Supplementary Methods

Subject Recruitment. Participants completed the Structured Clinical Interview for Diagnostic (SCID patient or nonpatient versions) to yield Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) (1) diagnoses. Probands met criteria for schizophrenia (SZ), schizoaffective (SZA) or psychotic bipolar disorder (PBP) as defined in (2)). Healthy comparison individuals (HC) were free of any lifetime psychosis and Axis 1 disorders according to the DSM IV. Participants were recruited at the five BSNIP sites via advertisements (see Table S1), online postings, and referral by word of mouth and local National Association on Mental Illness (NAMI) chapters. All subjects were assessed by experienced clinical raters including masters-level clinicians, doctoral-level clinical psychologists or psychiatrists to document diagnostic information using clinical data and structured interviews. Probands were administered the Positive and Negative Symptom Scale (PANSS (3)), the Young Mania Rating Scale (4), the Montgomery Asberg Depression Rating Scale (MADRS (5)), and the Global Assessment of Functioning scale (GAF; axis V of DSM-IV). Exclusion criteria included central neurological illness, mental retardation, current substance abuse based on toxicology and urine screening, past abuse within 6 months or dependence within 2 years or any prior extensive history of drug dependence (DSM-IV). Probands were on stable doses of psychotropic medication (Table S2) for $\geq$ 4 weeks.

Experimental Design for Auditory Oddball Paradigm. The auditory oddball task consisted of two different pitches; high (1500 Hz db) target and low standard (1000 Hz) tones. Stimulus duration was 50 ms with rise and decay of 10 ms. The interstimulus interval was 1300 ms, with stimuli comprising of 567 standard (85%) and 100 target tones (15%) presented in pseudorandom order. Participants were seated in a quiet electrically shielded booth (ambient sound = 61–63 dB; luminance = .11–.12 foot-candles) fixating at a computer monitor with cross hair, while listening to the tones delivered by headphone. Subjects were instructed to press a button with their right index finger when a target tone was detected. Uniformity in recording conditions was preserved across sites, which also used identical recording equipment and stimulus presentations.

Auditory Oddball Electroencephalogram (EEG) Data Acquisition. EEG was collected continuously from Neuroscan equipment equipped with 64 Ag/AgCl electrodes (Quik-Cap, Compumedrics, El Paso, Texas). Electrode impedances were maintained < 5 KΩ throughout the experiment. Electrodes were placed according to the International 10-10 EEG system (Figure S1). Eye movement recordings were collected by placing one electrode at the inner and outer canthi of the left and right eye. Nose served as a reference with a forehead ground. EEG recordings were amplified with a gain of 12,500 and digitized at a sampling rate of 1000 Hz using Neuroscan ACQUIRE and SynAmps 2 recording systems (Compumedics Neuroscan).

Structural Magnetic Resonance Imaging (sMRI) Data. We extracted 68 cortical and 14 subcortical brain volumes using sMRI data in 369 subjects (89 HC, 114 SZ and 166 PBP out of 449 subjects) (6) using FreeSurfer (7) version 5.347. (http://surfer.nmr.mgh.harvard.edu/fswiki/FreeSurferMethodsCitation). High-resolution isotropic T1-weighted MPRAGE sequences (TR=6.7 msec, TE= 3.1 msec, 8o flip angle, 256x240 matrix

size, total scan duration=10:52.6 minutes, 170 sagittal slices, 1mm slice thickness, 1x1x1.2 mm3 voxel resolution) were obtained following the Alzheimer's Disease Neuroimaging Initiative (http://www.loni.ucla.edu/ADNI) protocol.

Event-Related Potential (ERP) Data Processing. Raw EEG data were checked and adjusted for bad electrodes (< 5% for any subject) using spherical spline interpolation (BESA 5.3; MEGIS Software, Grafelfing, Germany). Data were subjected to blink and cardiac artifact removal by using independent component analysis (8). Data were segmented into epochs from 250 msec before to 720 msec after stimulus onset and digitally filtered between 0.5 Hz-55 Hz (zero-phase filter; roll-off: 6 and 48 dB/octave, respectively). Epochs were baseline corrected using mean voltage activity during 250 ms prestimulus period and excluded from analysis if voltage > 75 μV at any electrode; at least 60% of trials were accepted for all subjects.

