACNA1C risk variant affects reward responsiveness in healthy individuals. Transl. Psychiatry 4, e461.
Lee, M.S., Lowe, G., Flanagan, S., Kuchler, K., Glackin, C.A., 2000. Human Dermo-1 has attributes similar to twist in early bone development. Bone 27 (5), 591–602.
Lee, K.W., Woon, P.S., Teo, Y.Y., Sim, K., 2012a. Genome wide association studies (GWAS) and copy number variation (CNV) studies of the major psychoses: what have we learnt? Neurosci. Biobehav. Rev. 36 (1), 556–571.
Lee, S.H., DeCandia, T.R., Ripke, S., et al., 2012b. Estimating the proportion of variation in susceptibility to schizophrenia captured by common SNPs. Nat. Genet. 44 (3), 247–250.
Lee, A.S., De Jesus-Cortes, H., Kabir, Z.D., et al., 2016. The neuropsychiatric disease-associated gene cacna1c mediates survival of young hippocampal neurons. eNeuro 3 (2).
Lein, E.S., Hawrylycz, M.J., Ao, N., et al., 2007. Genome-wide atlas of gene expression in the adult mouse brain. Nature 445 (7124), 168–176.
Lencz, T., Morgan, T.V., Athanasiou, M., et al., 2007. Converging evidence for a pseudoautosomal cytokine receptor gene locus in schizophrenia. Mol. Psychiatr. 12 (6), 572–580.
Lencz, T., Guha, S., Liu, C., et al., 2013. Genome-wide association study implicates NDST3 in schizophrenia and bipolar disorder. Nat. Commun. 4, 2739.
Lennertz, L., Quednow, B.B., Benninghoff, J., Wagner, M., Maier, W., Mossner, R., 2011. Impact of TCF4 on the genetics of schizophrenia. Eur. Arch. Psychiatry Clin. Neurosci. 261 (Suppl. 2), S161–S165.
de Leon, J., 2003. Glucuronidation enzymes, genes and psychiatry. Int. J. Neuropsychopharmacol./Off. Scientific J. Collegium Int. Neuropsychopharmacol. (CINP) 6 (1), 57–72.
Levinson, D.F., Duan, J., Oh, S., et al., 2011. Copy number variants in schizophrenia: confirmation of five previous findings and new evidence for 3q29 microdeletions and VIPR2 duplications. Am. J. Psychiatry 168 (3), 302–316.
Levinson, D.F., Shi, J., Wang, K., et al., 2012. Genome-wide association study of multiplex schizophrenia pedigrees. Am. J. Psychiatry 169 (9), 963–973.
Li, L.Y., Liu, M.Y., Shih, H.M., Tsai, C.H., Chen, J.Y., 2006. Human cellular protein VRK2 interacts specifically with Epstein-Barr virus BHRF1, a homologue of Bcl-2, and enhances cell survival. J. Gen. Virol. 87 (Pt 10), 2869–2878.
Li, J., Wang, Y., Fan, X., et al., 2007. ZNF307, a novel zinc finger gene suppresses p53 and p21 pathway. Biochem. Biophys. Res. Commun. 363 (4), 895–900.
Li, M., Wang, Y., Zheng, X.B., et al., 2012. Meta-analysis and brain imaging data support the involvement of VRK2 (rs2312147) in schizophrenia susceptibility. Schizophr. Res. 142 (1–3), 200–205.
Li, J., Zhao, L., You, Y., et al., 2015. Schizophrenia related variants in CACNA1C also confer risk of autism. PLoS One 10 (7), e0133247.
Liang, X., Martin, E.R., Schnetz-Boutaud, N., et al., 2007. Effect of heterogeneity on the chromosome 10 risk in late-onset Alzheimer disease. Hum. Mutat. 28 (11), 1065–1073.
Liang, S., Wang, X.L., Zou, M.Y., et al., 2014. Family-based association study of ZNF533, DOCK4 and IMMP2L gene polymorphisms linked to autism in a northeastern Chinese Han population. J. Zhejiang Univ. - Sci. B 15 (3), 264–271.
Liberati, A., Altman, D.G., Tetzlaff, J., et al., 2009. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. PLoS Med. 6 (7), e1000100.

Lichtenstein, P., Yip, B.H., Björk, C., et al., 2009. Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: a population-based study. Lancet 373 (9659), 234–239.
Lim, C.H., Zain, S.M., Reynolds, G.P., et al., 2014. Genetic association of LMAN2L gene in schizophrenia and bipolar disorder and its interaction with ANK3 gene polymorphism. Progress in neuro-psychopharmacology & biological psychiatry 54, 157–162.
Lin, P.-I., Vance, J.M., Pericak-Vance, M.A., Martin, E.R., 2007. No gene is an island: the flip-flop phenomenon. Am. J. Hum. Genet. 80 (3), 531–538.
Liou, Y.J., Bai, Y.M., Lin, E., et al., 2010. Gene–gene interactions of the INSIG1 and INSIG2 in metabolic syndrome in schizophrenic patients treated with atypical antipsychotics. Pharmacogenomics J. 12, 54.
Liu, Y., Chen, G., Norton, N., et al., 2009. Whole genome association study in a homogenous population in Shandong peninsula of China reveals JARID2 as a susceptibility gene for schizophrenia. J. Biomed. Biotechnol. 2009, 536918.
Liu, H., Fan, G., Xu, K., Wang, F., 2011. Changes in cerebellar functional connectivity and anatomical connectivity in schizophrenia: a combined resting-state functional MRI and diffusion tensor imaging study. J. Magn. Reson. Imaging 34 (6), 1430–1438.
Logue, M.W., Solovieff, N., Leussis, M.P., et al., 2013. The ankyrin-3 gene is associated with posttraumatic stress disorder and externalizing comorbidity. Psychoneuroendocrinology 38 (10), 2249–2257.
Lonic, A., Powell, J.A., Kong, Y., et al., 2013. Phosphorylation of serine 779 in fibroblast growth factor receptor 1 and 2 by protein kinase C(epsilon) regulates Ras/mitogen-activated protein kinase signaling and neuronal differentiation. J. Biol. Chem. 288 (21), 14874–14885.
Lucido, A.L., Suarez Sanchez, F., Thostrup, P., et al., 2009. Rapid assembly of functional presynaptic boutons triggered by adhesive contacts. J. Neurosci. 29 (40), 12449–12466.
Ludwig, B., Dwivedi, Y., 2016. Dissecting bipolar disorder complexity through epigenomic approach. Mol. Psychiatr. 21 (11), 1490–1498.
Ludwig, K.U., Samann, P., Alexander, M., et al., 2013. A common variant in myosin-18B contributes to mathematical abilities in children with dyslexia and intraparietal sulcus variability in adults. Transl. Psychiatry 3, e229.
Lv, N., Qu, J., Long, H., et al., 2015a. Association study between polymorphisms in the CACNA1A, CACNA1C, and CACNA1H genes and drug-resistant epilepsy in the Chinese Han population. Seizure 30, 64–69.