Spatial Principal Component Analysis. EEG data from the 64 channels were effectively compressed and summarized using spatial principal components analysis (PCA) on grand average data (9, 10) implemented using BESA (MEGIS Software) and Matlab (The Mathworks, Natick, Massachusetts) to identify spatial patterns in the EEG topography (Refer to article (9) for details). Each electrode was averaged for both target and standard conditions and PCA with promax oblique vector rotation and Kaiser (11) normalization was applied on a 64 x 64 sensor covariance matrix (98 time points as observations for 64 sensors) for each condition separately. Based on eigenvalues, two target components (accounting for 65.7% and 29.6% of the variance) and one standard component (86.8% of the variance) were identified. For the target and standard condition, component weights were multiplied by each subject's grand average data, summed across sensors, and normalized by the sum of the component weights, yielding one waveform per component for each subject. The two target components comprised a late parietal (P3b) and early

frontal topography respectively and the standard component had an early frontal topography (Figure 1 in the text). The reduced component waveforms were concatenated (standard waveform followed by parietal target and frontal target) to yield a single phenotype vector for each subject for the multivariate genetic association analysis.

Single Nucleotide Polymorphism (SNP) Data Processing. Genotyped SNP data typically represented as homozygous (AA, BB) and heterozygous (AB or BA) were converted to numerical format by additive coding for the number of minor alleles (AA = 0, AB=1 and BB=2, assuming B is minor allele). Figure S2 illustrates the processing pipeline (12) for SNP data quality control using Plink (13). Raw SNP data were inspected for poorly genotyped subjects by checking for discordant sex, elevated missing rate (> 3%), unusual heterozygosity (> 3 SD from mean) and unusual similarity or relatedness between individuals. Individuals failing quality control were removed. Further, individual markers were removed, if marker missing rate was > 5%, rare variants with minor allele frequency < 0.05 and SNP correlated above 0.8 in block sizes of 200. Additional exclusion criteria included failure of Hardy Weinberg equilibrium ($p<0.00001$) and significantly differing genotype call rates between cases (SZ and PBP probands) and healthy comparison subjects (HC) ($p<0.00001$). Missing SNP data were imputed using the most frequent genotype. Finally, SNP data were corrected for population stratification bias by extracting the components contributing to the ethnicity structure using PCA (see q-q plot in Figure S3). In the current sample, the top three PCA components were significantly associated ($p=2.3x10^{-273}$, $p=7.03x10^{-91}$ and $p=2.66x10^{-93}$) with self-reported ethnicity, but no significant case-control difference was detected. A total of 575,687 SNP were retained from the quality control process for the initial univariate analyses that further reduced the marker size for multivariate association.

Para-ICA like any other multivariate approach is limited by the high dimensionality posed by the numerous variables in the model that degrade the cumulative signal level from the linear combination of SNPs due to the disproportion between the SNPs to the observation cases. To overcome this problem, we applied univariate analysis using logistic regression to each of the 575,687 markers between cases and HC, similar to the strategy employed in a recent study (14). The regression was conducted separately for SZ and PBP probands vs HC. SNPs that passed a nominal $p<0.05$ uncorrected threshold from either of the two univariate analyses were combined and then queried using online databases dbsnp (http://www.ncbi.nlm.nih.gov/SNP/) and genome variation server (http://gvs.gs.washington.edu/GVS137/) to determine the functional annotation for each marker. Finally, 20,329 SNPs from coding and non-coding regions of the gene with nominal $p<0.05$ uncorrected significance level from the regression analyses were selected for the parallel independent component analysis (Para-ICA) based association in conjunction with ERP data.

Para-ICA based SNP-ERP Association. The genetic SNP and ERP data were jointly analyzed using the Para-ICA method (15) implemented in the fusion ICA toolbox V2.0c (https://icatb.sourceforge.net) developed in Matlab. Para-ICA estimates independent components on the genetic and the ERP data and jointly evaluates the association between the two data modalities (Figure S4). This approach is an extension of the common ICA technique to two data modalities or features, by including an additional constraint of maximizing the correlation between the two modalities, while simultaneously maximizing the independence on each feature using information theoretic based entropy cost function. Para-ICA is a statistically efficient data-driven multivariate technique that linearly combines multiple gene variants yielding a cumulative entity that is linked simultaneously to linearly related complex phenotypes. Unlike univariate

genome wide association tests, the complex phenotype can include multiple biological measures. Para-ICA parses the underlying structure or pattern in both the genetic and phenotype data mixture and relates them. Para-ICA offers various advantages: 1) data-driven analysis without prior assumptions on the underlying data distribution. 2) separation of unstructured complex noise sources from true biological signals producing better signal-to-noise ratio 3) efficiency in handling large scale data especially when fusing multiple data domains by data reduction, yielding statistical predictive efficacy by accounting for multiple comparison on reduced factors.

The ERP data matrix (N=449 X 294 (3x98 time points)) was formed by concatenating the PCA-compressed components including one standard and 2 target waveforms for each subject. The number of components for the pooled concatenated ERP data (data from SZ, PBP and HCs) was estimated to be 8 based on the minimum descriptor length criteria (16), a common method used in prior ICA based studies (17). The SNP data was organized as a matrix of subjects by SNPs (N=449 X 20329). For the pooled SNP data, the number of independent components was selected as 11 based on the consistency tool that checked for the maximum reliability of the components (18). A leave-one-out cross validation was conducted using the same parameters used in the original run to assess the reliability of SNP and ERP components. The ERP and SNP component in the each significantly associated pair from the original run was correlated with components from each run of the leave-one-out analysis (n=449). The component in each modality in each run that best matched the original pair was identified based on correlation. The average within modality correlation from different runs for each significantly associated pair was used as the final reliability index.

Pathway Analysis. The enrichment analysis determined the hierarchical processes including biological and metabolic attributes underlying the genes with reference to predefined pathway

maps and gene ontologies (GO) repository available in GeneGo. Statistical significance levels quantifying the enrichment were estimated using hypergeometric distribution based on the likelihood that significant genes were over-regulated in specific pathways and processes. Significance values were adjusted for multiple comparisons using false discovery rate correction.

TABLE S1. Demographic Information for Schizophrenia and Psychotic Bipolar Probands and Healthy Comparison Subjects

Variable	Healthy comparison subjects (N=95)		Schizophrenia (N=144)		Psychotic bipolar disorder (N=210)		p
	Mean	SD	Mean	SD	Mean	SD	
Age (years) [a]	36.01	11.79	32.88	11.73	35.29	12.21	< 0.049
PANSS-positive	-	-	16.15	5.35	14.14	5.45	
PANSS-negative	-	-	16.7	5.76	13.15	4.85	
PANSS-general	-	-	32.37	9.02	30.25	9.04	
SBS	-	-	7.68	1.33	1.99	1.77	
WRAT-4[b,c]	103.87	12.83	96.37	15.84	101.21	14.05	< 0.0001
CPZ equivalent[d]	-	-	614.12	502.89	417.53	389.42	
	N	%	N	%	N	%	
Sex[e]							
Male	39	41	104	72	84	40	< 2.23e-9
Female	56	59	40	28	126	60	
Site							
Baltimore	14	14.7	34	23.6	43	20.4	< 0.11
Boston[f]	0	0	3	2	0	0	
Chicago	26	27.4	31	21.5	67	31.9	
Dallas[f]	7	7.3	16	11.1	27	12.8	
Detroit	17	17.9	15	10.4	22	10.4	
Hartford	31	32.6	45	31.2	53	25.2	
Schizoaffective disorder	0	0	28	19.4	57	27.1	
Race							
Caucasian	54	56.8	77	53.4	139	66.1	
African-American	26	27.3	45	31.2	41	19.5	
Hispanic	6	6.3	2	1	4	1.9	
Asian	5	2.3	13	9	17	8	
Mixed	4	4.2	7	4.8	9	4.2	