Lv, M.H., Tan, Y.L., Yan, S.X., et al., 2015b. Decreased serum TNF-alpha levels in chronic schizophrenia patients on long-term antipsychotics: correlation with psychopathology and cognition. Psychopharmacology (Berlin) 232 (1), 165–172.
Mah, S., Nelson, M.R., Delisi, L.E., et al., 2006. Identification of the semaphorin receptor PLXNA2 as a candidate for susceptibility to schizophrenia. Mol. Psychiatr. 11 (5), 471–478.
Maiweilidan, Y., Klauza, I., Kordeli, E., 2011. Novel interactions of ankyrins-G at the costameres: the muscle-specific Obscurin/Titin-Binding-related Domain (OTBD) binds plectin and filamin C. Exp. Cell Res. 317 (6), 724–736.
Malhotra, D., Sebat, J., 2012. CNVs: harbingers of a rare variant revolution in psychiatric genetics. Cell 148 (6), 1223–1241.
Marigorta, U.M., Gibson, G., 2014. A simulation study of gene-by-environment interactions in GWAS implies ample hidden effects. Front. Genet. 5, 225.
Van de Mark, D., Kong, D., Loncarek, J., Stearns, T., 2015. MDM1 is a microtubule-binding protein that negatively regulates centriole duplication. Mol. Biol. Cell 26 (21), 3788–3802.
Martin, E.R., Bronson, P.G., Li, Y.J., et al., 2005. Interaction between the alpha-T catenin gene (VR22) and APOE in Alzheimer's disease. J. Med. Genet. 42 (10), 787–792.
Martinez de Arrieta, C., Morte, B., Coloma, A., Bernal, J., 1999. The human RC3 gene homolog, NRGN contains a thyroid hormone-responsive element located in the first intron. Endocrinology 140 (1), 335–343.
Martinez-Cerdeno, V., Galazo, M.J., Cavada, C., Clasca, F., 2002. Reelin immunoreactivity in the adult primate brain: intracellular localization in projecting and local circuit neurons of the cerebral cortex, hippocampus and subcortical regions. Cerebr. Cortex 12 (12), 1298–1311.
McCarthy, S.E., Makarov, V., Kirov, G., et al., 2009. Microduplications of 16p11.2 are associated with schizophrenia. Nat. Genet. 41 (11), 1223–1227.
McCarthy, M.J., Le Roux, M.J., Wei, H., Beesley, S., Kelsoe, J.R., Welsh, D.K., 2016. Calcium channel genes associated with bipolar disorder modulate lithium's amplification of circadian rhythms. Neuropharmacology 101, 439–448.
McCoy, J.G., Avery, D.D., 1990. Bombesin: potential integrative peptide for feeding and satiety. Peptides 11 (3), 595–607.
McMahon, F.J., Akula, N., Schulze, T.G., et al., 2010. Meta-analysis of genome-wide association data identifies a risk locus for major mood disorders on 3p21.1. Nat. Genet. 42 (2), 128–131.
Meier, S., Mattheisen, M., Vassos, E., et al., 2012. Genome-wide significant association between a 'negative mood delusions' dimension in bipolar disorder and genetic variation on chromosome 3q26.1. Transl. Psychiatry 2, e165.
Meller, C.A., Henriques, J.A., Schwartsmann, G., Roesler, R., 2004. The bombesin/gastrin releasing peptide receptor antagonist RC-3095 blocks apomorphine but not MK-801-induced stereotypy in mice. Peptides 25 (4), 585–588.
Merali, Z., Bedard, T., Andrews, N., et al., 2006. Bombesin receptors as a novel anti-anxiety therapeutic target: BB1 receptor actions on anxiety through alterations of serotonin activity. J. Neurosci. 26 (41), 10387–10396.
Merikangas, K.R., Pato, M., 2009. Recent developments in the epidemiology of bipolar disorder in adults and children: magnitude, correlates, and future directions. Clin. Psychol. Sci. Pract. 16 (2), 121–133.
Minamino, N., Kangawa, K., Matsuo, H., 1983. Neuromedin B: a novel bombesin-like peptide identified in porcine spinal cord. Biochem. Biophys. Res. Commun. 114 (2), 541–548.
Mitchell, A.J., Vancampfort, D., Sweers, K., van Winkel, R., Yu, W., De Hert, M., 2013. Prevalence of metabolic syndrome and metabolic abnormalities in schizophrenia and related disorders–a systematic review and meta-analysis. Schizophr. Bull. 39 (2), 306–318.
Miyamoto, Y., Yamauchi, J., 2010. Cellular signaling of Dock family proteins in neural function. Cell. Signal. 22 (2), 175–182.
Miyashita, A., Arai, H., Asada, T., et al., 2007. Genetic association of CTNNA3 with late-onset Alzheimer's disease in females. Hum. Mol. Genet. 16 (23), 2854–2869.
Mochida, G.H., Ganesh, V.S., Felie, J.M., et al., 2010. A homozygous mutation in the tight-junction protein JAM3 causes hemorrhagic destruction of the brain, subependymal calcification, and congenital cataracts. Am. J. Hum. Genet. 87 (6), 882–889.
Modinos, G., Iyegbe, C., Prata, D., et al., 2013. Molecular genetic gene-environment studies using candidate genes in schizophrenia: a systematic review. Schizophr. Res. 150 (2–3), 356–365.
Mohammadi, M., Dikic, I., Sorokin, A., Burgess, W.H., Jaye, M., Schlessinger, J., 1996. Identification of six novel autophosphorylation sites on fibroblast growth factor receptor 1 and elucidation of their importance in receptor activation and signal transduction. Mol. Cell Biol. 16 (3), 977–989.
Mohnke, S., Erk, S., Schnell, K., et al., 2014. Further evidence for the impact of a genome-wide-supported psychosis risk variant in ZNF804A on the Theory of Mind Network. Neuropsychopharmacology 39 (5), 1196–1205.
Mokhtari, R., Lachman, H.M., 2016. The major histocompatibility complex (MHC) in schizophrenia: a review. J. Clin. Cell. Immunol. 7 (6).
Mons, N., Guillou, J.L., Decorte, L., Jaffard, R., 2003. Spatial learning induces differential changes in calcium/calmodulin-stimulated (ACI) and calcium-insensitive (ACII) adenylyl cyclases in the mouse hippocampus. Neurobiol. Learn. Mem. 79 (3), 226–235.
Moosmang, S., Haider, N., Klugbauer, N., et al., 2005. Role of hippocampal Cav1.2 Ca2+ channels in NMDA receptor-independent synaptic plasticity and spatial memory. J. Neurosci. 25 (43), 9883–9892.
Morar, B., Schwab, S.G., Albus, M., Maier, W., Lerer, B., Wildenauer, D.B., 2007. Evaluation of association of SNPs in the TNF alpha gene region with schizophrenia. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 144B (3), 318–324.
Morgan, A.R., Hamilton, G., Turic, D., et al., 2008. Association analysis of 528 intra-genic SNPs in a region of chromosome 10 linked to late onset Alzheimer's disease. Am. J. Med. Genet. Part B, Neuropsychiatric genetics : the official publication of the International Society of Psychiatric Genetics 147B (6), 727–731.
Moskvina, V., Craddock, N., Holmans, P., et al., 2009. Gene-wide analyses of genome-wide association data sets: evidence for multiple common risk alleles for schizophrenia and bipolar disorder and for overlap in genetic risk. Mol. Psychiatr. 14 (3), 252–260.
Moya, P.R., Murphy, D.L., McMahon, F.J., Wendland, J.R., 2010. Increased gene expression of diacylglycerol kinase eta in bipolar disorder. Int. J. Neuropsychopharmacol./Off. Scientific J. Collegium Int. Neuropsychopharmacol. (CINP) 13 (8), 1127–1128.
Mueller, B., Ahnert, P., Burkhardt, J., et al., 2014. Genetic risk variants for dyslexia on chromosome 18 in a German cohort. Genes Brain Behav. 13 (3), 350–356.
Muhleisen, T.W., Mattheisen, M., Strohmaier, J., et al., 2012. Association between schizophrenia and common variation in neurocan (NCAN), a genetic risk factor for bipolar disorder. Schizophr. Res. 138 (1), 69–73.
Muhleisen, T.W., Leber, M., Schulze, T.G., et al., 2014. Genome-wide association study reveals two new risk loci for bipolar disorder. Nat. Commun. 5, 3339.
Mullen, T.E., Marzluff, W.F., 2008. Degradation of histone mRNA requires oligouridylation followed by decapping and simultaneous degradation of the mRNA both 5' to 3' and 3' to 5'. Genes Dev. 22 (1), 50–65.
Murakami, T., Sakane, F., Imai, S., Houkin, K., Kanoh, H., 2003. Identification and characterization of two splice variants of human diacylglycerol kinase eta. J. Biol. Chem. 278 (36), 34364–34372.
Nakamura, E., Kadomatsu, K., Yuasa, S., et al., 1998. Disruption of the midkine gene (Mdk) resulted in altered expression of a calcium binding protein in the hippocampus of infant mice and their abnormal behaviour. Genes CellsMolecular Cell. Mech. 3 (12), 811–822.
Nassan, M., Li, Q., Croarkin, P.E., et al., 2017. A genome wide association study suggests the association of muskelin with early onset bipolar disorder: implications for a GABAergic epileptogenic neurogenesis model. J. Affect. Disord. 208, 120–129.
Need, A.C., Ge, D., Weale, M.E., et al., 2009. A genome-wide investigation of SNPs and CNVs in schizophrenia. PLoS Genet. 5 (2), e1000373.
Needleman, L.A., Liu, X.B., El-Sabeawy, F., Jones, E.G., McAllister, A.K., 2010. MHC class I molecules are present both pre- and postsynaptically in the visual cortex during postnatal development and in adulthood. Proc. Natl. Acad. Sci. U.S.A. 107 (39), 16999–17004.
van Nieuwenhuijze, A., Koenders, M., Roeleveld, D., Sleeman, M.A., van den Berg, W., Wicks, I.P., 2013. GM-CSF as a therapeutic target in inflammatory diseases. Mol. Immunol. 56 (4), 675–682.
Nobili, A., Krashia, P., Cordella, A., et al., 2018. Ambra1 shapes hippocampal inhibition/excitation balance: role in neurodevelopmental disorders. Mol. Neurobiol. 55 (10), 7921–7940.
Nyegaard, M., Demontis, D., Foldager, L., et al., 2010. CACNA1C (rs1006737) is associated with schizophrenia. Mol. Psychiatr. 15 (2), 119–121.
ODonovan, M.C., Craddock, N., Norton, N., et al., 2008. Identification of loci associated with schizophrenia by genome-wide association and follow-up. Nat. Genet. 40 (9), 1053–1055.
ODonovan, M.C., Norton, N., Williams, H., et al., 2009. Analysis of 10 independent samples provides evidence for association between schizophrenia and a SNP flanking

fibroblast growth factor receptor 2. Mol. Psychiatr. 14 (1), 30–36.
Oh, S., Lee, J., Kwon, M.S., Weir, B., Ha, K., Park, T., 2012. A novel method to identify high order gene-gene interactions in genome-wide association studies: gene-based MDR. BMC Bioinf. 13 (Suppl. 9), S5.
Ohgake, S., Shimizu, E., Hashimoto, K., et al., 2009. Dopaminergic hypofunctions and prepulse inhibition deficits in mice lacking midkine. Progress Neuro-psychopharmacol. Biologic. Psychiatry 33 (3), 541–546.
Okuda, H., Kiuchi, H., Takao, T., et al., 2015. A novel transcriptional factor Nkapl is a germ cell-specific suppressor of Notch signaling and is indispensable for spermatogenesis. PLoS One 10 (4), e0124293.
van Os, J., Rutten, B.P., Poulton, R., 2008. Gene-environment interactions in schizophrenia: review of epidemiological findings and future directions. Schizophr. Bull. 34 (6), 1066–1082.
Ota, M., Hori, H., Sato, N., et al., 2016. Effects of ankyrin 3 gene risk variants on brain structures in patients with bipolar disorder and healthy subjects. Psychiatr. Clin. Neurosci. 70 (11), 498–506.
Pagnamenta, A.T., Bacchelli, E., de Jonge, M.V., et al., 2010. Characterization of a family with rare deletions in CNTNAP5 and DOCK4 suggests novel risk loci for autism and dyslexia. Biol. Psychiatry 68 (4), 320–328.
Pan, W., Zadina, J.E., Harlan, R.E., Weber, J.T., Banks, W.A., Kastin, A.J., 1997. Tumor necrosis factor-alpha: a neuromodulator in the CNS. Neurosci. Biobehav. Rev. 21 (5), 603–613.
Panaccione, I., Napoletano, F., Forte, A.M., et al., 2013. Neurodevelopment in schizophrenia: the role of the wnt pathways. Curr. Neuropharmacol. 11 (5), 535–558.
Pantazopoulos, H., Woo, T.U., Lim, M.P., Lange, N., Berretta, S., 2010. Extracellular matrix-glial abnormalities in the amygdala and entorhinal cortex of subjects diagnosed with schizophrenia. Arch. Gen. Psychiatr. 67 (2), 155–166.
Park, N., Juo, S.H., Cheng, R., et al., 2004. Linkage analysis of psychosis in bipolar pedigrees suggests novel putative loci for bipolar disorder and shared susceptibility with schizophrenia. Mol. Psychiatr. 9 (12), 1091–1099.
Pasternak, O., Kubicki, M., Shenton, M.E., 2016. In vivo imaging of neuroinflammation in schizophrenia. Schizophr. Res. 173 (3), 200–212.