[a]Healthy comparison group > Schizophrenia probands
[b]Healthy comparison group > Schizophrenia probands
[c]3 subjects including 1 healthy comparison and 2 schizophrenia patients did not have WRAT-4 score
[d]CPZ equivalent dosage data were available for 85 schizophrenia and 130 psychotic bipolar probands
[e]Disproportionate number of males in probands and disproportionate number of females in psychotic bipolar probands
[f]There were only 3 subjects from the Boston site and since identical EEG equipment was used to collect data at both Boston and Detroit sites managed by the same investigator (two sites were merged in the study). Site was used as a factor with 5 levels in the statistical analysis.
CPZ, chlorpromazine; PANSS, Positive and Negative Syndrome Scale SBS, Schizo-Bipolar Scale; SD, standard deviation; WRAT-4 wide range achievement test, 4th edition

TABLE S2. Medication Information for Probands

	Psychotic bipolar disorder (N=210)		Schizophrenia (N=144)		Healthy comparison subjects (N=95)	
	N	%	N	%	N	%
Unknown medication history	1	0.4	1	0.7	1	1.1
Medication data below are for subjects with medication history reported	209	99.6	143	99.3	94	98.9
No medication taken	6	2.8	4	2.8	40	42.6
Not on psychotropic medications	9	4.3	8	5.6	88	96.3
On more than one psychotropic medications	175	83.7	108	75.5	1	1
Anticholinergic/Antiparkinsonian	24	11.4	24	16.7	0	0
Antidepressant (Any)	106	50.7	59	41.2	1	1
A. Tricyclic	7	3.3	1	0.7	0	0
B. MAO inhibitors	0	0	0	0	0	0
C. SSRI/SNRI	51	24.4	42	29.3	1	1
D. Miscellaneous	48	22.9	16	11.1	0	0
Antipsychotic (Any)	165	78.9	133	93	0	0
A. First generation	21	10	13	9	0	0
B. Second generation	144	68.8	119	83.2	0	0
Anxiolytic/Hypnotic,	71	33.9	36	25.1	0	0
Mood Stabilizer (Any)	151	72.2	39	27.2	0	0
A. Lithium	44	21.05	12	8.4	0	0
B. Anticonvulsants	107	51.1	27	18.8	0	0
Miscellaneous, Centrally Active	8	3.8	4	2.8	0	0
Stimulants	17	8.1	6	4.2	0	0

MAO, monoamine oxidase inhibitor; SNRI, serotonin-norepinephrine reuptake inhibitors; SSRI, selective serotonin reuptake inhibitors

TABLE S3. Results of enrichment analysis including GeneGo pathway maps, process networks, metabolic networks, and gene ontology processes associated with contributing genes from components G1, G4, and G9. Bold p values indicate significance ($p<0.05$) after false discovery rate (FDR) correction.