Peet, M., Ramchand, C.N., Lee, J., et al., 1998. Association of the Ban I dimorphic site at the human cytosolic phospholipase A2 gene with schizophrenia. Psychiatr. Genet. 8 (3), 191–192.
Perrier, E., Pompei, F., Ruberto, G., Vassos, E., Collier, D., Frangou, S., 2011. Initial evidence for the role of CACNA1C on subcortical brain morphology in patients with bipolar disorder. Eur. Psychiatry 26 (3), 135–137.
Perrone-Capano, C., Di Porzio, U., 2000. Genetic and epigenetic control of midbrain dopaminergic neuron development. Int. J. Dev. Biol. 44 (6), 679–687.
Pers, T.H., Karjalainen, J.M., Chan, Y., et al., 2015. Biological interpretation of genome-wide association studies using predicted gene functions. Nat. Commun. 6, 5890.
Petek, E., Windpassinger, C., Vincent, J.B., et al., 2001. Disruption of a novel gene (IMMP2L) by a breakpoint in 7q31 associated with Tourette syndrome. Am. J. Hum. Genet. 68 (4), 848–858.
Pisante, A., Bronstein, M., Yakir, B., Darvasi, A., 2009. A variant in the reelin gene increases the risk of schizophrenia and schizoaffective disorder but not bipolar disorder. Psychiatr. Genet. 19 (4), 212.
Ponomarev, E.D., Shriver, L.P., Maresz, K., Pedras-Vasconcelos, J., Verthelyi, D., Dittel, B.N., 2007. GM-CSF production by autoreactive T cells is required for the activation of microglial cells and the onset of experimental autoimmune encephalomyelitis. J. Immunol. 178 (1), 39–48.
Porteous, D.J., Thomson, P., Brandon, N.J., Millar, J.K., 2006. The genetics and biology of DISC1–an emerging role in psychosis and cognition. Biol. Psychiatry 60 (2), 123–131.
Prichard, L., Deloulme, J.C., Storm, D.R., 1999. Interactions between neurogranin and calmodulin in vivo. J. Biol. Chem. 274 (12), 7689–7694.
Prontera, P., Bernardini, L., Stangoni, G., et al., 2011. Deletion 2p15-16.1 syndrome: case report and review. Am. J. Med. Genet. 155A (10), 2473–2478.
Psychiatric GWAS Consortium Bipolar Disorder Working Group, 2011. Large-scale genome-wide association analysis of bipolar disorder identifies a new susceptibility locus near ODZ4. Nat. Genet. 43 (10), 977–983.
Quednow, B.B., Ettinger, U., Mossner, R., et al., 2011. The schizophrenia risk allele C of the TCF4 rs9960767 polymorphism disrupts sensorimotor gating in schizophrenia spectrum and healthy volunteers. J. Neurosci. 31 (18), 6684–6691.
Rajcan-Separovic, E., Harvard, C., Liu, X., et al., 2007. Clinical and molecular cytogenetic characterisation of a newly recognised microdeletion syndrome involving 2p15-16.1. J. Med. Genet. 44 (4), 269–276.
Rauch, U., Feng, K., Zhou, X.H., 2001. Neurocan: a brain chondroitin sulfate proteoglycan. Cell. Mol. Life Sci. 58 (12–13), 1842–1856.
Reierson, G.W., Guo, S., Mastronardi, C., Licinio, J., Wong, M.L., 2011. cGMP signaling, phosphodiesterases and major depressive disorder. Curr. Neuropharmacol. 9 (4), 715–727.
Ridwan, S., Bauer, H., Frauenknecht, K., von Pein, H., Sommer, C.J., 2012. Distribution of granulocyte-monocyte colony-stimulating factor and its receptor alpha-subunit in the adult human brain with specific reference to Alzheimer's disease. J. Neural Transm. 119 (11), 1389–1406.
Rietschel, M., Mattheisen, M., Degenhardt, F., et al., 2012. Association between genetic variation in a region on chromosome 11 and schizophrenia in large samples from Europe. Mol. Psychiatr. 17 (9), 906–917.
Rietveld, C.A., Medland, S.E., Derringer, J., et al., 2013. GWAS of 126,559 individuals identifies genetic variants associated with educational attainment. Science (New York, NY) 340 (6139), 1467–1471.
Rikiyama, T., Curtis, J., Oikawa, M., et al., 2003. GCF2: expression and molecular analysis of repression. Biochim. Biophys. Acta 1629 (1–3), 15–25.
Riley, B., Kuo, P.H., Maher, B.S., et al., 2009. The dystrobrevin binding protein 1 (DTNBP1) gene is associated with schizophrenia in the Irish Case Control Study of Schizophrenia (ICCSS) sample. Schizophr. Res. 115 (2–3), 245–253.
Ripke, S., O'Dushlaine, C., Chambert, K., et al., 2013. Genome-wide association analysis identifies 13 new risk loci for schizophrenia. Nat. Genet. 45 (10), 1150–1159.
Rivero, O., Sich, S., Popp, S., Schmitt, A., Franke, B., Lesch, K.P., 2013. Impact of the ADHD-susceptibility gene CDH13 on development and function of brain networks. Eur. Neuropsychopharmacol. 23 (6), 492–507.
Roda, A., Chendo, I., Kunz, M., 2015. Biomarkers and staging of bipolar disorder: a systematic review. Trends Psychiatry Psychother 37 (1), 3–11.
Rodriguez-Santiago, B., Brunet, A., Sobrino, B., et al., 2010. Association of common copy number variants at the glutathione S-transferase genes and rare novel genomic changes with schizophrenia. Mol. Psychiatr. 15 (10), 1023–1033.
Rolland, T., Tasan, M., Charloteaux, B., et al., 2014. A proteome-scale map of the human interactome network. Cell 159 (5), 1212–1226.
Rolstad, S., Palsson, E., Ekman, C.J., Eriksson, E., Sellgren, C., Landen, M., 2015. Polymorphisms of dopamine pathway genes NRG1 and LMX1A are associated with cognitive performance in bipolar disorder. Bipolar Disord. 17 (8), 859–868.
Roth, T.L., Lubin, F.D., Sodhi, M., Kleinman, J.E., 2009. Epigenetic mechanisms in schizophrenia. Biochim. Biophys. Acta 1790 (9), 869–877.
Roussos, P., Bitsios, P., Giakoumaki, S.G., et al., 2013. CACNA1C as a risk factor for schizotypal personality disorder and schizotypy in healthy individuals. Psychiatr. Res. 206 (1), 122–123.
Rudic, R.D., McNamara, P., Curtis, A.M., et al., 2004. BMAL1 and CLOCK, two essential components of the circadian clock, are involved in glucose homeostasis. PLoS Biol. 2 (11), e377.
Rujescu, D., Ingason, A., Cichon, S., et al., 2009. Disruption of the neurexin 1 gene is associated with schizophrenia. Hum. Mol. Genet. 18 (5), 988–996.
Rybakowski, J.K., Dmitrzak-Weglar, M., Kliwicki, S., Hauser, J., 2014. Polymorphism of circadian clock genes and prophylactic lithium response. Bipolar Disord. 16 (2), 151–158.
Salatino-Oliveira, A., Genro, J.P., Polanczyk, G., et al., 2015. Cadherin-13 gene is associated with hyperactive/impulsive symptoms in attention/deficit hyperactivity disorder. *American journal of medical genetics Part B, Neuropsychiatric genetics*. 168B. the official publication of the International Society of Psychiatric Genetics, pp. 162–169 (3).
Sanchez-Tillo, E., de Barrios, O., Valls, E., Darling, D.S., Castells, A., Postigo, A., 2015. ZEB1 and TCF4 reciprocally modulate their transcriptional activities to regulate Wnt target gene expression. Oncogene 34 (46), 5760–5770.
Sansam, C.L., Shepard, J.L., Lai, K., et al., 2006. DTL/CDT2 is essential for both CDT1 regulation and the early G2/M checkpoint. Genes Dev. 20 (22), 3117–3129.
Sasayama, D., Hiraishi, A., Tatsumi, M., et al., 2013. Possible association of CUX1 gene polymorphisms with antidepressant response in major depressive disorder. Pharmacogenomics J. 13 (4), 354–358.
Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014. Biological insights from 108 schizophrenia-associated genetic loci. Nature 511 (7510), 421–427.
Scholz, M., Blaheta, R.A., Vogel, J., Doerr, H.W., Cinatl Jr., J., 1999. Cytomegalovirus-induced transendothelial cell migration. a closer look at intercellular communication mechanisms. Intervirology 42 (5–6), 350–356.
Schulte, E.C., Stahl, I., Czamara, D., et al., 2013. Rare variants in PLXNA4 and Parkinson's disease. PLoS One 8 (11), e79145.
Schultz, C.C., Muhleisen, T.W., Nenadic, I., et al., 2014. Common variation in NCAN, a risk factor for bipolar disorder and schizophrenia, influences local cortical folding in schizophrenia. Psychol. Med. 44 (4), 811–820.
Seet, L.F., Hong, W., 2006. The Phox (PX) domain proteins and membrane traffic. Biochim. Biophys. Acta 1761 (8), 878–896.
Seripa, D., Matera, M.G., Franceschi, M., et al., 2008. The RELN locus in Alzheimer's disease. J. Alzheimer's Dis. 14 (3), 335–344.
Shatz, C.J., 2009. MHC class I: an unexpected role in neuronal plasticity. Neuron 64 (1), 40–45.
Shen, J.X., Wachten, S., Halls, M.L., Everett, K.L., Cooper, D.M., 2012. Muscarinic receptors stimulate AC2 by novel phosphorylation sites, whereas Gbetagamma subunits exert opposing effects depending on the G-protein source. Biochem. J. 447 (3), 393–405.
Shen, Y., Xun, G., Guo, H., et al., 2016. Association and gene-gene interactions study of reelin signaling pathway related genes with autism in the Han Chinese population. Autism Res. 9 (4), 436–442.
Shi, J., Levinson, D.F., Duan, J., et al., 2009. Common variants on chromosome 6p22.1 are associated with schizophrenia. Nature 460 (7256), 753–757.
Shi, Y., Li, Z., Xu, Q., et al., 2011. Common variants on 8p12 and 1q24.2 confer risk of schizophrenia. Nat. Genet. 43 (12), 1224–1227.
Shibata, H., Yamamoto, K., Sun, Z., et al., 2013. Genome-wide association study of schizophrenia using microsatellite markers in the Japanese population. Psychiatric genetics.
Shifman, S., Johannesson, M., Bronstein, M., et al., 2008. Genome-wide association identifies a common variant in the reelin gene that increases the risk of schizophrenia only in women. PLoS Genet. 4 (2), e28.
Shiina, T., Hosomichi, K., Inoko, H., Kulski, J.K., 2009. The HLA genomic loci map: expression, interaction, diversity and disease. J. Hum. Genet. 54 (1), 15–39.
Shinnick-Gallagher, P., McKernan, M.G., Xie, J., Zinebi, F., 2003. L-type voltage-gated calcium channels are involved in the in vivo and in vitro expression of fear conditioning. Ann. N. Y. Acad. Sci. 985, 135–149.
Shirasawa, S., Harada, H., Furugaki, K., et al., 2004. SNPs in the promoter of a B cell-specific antisense transcript, SAS-ZFAT, determine susceptibility to autoimmune thyroid disease. Hum. Mol. Genet. 13 (19), 2221–2231.
Shiu, T.Y., Huang, T.Y., Huang, S.M., et al., 2013. Nuclear factor kappaB down-regulates human UDP-glucuronosyltransferase 1A1: a novel mechanism involved in

inflammation-associated hyperbilirubinaemia. Biochem. J. 449 (3), 761–770.
Sinnegger-Brauns, M.J., Huber, I.G., Koschak, A., et al., 2009. Expression and 1,4-dihydropyridine-binding properties of brain L-type calcium channel isoforms. Mol. Pharmacol. 75 (2), 407–414.
SJ, L., ODM, C., G, H., et al., 2008. Rare chromosomal deletions and duplications increase risk of schizophrenia. Nature 455 (7210), 237–241.
Sklar, P., Smoller, J.W., Fan, J., et al., 2008. Whole-genome association study of bipolar disorder. Mol. Psychiatr. 13 (6), 558–569.
Sleiman, P., Wang, D., Glessner, J., et al., 2013. GWAS meta analysis identifies TSNARE1 as a novel Schizophrenia/Bipolar susceptibility locus. Sci. Rep. 3, 3075.
Smith, E.N., Koller, D.L., Panganiban, C., et al., 2011. Genome-wide association of bipolar disorder suggests an enrichment of replicable associations in regions near genes. PLoS Genet. 7 (6), e1002134.
Smrt, R.D., Szulwach, K.E., Pfeiffer, R.L., et al., 2010. MicroRNA miR-137 regulates neuronal maturation by targeting ubiquitin ligase mind bomb-1. Stem Cell. 28 (6), 1060–1070.
Soeiro-de-Souza, M.G., Otaduy, M.C., Dias, C.Z., Bio, D.S., Machado-Vieira, R., Moreno, R.A., 2012. The impact of the CACNA1C risk allele on limbic structures and facial emotions recognition in bipolar disorder subjects and healthy controls. J. Affect. Disord. 141 (1), 94–101.
Soeiro-de-Souza, M.G., Bio, D.S., Dias, V.V., Vieta, E., Machado-Vieira, R., Moreno, R.A., 2013. The CACNA1C risk allele selectively impacts on executive function in bipolar type I disorder. Acta Psychiatr. Scand. 128 (5), 362–369.