Pathways for G1			
Pathway maps	p (uncorrected)	p (FDR)	Ratio = In data/Total
Development_slit-robo signaling	1.93E-3	0.29	3/30
Neurophysiological process_NMDA-dependent postsynaptic LTP in CA1 hippocampal neurons	4.39E-3	0.29	4/80
Cell cycle_nucleocytoplasmic transport of CDK/cyclins	5.86E-3	0.29	2/14
Neurophysiological process_ACM regulation of nerve impulse	6.56E-3	0.29	3/46
Neurophysiological process_dopamine D2 recetor signaling in CNS	6.96E-3	0.29	3/47
Mechanism of action of CCR4 antagonists in asthma and atopic dermatitis	8.35E-3	0.29	1/1
Development role of HDAC and calcium/calmodulin-dependent kinase (caMK) in control of skeletal myogenesis	1.02E-2	0.29	3/54
wtCFTR and delta508 traffic/clathrin coated vesicles formation	1.07E-2	0.29	2/19
Muscle contract_ACM regulation of smooth muscle contraction	1.12E-2	0.29	3/56
Cytoskeleton remodeling_FAK signaling	1.18E-2	0.29	3/57
Process networks			
Development_neurogenesis_axonal guidance	3.85E-5	**4.39E-3**	12/230
Cell adhesion_cadherins	5.21E-4	**2.37E-2**	9/180
Cell adhesion_synaptic contact	6.12E-4	**2.32E-2**	9/184
Reproduction_feeding and neurohormone signaling	1.61E-3	**4.6E-2**	9/211
Development_neurogenesis_synaptogenesis	2.28E-3	**5.E-2**	8/180
Neurophysiological process_LTP	4.16E-3	7.9E-2	5/82
Cell adhesion_attractive and repulsive receptors	7.58E-3	1.23E-1	7/175
Calcium transport	1.22E-2	1.75E-1	7/192
Neurophysiological process_transmission of nerve impulse	2.01E-2	2.55E-1	7/212
Cell adhesion_amyloid proteins	4.12E-2	4.69E-1	6/195
Metabolic networks			
Lysophosphatidylserine pathway	7.46E-6	**4.77E-4**	6/82
Sphingomyelin pathway	3.14E-4	**1.0E-2**	5/102
Lipid metabolism_phosphatidylinositol metabolism	1.54E-3	**3.29E-2**	4/85
Glutamic acid pathway	3.23E-3	5.18E-2	4/104
L-ornithine pathways and transport	7.16E-3	7.84E-2	4/130
L-glutamate pathways and transport	7.35E-3	7.84E-2	4/131
Glutamic acid pathways and transport	1.04E-2	9.56E-2	4/145
O-hexanoyl-(L)-carnitine pathway	1.52E-2	9.76E-2	3/88
1,2,-didocosapentaenoyl-sn-glycerol_3-phosphate_pathway	1.71E-2	9.76E-2	3/92
Myristoyl-L-carnitine pathway	1.76E-2	9.76E-2	3/93
Go processes			
Nervous system development	4.23E-15	**1.70E-11**	72/2469
Cell differentiation	1.42E-12	**2.86E-9**	83/3483
System development	3.05e-12	**4.08E-9**	100/4718
Cellular developmental process	9.55e-12	**9.59E-9**	83/3609
Neurogenesis	1.47e-11	**1.18E-8**	52/1708
Anatomical structure development	1.85e-11	**1.24E-8**	106/5304
Learning	3.48e-11	**1.99E-8**	16/162
Regulation of synapse structure and activity	4.03e-11	**2.02E-8**	13/95
Multicellular organismal development	8.15e-11	**3.63E-8**	106/5430
Single-organism developmental process	9.41e-11	**3.78E-8**	95/4624