Sohn, H., Kim, B., Kim, K.H., Kim, M.K., Choi, T.K., Lee, S.H., 2014. Effects of VRK2 (rs2312147) on white matter connectivity in patients with schizophrenia. PLoS One 9 (7), e103519.
Sousa, I., Clark, T.G., Holt, R., et al., 2010. Polymorphisms in leucine-rich repeat genes are associated with autism spectrum disorder susceptibility in populations of European ancestry. Mol. Autism. 1 (1), 7.
Spencer, C.C., Su, Z., Donnelly, P., Marchini, J., 2009. Designing genome-wide association studies: sample size, power, imputation, and the choice of genotyping chip. PLoS Genet. 5 (5), e1000477.
Stefansson, H., Ophoff, R.A., Steinberg, S., et al., 2009. Common variants conferring risk of schizophrenia. Nature 460 (7256), 744–747.
Stefansson, H., Meyer-Lindenberg, A., Steinberg, S., et al., 2014. CNVs conferring risk of autism or schizophrenia affect cognition in controls. Nature 505 (7483), 361–366.
Steinberg, R., Shemer-Avni, Y., Adler, N., Neuman-Silberberg, S., 2008. Human cytomegalovirus immediate-early-gene expression disrupts embryogenesis in transgenic Drosophila. Transgenic Res. 17 (1), 105–119.
Steinberg, S., de Jong, S., Andreassen, O.A., et al., 2011. Common variants at VRK2 and TCF4 conferring risk of schizophrenia. Hum. Mol. Genet. 20 (20), 4076–4081.
Steinberg, S., de Jong, S., Mattheisen, M., et al., 2012. Common variant at 16p11.2 conferring risk of psychosis. Mol. Psychiatr. 19 (1), 108–114.
Stellwagen, D., Malenka, R.C., 2006. Synaptic scaling mediated by glial TNF-alpha. Nature 440 (7087), 1054–1059.
Strous, R.D., Shoenfeld, Y., 2006. Schizophrenia, autoimmunity and immune system dysregulation: a comprehensive model updated and revisited. J. Autoimmun. 27 (2), 71–80.
Suda, S., Iwata, K., Shimmura, C., et al., 2011. Decreased expression of axon-guidance receptors in the anterior cingulate cortex in autism. Mol. Autism. 2 (1), 14.
Sullivan, P.F., Kendler, K.S., Neale, M.C., 2003. Schizophrenia as a complex trait: evidence from a meta-analysis of twin studies. Arch. Gen. Psychiatr. 60 (12), 1187–1192.
Sullivan, P.F., Lin, D., Tzeng, J.Y., et al., 2008. Genomewide association for schizophrenia in the CATIE study: results of stage 1. Mol. Psychiatr. 13 (6), 570–584.
Sun, Y., Zhao, L.Y., Wang, G.B., et al., 2016. ZNF804A variants confer risk for heroin addiction and affect decision making and gray matter volume in heroin abusers. Addict. Biol. 21 (3), 657–666.
Suriano, A.R., Sanford, A.N., Kim, N., et al., 2005. GCF2/LRRFIP1 represses tumor necrosis factor alpha expression. Mol. Cell Biol. 25 (20), 9073–9081.
Szperl, A.M., Golachowska, M.R., Bruinenberg, M., et al., 2011. Functional characterization of mutations in the myosin Vb gene associated with microvillus inclusion disease. J. Pediatr. Gastroenterol. Nutr. 52 (3), 307–313.
Szulwach, K.E., Li, X., Smrt, R.D., et al., 2010. Cross talk between microRNA and epigenetic regulation in adult neurogenesis. J. Cell Biol. 189 (1), 127–141.
Takeuchi, T., Kojima, M., Nakajima, K., Kondo, S., 1999. Jumonji gene is essential for the neurulation and cardiac development of mouse embryos with a C3H/He background. Mech. Dev. 86 (1–2), 29–38.
Takeuchi, T., Misaki, A., Liang, S.B., et al., 2000. Expression of T-cadherin (CDH13, H-Cadherin) in human brain and its characteristics as a negative growth regulator of epidermal growth factor in neuroblastoma cells. J. Neurochem. 74 (4), 1489–1497.
Tamagnone, L., Artigiani, S., Chen, H., et al., 1999. Plexins are a large family of receptors for transmembrane, secreted, and GPI-anchored semaphorins in vertebrates. Cell 99 (1), 71–80.
Teixeira, C.M., Kron, M.M., Masachs, N., et al., 2012. Cell-autonomous inactivation of the reelin pathway impairs adult neurogenesis in the hippocampus. J. Neurosci. 32 (35), 12051–12065.
Terai, K., Abbas, T., Jazaeri, A.A., Dutta, A., 2010. CRL4(Cdt2) E3 ubiquitin ligase monoubiquitinates PCNA to promote translesion DNA synthesis. Mol. Cell. 37 (1), 143–149.
Terwisscha van Scheltinga, A.F., Bakker, S.C., Kahn, R.S., 2010. Fibroblast growth factors in schizophrenia. Schizophr. Bull. 36 (6), 1157–1166.
The Schizophrenia Psychiatric Genome-Wide Association Study (GWAS) Consortium, 2011. Genome-wide association study identifies five new schizophrenia loci. Nat. Genet. 43 (10), 969–976.
Thimm, M., Kircher, T., Kellermann, T., et al., 2011. Effects of a CACNA1C genotype on attention networks in healthy individuals. Psychol. Med. 41 (7), 1551–1561.
Thomas, D., 2010. Methods for investigating gene-environment interactions in candidate pathway and genome-wide association studies. Annu. Rev. Public Health 31, 21–36.
Tkachev, D., Mimmack, M.L., Ryan, M.M., et al., 2003. Oligodendrocyte dysfunction in schizophrenia and bipolar disorder. Lancet 362 (9386), 798–805.
Tokita, Y., Keino, H., Matsui, F., et al., 2001. Regulation of neuregulin expression in the injured rat brain and cultured astrocytes. J. Neurosci. 21 (4), 1257–1264.
Tominaga, M., Tomooka, Y., 2002. Novel genes cloned from a neuronal cell line newly established from a cerebellum of an adult p53(-/-) mouse. Biochem. Biophys. Res. Commun. 297 (3), 473–479.
Treutlein, J., Cichon, S., Ridinger, M., et al., 2009. Genome-wide association study of alcohol dependence. Arch. Gen. Psychiatr. 66 (7), 773–784.
Tsai, G., Coyle, J.T., 2002. Glutamatergic mechanisms in schizophrenia. Annu. Rev. Pharmacol. Toxicol. 42, 165–179.
Tsai, T.C., Lee, Y.L., Hsiao, W.C., Tsao, Y.P., Chen, S.L., 2005. NRIP, a novel nuclear receptor interaction protein, enhances the transcriptional activity of nuclear receptors. J. Biol. Chem. 280 (20), 20000–20009.