Continued

Pathways for G4			
Pathway maps			
ATP metabolism	4.01E-4	1.14E-1	6/106
Signal transduction_cAMP signaling	4.94E-3	2.53E-1	3/38
Development_gastrin in differentiation of gastric mucosa	4.94E-3	2.53E-1	3/38
Signal transduction_PKB signaling	7.0E-3	2.53E-1	3/43
Cell cycle_nucleocytoplasmic transport of CDK/cyclins	7.01E-3	2.53E-1	2/14
Development_VEGF signaling via VEGFR2-generic cascades	7.21E-3	2.53E-1	4/84
Regulation of lipid metabolism_regulation of lipid metabolism by niacin and isoprenaline	7.95E-3	2.53E-1	3/45
G-protein signaling_regulation of cAMP levels by ACM	7.95E-3	2.53E-1	3/45
Immune response_MIF_the neuroendocorine-macrophage connector	8.44E-3	2.53E-1	3/46
Cell adhesion_integrin-mediated cell adhesion and migration	9.5E-3	2.53E-1	3/48
Process networks			
Cell adhesion_synaptic contact	7.43E-5	**8.99E-3**	11/184
Development _neurogenesis_synaptogenesis	1.25E-3	**5.0E-2**	9/180
Cell adhesion_cadherins	1.25E-3	**5.0E-2**	9/180
Development_skeletal muscle development	5.13E-3	1.45E-1	7/144
Cell adhesion_amyloid proteins	7.67E-3	1.45E-1	8/195
Proteolysis_connective tissue degradation	7.94E-3	1.45E-1	6/119
Cell adhesion_platelet aggregation	8.43E-3	1.45E-1	7/158
Development_neurogenesis_axonal guidance	1.95E-2	2.53E-1	8/230
Development neuromuscular junction	2.08E-2	2.53E-1	6/147
Apoptosis_anti-apoptosis mediated by external signals via PI3K/AKT	2.09E-2	2.53E-1	8/233
Metabolic networks			
N-acyl-sphingosine phosphate pathways	1.48E-6	**1.70E-4**	7/99
Lysophosphatidylserine pathways	7.46E-6	**4.29E-4**	6/82
Phosphatidic acid pathways	8.5E-5	**3.26E-3**	3/15
Cermaide pathway	1.06E-3	**3.07E-2**	4/77
1,2-didocosapentaenoyl-sn-glycerol_3-phosphate pathway	2.07E-3	**4.61E-2**	4/92
1,2-dioleoyl-sn-glycerol_3-phosphate pathway	2.42E-3	**4.62E-2**	4/96
1,2-didocosahexaenoyl-sn-glycerol_3-phosphate pathway	2.8E-3	**4.62E-2**	4/100
Phosphatidylinositol-4,5-diphosphate pathway	1.86E-2	2.68E-1	3/95
Sphingomyelin pathway	2.25E-2	2.87E-1	3/102
Aminoacid metabolism_asparagine, aspartic acid, arginine metabolism	7.52E-2	4.14E-1	2/76
GO processes			
Cell adhesion	6.95E-11	**1.39E-7**	39/972
Biological adhesion	8.58E-11	**1.39E-7**	39/979
Neuron development	2.99E-10	**3.23E-7**	39/1022
Neuron differentiation	4.11E-10	**3.33E-7**	43/1222
Regulation of synapse assembly	7.86E-10	**5.09E-7**	10/53
Regulation of synapse structure and activity	1.97E-10	**1.06E-6**	12/95
Positive regulation of nervous system development	4.84E-9	**1.76E-6**	8/33
Positive regulation of synapse assembly	4.84E-9	**1.76E-6**	8/33
Regulation of synapse organization	4.91E-9	**1.76E-6**	11/82
Negative regulation of filopodium assembly	5.45E-9	**1.76E-6**	5/7

Continued

Pathways for G9			
Pathway maps	p (uncorrected)	p (FDR)	Ratio = In data/Total
Immune response_alternative complement pathway	3.18E-8	**8.3E-6**	7/39
Immune response_lectin induced complement pathway	1.65E-7	**2.16E-5**	7/49
Immune response_classical complement pathway	2.53E-5	**2.2E-5**	7/52
G-protein signaling_RhoA regulation pathway	1.91E-4	**1.24E-2**	4/34
Development_slit-robo signaling	2.09E-3	1.09E-1	3/30
G-protein signaling_G-protein alpha-12 signaling pathway	3.83E-3	1.66E-1	3/37
Development_thormbopoietin-regulated cell processes	6.66E-3	1.91E-1	3/45
Beta-2-adrenergic-dependent CFTR expression	7.10E-3	1.91E-1	2/15
Development_PIP3 signaling in cardiac myocytes	7.52E-3	1.91E-1	3/47
Development_TGF-beta-dependent induction of EMT via MAPK	7.52E-3	1.91E-1	3/47
Process networks			
Cell adhesion_synaptic contact	7.77E-6	**9.72E-4**	13/184
Cell adhesion_cadherins	6.5E-4	**4.06E-2**	10/180
Inflammation_complement system	1.13E-3	**4.71E-2**	6/73
Cell adhesion_attractive and repulsive receptors	2.08E-3	6.3E-2	9/175
Development_neurogenesis synaptogenesis	2.52E-3	6.3E-2	9/180
Signal transduction_cholecytokinin signaling	7.35E-3	1.37E-1	6/106
Cytoskeleton actin filaments	7.67E-3	1.37E-1	8/176
Apoptosis_apoptosis stimulation by external signals	9.08E-3	1.42E-1	7/145
Cytoskeleton_cytoplasmic microtubules	1.08E-2	1.49E-1	6/115
Signal transduction_Wnt signaling	2.47E-2	3.02E-1	7/177
Metabolic networks			
O-hexadecaoyl-(L)-carnitine pathway	1.93E-5	**2.47E-3**	5/74
Lysophosphatidylserine pathway	6.24E-3	2.83E-1	3/82
Phosphatidylinositol-4,5-diphosphate pathway	9.37E-3	2.83E-1	3/95
CYP2C9-2-insulin-C/EBP-IRS1	1.07E-2	2.83E-1	3/100
CYP2C9-1-insulin-C/EBP-IRS1	1.10E-2	2.83E-1	3/101
1-linoleoyl-glycerol_3-phosphate pathway	6.12E-2	3.75E-1	2/87
CYP2D6-5-Glucagon-HNF4	6.12E-2	3.75E-1	2/87
CYP2C9-3-Glucagon-HNF4alpha	6.62E-2	3.75E-1	2/91
CYP2C9-2-Glucagon-HNF4alpha	6.75E-2	3.75E-1	2/92
Acyl-L-carnitine pathway	6.75E-2	3.75E-1	2/92
GO processes			
Nervous system development	2.78E-15	**9.85E-12**	76/2469
Cell projection organization	2.31E-14	**4.09E-11**	47/1105
Cell differentiation	4.25E-14	**5.02E-11**	91/3483
Cell development	5.84E-14	**5.18E-11**	61/1805
Cellular developmental process	1.26E-13	**8.96E-11**	92/3609
System development	1.52E-13	**9.02E-11**	109/4718
Neurogenesis	2.30E-13	**1.16E-10**	58/1708
Multicellular organismal signaling	3.04E-13	**1.34E-10**	42/964
Neuron development	4.95E-13	**1.94E-10**	43/1022
Multicellular organismal development	5.88E-13	**1.94E-10**	118/5430