Twal, W.O., Czirok, A., Hegedus, B., et al., 2001. Fibulin-1 suppression of fibronectin-regulated cell adhesion and motility. J. Cell Sci. 114 (Pt 24), 4587–4598.
Ueda, S., Fujimoto, S., Hiramoto, K., Negishi, M., Katoh, H., 2008. Dock4 regulates dendritic development in hippocampal neurons. J. Neurosci. Res. 86 (14), 3052–3061.
Uhl, G.R., Drgon, T., Liu, Q.R., et al., 2008. Genome-wide association for methamphetamine dependence: convergent results from 2 samples. Arch. Gen. Psychiatr. 65 (3), 345–355.
Vacic, V., McCarthy, S., Malhotra, D., et al., 2011. Duplications of the neuropeptide receptor gene VIPR2 confer significant risk for schizophrenia. Nature 471 (7339), 499–503.
Vanoye, C.G., Welch, R.C., Daniels, M.A., et al., 2009. Distinct subdomains of the KCNQ1 S6 segment determine channel modulation by different KCNE subunits. J. Gen. Physiol. 134 (3), 207–217.
Venkatasubramanian, G., Debnath, M., 2013. The TRIPS (Toll-like receptors in immuno-inflammatory pathogenesis) Hypothesis: a novel postulate to understand schizophrenia. Progress in neuro-psychopharmacology & biological psychiatry 44, 301–311.
Videnovic, A., Lazar, A.S., Barker, R.A., Overeem, S., 2014. The clocks that time us'–circadian rhythms in neurodegenerative disorders. Nat. Rev. Neurol. 10 (12), 683–693.
Volcik, K.A., Zhu, H., Finnell, R.H., Shaw, G.M., Canfield, M., Lammer, E.J., 2004. Evaluation of the jumonji gene and risk for spina bifida and congenital heart defects. Am. J. Med. Genet. 126A (2), 215–217.
Vrijenhoek, T., Buizer-Voskamp, J.E., van der Stelt, I., et al., 2008. Recurrent CNVs disrupt three candidate genes in schizophrenia patients. Am. J. Hum. Genet. 83 (4), 504–510.
Walsh, T., McClellan, J.M., McCarthy, S.E., et al., 2008. Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. Science (New York, NY) 320 (5875), 539–543.
Walton, E., Geisler, D., Hass, J., et al., 2013. The impact of genome-wide supported schizophrenia risk variants in the neurogranin gene on brain structure and function. PLoS One 8 (10), e76815.
Wang, K., Zhang, H., Ma, D., et al., 2009. Common genetic variants on 5p14.1 associate with autism spectrum disorders. Nature 459 (7246), 528–533.
Wang, K.S., Liu, X., Zhang, Q., Aragam, N., Pan, Y., 2011a. Genome-wide association analysis of age at onset in schizophrenia in a European-American sample. Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Pub. Int. Soc. Psychiatric Genet. 156B (6), 671–680.
Wang, F., McIntosh, A.M., He, Y., Gelernter, J., Blumberg, H.P., 2011b. The association of genetic variation in CACNA1C with structure and function of a frontotemporal system. Bipolar Disord. 13 (7–8), 696–700.
Wang, Z., Yang, B., Liu, Y., et al., 2015. Further evidence supporting the association of NKAPL with schizophrenia. Neurosci. Lett. 605, 49–52.
Weber, H., Kittel-Schneider, S., Gessner, A., et al., 2011. Cross-disorder analysis of bipolar risk genes: further evidence of DGKH as a risk gene for bipolar disorder, but also unipolar depression and adult ADHD. Neuropsychopharmacology 36 (10), 2076–2085.
Wellcome Trust Case Control, C., 2007. Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. Nature 447 (7145), 661–678.
Whalley, H.C., Papmeyer, M., Romaniuk, L., et al., 2012. Effect of variation in diacylglycerol kinase eta (DGKH) gene on brain function in a cohort at familial risk of bipolar disorder. Neuropsychopharmacology 37 (4), 919–928.
Williams, H.J., Norton, N., Dwyer, S., et al., 2011a. Fine mapping of ZNF804A and genome-wide significant evidence for its involvement in schizophrenia and bipolar disorder. Mol. Psychiatr. 16 (4), 429–441.
Williams, H.J., Craddock, N., Russo, G., et al., 2011b. Most genome-wide significant susceptibility loci for schizophrenia and bipolar disorder reported to date cross-traditional diagnostic boundaries. Hum. Mol. Genet. 20 (2), 387–391.
Wirgenes, K.V., Tesli, M., Inderhaug, E., et al., 2014. ANK3 gene expression in bipolar disorder and schizophrenia. Br. J. Psychiatry 205 (3), 244–245.
de Witte, L.D., van Mierlo, H.C., Litjens, M., et al., 2015. The association between antibodies to neurotropic pathogens and schizophrenia: a case-control study. NPJ Schizophr 1, 15041.
Wolf, C., Mohr, H., Schneider-Axmann, T., et al., 2014. CACNA1C genotype explains interindividual differences in amygdala volume among patients with schizophrenia. Eur. Arch. Psychiatry Clin. Neurosci. 264 (2), 93–102.
Wong, E.H., So, H.C., Li, M., et al., 2014. Common variants on Xq28 conferring risk of schizophrenia in Han Chinese. Schizophr. Bull. 40 (4), 777–786.

Wood, T.L., Bercury, K.K., Cifelli, S.E., et al., 2013. mTOR: a link from the extracellular milieu to transcriptional regulation of oligodendrocyte development. ASN Neuro 5 (1), e00108.
Wulff, K., Dijk, D.J., Middleton, B., Foster, R.G., Joyce, E.M., 2012. Sleep and circadian rhythm disruption in schizophrenia. Br. J. Psychiatry 200 (4), 308–316.
Xiao, Y., Peng, Y., Wan, J., et al., 2013. The atypical guanine nucleotide exchange factor Dock4 regulates neurite differentiation through modulation of Rac1 GTPase and actin dynamics. J. Biol. Chem. 288 (27), 20034–20045.
Xu, Z., Croslan, D.R., Harris, A.E., Ford, G.D., Ford, B.D., 2006. Extended therapeutic window and functional recovery after intraarterial administration of neuregulin-1 after focal ischemic stroke. J. Cereb. Blood Flow Metab. 26 (4), 527–535.
Xu, B., Roos, J.L., Levy, S., van Rensburg, E.J., Gogos, J.A., Karayiorgou, M., 2008. Strong association of de novo copy number mutations with sporadic schizophrenia. Nat. Genet. 40 (7), 880–885.
Xu, B., Woodroffe, A., Rodriguez-Murillo, L., et al., 2009. Elucidating the genetic architecture of familial schizophrenia using rare copy number variant and linkage scans. Proc. Natl. Acad. Sci. U.S.A. 106 (39), 16746–16751.
Xu, W., Cohen-Woods, S., Chen, Q., et al., 2014. Genome-wide association study of bipolar disorder in Canadian and UK populations corroborates disease loci including SYNE1 and CSMD1. BMC Med. Genet. 15, 2.
Xue, Y., Canman, J.C., Lee, C.S., et al., 2000. The human SWI/SNF-B chromatin-remodeling complex is related to yeast rsc and localizes at kinetochores of mitotic chromosomes. Proc. Natl. Acad. Sci. U.S.A. 97 (24), 13015–13020.
Yajnik, V., Paulding, C., Sordella, R., et al., 2003. DOCK4, a GTPase activator, is disrupted during tumorigenesis. Cell 112 (5), 673–684.
Yamada, K., Santo-Yamada, Y., Wada, K., 2002. Restraint stress impaired maternal behavior in female mice lacking the neuromedin B receptor (NMB-R) gene. Neurosci. Lett. 330 (2), 163–166.
Yamada, K., Iwayama, Y., Hattori, E., et al., 2011. Genome-wide association study of schizophrenia in Japanese population. PLoS One 6 (6), e20468.
Yang, J., Korley, F.K., Dai, M., Everett, A.D., 2015. Serum neurogranin measurement as a biomarker of acute traumatic brain injury. Clin. Biochem. 48 (13–14), 843–848.
Yang J, Lee SH, Goddard ME, Visscher PM. GCTA: a tool for genome-wide complex trait analysis. Am. J. Hum. Genet..88(1):76-82.
Yao, H., Ye, J., 2008. Long chain acyl-CoA synthetase 3-mediated phosphatidylcholine synthesis is required for assembly of very low density lipoproteins in human hepatoma Huh7 cells. J. Biol. Chem. 283 (2), 849–854.
Yao, Y., Schroder, J., Nellaker, C., et al., 2008. Elevated levels of human endogenous retrovirus-W transcripts in blood cells from patients with first episode schizophrenia. Genes Brain Behav. 7 (1), 103–112.
Yap, A.S., Brieher, W.M., Gumbiner, B.M., 1997. Molecular and functional analysis of cadherin-based adherens junctions. Annu. Rev. Cell Dev. Biol. 13, 119–146.
Yolken, R.H., Karlsson, H., Yee, F., Johnston-Wilson, N.L., Torrey, E.F., 2000. Endogenous retroviruses and schizophrenia. Brain Res Brain Res Rev 31 (2–3), 193–199.
Yoshida, Y., Tsunoda, T., Takashima, Y., et al., 2010. ZFAT is essential for endothelial cell assembly and the branch point formation of capillary-like structures in an angiogenesis model. Cell. Mol. Biol. Lett. 15 (4), 541–550.
Yoshimi, A., Suda, A., Hayano, F., et al., 2016. Effects of NRG1 genotypes on orbitofrontal sulcogyral patterns in Japanese patients diagnosed with schizophrenia. Psychiatr. Clin. Neurosci. 70 (7), 261–268.
Yosifova, A., Mushiroda, T., Kubo, M., et al., 2011. Genome-wide association study on bipolar disorder in the Bulgarian population. Genes Brain Behav. 10 (7), 789–797.
Yue, W.H., Wang, H.F., Sun, L.D., et al., 2011. Genome-wide association study identifies a susceptibility locus for schizophrenia in Han Chinese at 11p11.2. Nat. Genet. 43 (12), 1228–1231.
Yueh, M.F., Chen, S., Nguyen, N., Tukey, R.H., 2014. Developmental onset of bilirubin-induced neurotoxicity involves Toll-like receptor 2-dependent signaling in humanized UDP-glucuronosyltransferase1 mice. J. Biol. Chem. 289 (8), 4699–4709.
Yui, K., Imataka, G., Nakamura, H., Ohara, N., Naito, Y., 2015. Eicosanoids derived from arachidonic acid and their family prostaglandins and cyclooxygenase in psychiatric disorders. Curr. Neuropharmacol. 13 (6), 776–785.
Zhang, Y., Griffin, K., Mondal, N., Parvin, J.D., 2004. Phosphorylation of histone H2A inhibits transcription on chromatin templates. J. Biol. Chem. 279 (21), 21866–21872.
Zhang, F., Sha, J., Wood, T.G., et al., 2008. Alteration in the activation state of new inflammation-associated targets by phospholipase A2-activating protein (PLAA). Cell. Signal. 20 (5), 844–861.
Zhang, X., Lei, K., Yuan, X., et al., 2009a. SUN1/2 and Syne/Nesprin-1/2 complexes connect centrosome to the nucleus during neurogenesis and neuronal migration in mice. Neuron 64 (2), 173–187.
Zhang, F., Ma, J., Wu, J., et al., 2009b. PALB2 links BRCA1 and BRCA2 in the DNA-damage response. Curr. Biol. 19 (6), 524–529.
Zhang, J., Felder, A., Liu, Y., et al., 2010. Nesprin 1 is critical for nuclear positioning and anchorage. Hum. Mol. Genet. 19 (2), 329–341.
Zhang, L., Yang, H., Zhao, H., Zhao, C., 2011a. Calcium-related signaling pathways contributed to dopamine-induced cortical neuron apoptosis. Neurochem. Int. 58 (3), 281–294.
Zhang, R., Lu, S.M., Qiu, C., et al., 2011b. Population-based and family-based association studies of ZNF804A locus and schizophrenia. Mol. Psychiatr. 16 (4), 360–361.
Zhang, Q., Shen, Q., Xu, Z., et al., 2012. The effects of CACNA1C gene polymorphism on spatial working memory in both healthy controls and patients with schizophrenia or bipolar disorder. Neuropsychopharmacology 37 (3), 677–684.
Zhang, C., Cai, J., Zhang, J., et al., 2014. Genetic modulation of working memory deficits by ankyrin 3 gene in schizophrenia. Progress in neuro-psychopharmacology & biological psychiatry 50, 110–115.
Zhang, B., Gao, C.Y., Zhang, H.B., et al., 2015a. Association of the VRK2 gene rs3732136 polymorphism with schizophrenia in a Northwest Chinese Han population. Genet. Mol. Res. 14 (3), 9404–9411.
Zhang, B., Li, D.X., Lu, N., Fan, Q.R., Li, W.H., Feng, Z.F., 2015b. Lack of association between the TSPAN18 gene and schizophrenia based on new data from han Chinese and a meta-analysis. Int. J. Mol. Sci. 16 (6), 11864–11872.
Zhang, C., Lu, W., Wang, Z., et al., 2016a. A comprehensive analysis of NDST3 for schizophrenia and bipolar disorder in Han Chinese. Transl. Psychiatry 6, e701.
Zhang, Z., Chen, X., Yu, P., et al., 2016b. Effect of rs1344706 in the ZNF804A gene on the connectivity between the hippocampal formation and posterior cingulate cortex. Schizophr. Res. 170 (1), 48–54.
Zhu, W.Y., Jiang, P., He, X., et al., 2016. Contribution of NRG1 gene polymorphisms in temporal lobe epilepsy. J. Child Neurol. 31 (3), 271–276.