ACM, astrocyte conditioned medium; ATP, adenosine triphosphate; CA1, cornu Ammonis1; cAMP, cyclic adenosine monophosphate; CCR4, CC-chemokine receptor 4; CDK, cyclin-dependent kinase; CFTR, cystic fibrosis transmembrane conductance regulator; CNS, central nervous system; EBP, enhancer binding protein; EMT, epithelial-mesenchymal transition; FAK, focal adhesion kinase; HNF, hepatic nuclear factor; IRS1, insulin receptor substrate 1; LTP, long term potentiation; MAPK, mitogen-activated protein kinase; MIF, migration inhibitor factor;

NMDA, N-methyl-D-Aspartate; PI3K, phosphoinositide 3-kinase; PIP3, phosphatidylinositol (3,4,5)-triphosphate; PKB, protein kinase B; TGF, transforming growth factor; VEGF, vascular endothelial growth factor, VEGFR, vascular endothelial growth factor receptor,

TABLE S4. Pathways, disease risk, and brain regions associated with top 20 genes from gene components G1, G4, and G9. Expression Z-scores from Allen brain atlas database was used to identify brain regions. Other mental disorders associated with these genes from prior studies are also provided (references in online supplementary text). Gene information was obtained from Dbsnp and Genecard.

Gene network G1				
Gene	SNP	Disease risk	Pathway involved	Allen atlas location
DCC	rs16956411[I]	SZ (19)		HP, NA & SN
BOC	rs775228[I]		Hedgehog signaling	CC, HP & AM
SEC14L2	rs4820845[I]			SRG, SN & GP
PDLIM5	rs13121500[I]	SZ (20), PBP (21, 22) & MDD (23)		HT, PT & SN
HDAC9	rs12699994[I]	SZ (24)	Immune response, histone modification & valproic acid	SRG, HT & TH
MAML3	rs7678266[I]		NOTCH signaling	CC, IRG & HT
B3GNTL1	rs1001865[I]		Post-translational protein modification	TH, IRG & TP
TBCD	rs3785520[I]		Protein folding	HP, SRG & ST
SNAP91	rs1546977[I]	MIPBP (25)	Transport clathrin-coated vesicle cycle	HP, CC & TH
LY9	rs574610[Utr-3]			HT, TH & CC
CD244	rs485618[Utr-3]		Hemostasis	MOG, TL & IN
CDKAL1	rs7758129[I]			CC, HT & SN
ESRRG	rs3929399[I]		Generic & nuclear transcription	CN, CC & CU
MCTP2	rs1655455[I]	SZ (26), PBP (27), MDD (28) & ASD (29, 30)		IFG, HT & ST
PIP4K2A	rs7071450[I]	SZ (31)	Ca^{2+}, cAMP & lipid signaling, regulates actin cytoskeleton and phospholipid metabolism	TH, POG & CN
TMEFF2	rs10185068[I]			AM, TH & SG
ZC3H18	rs12445653[I]			CC, ST & GP
VWA3B	rs10211067[Utr-5]			HT, AM & ST
RASGRP3	rs11687777[I]		G-protein H-RAS regulation, MAPK; B cell receptor signaling	CN, GP & TH
ANKIB1	rs721015[I]			CN, TH & SN
Gene network G4				
Gene	SNP	Disease risk	Pathway involved	Allen atlas location
MSRA	rs7459532[I]	SZ (32, 33), PBP (34) & AD (35)		CC, HP &TH
XKR6	rs2409691[I]			CC, AM & HP
RP1L1	rs7386213[I]			CC, TH & HT
BLK	rs2618451[I]		Tyrosine kinases, Immune system, lymphocyte & B cell receptor signaling	CC, FO & HP
TNKS	rs7840706[I]		NAD metabolism	CC, VT & CN
TPO	rs2276702[I]		Metabolic, thyroxine biosynthesis	CC, HT & ITG
IL1F10	rs6761276[M]			HT, SN & AM
MFHAS1	rs4841044[I]			CC, VT & OL
ADAMTS16	rs270178[I]			CC, SG & HP
DOCK8	rs10967788[I]	ASD (36) & MR (37)	Hemostasis	CN, GP & SN
TAF8	rs6917299[I]		Ligand-dependent transcription of retinoid target genes, Basal transcription factors	SRG, VT & AM

Continued

GSG1L	rs1645362[I]			TH, SN & ST
ODZ3	rs957053[I]			TH, AM & HP
VAT1L	rs9933953[I]			AM, HT & LC
USH2A	rs17025267[I]			TH, AM & VT
COL2A1[‡]	rs1793923[I]		CA intergin-inside-out signaling, focal adhesion & ECM receptor interaction	CC, HT & LC
INPP5K	rs1109303[I]		Metabolic, phosphatidylinositol signaling system	TH, SN & HT
NRXN3	rs10782463[I]	SZ (38), ASD (39) & ALD (40)	Brain CAM, GABA signaling	CC, IFG & AM
PNPLA1	rs12197079[M]			SN, AM & CC
PKP3	rs7105848[I]			MOG, HT & ITG
Gene network G9				
Gene	SNP	Disease risk	Pathway involved	Allen atlas location
ME1	rs1170348[I]			HT, VT & LC
PEMT	rs11078389[I]	SZ (41) & AD (42)	Metabolic, acetylcholine synthesis	CC, IFG & HT
GPC6	rs4369513[I]		WNT signaling, metabolism	HT, VT & LC
SNAP91	rs217291[I]			CC, HP & TH
DLGAP1	rs1465947[I]	SZ (43) & OCD (44)	Glutamatergic synapse	HP, SOG & IFG
PRSS35	rs592911[I]	MIPBP (25)		AM, SG & NA
PCSK5	rs2842467[I]		Signal transduction & NGF signaling	HT, GP & HP
FRK	rs12662901[I]	SZ,-AL (45) & AD (46)		GP, SN & AM
KCTD8	rs2020159[I]			CC, TH & AM
PDLIM1	rs11593722[I]	ADHD (47) & AD (48, 49)		CC, SFG & TP
CYP2C19	rs10786172[I]	D (50) & P (51)	Estrogen biosynthesis, estrone metabolism & arachidonic acid	CG, TH & PG
ANO2	rs1035066[I]		Ion channel transport	HT, ST & AM
SRRM4	rs1405050[I]			CC, SRG & IOG
YSK4	rs4953941[I]			AM, HT & ST
CNTNAP2[§, ¶]	rs700281[I]	SZ (52, 53), MDD (53), P(54), PBP (52), ASD (55, 56) & ADHD (57)	CAM	MOG, TH & LC
PRDM16	rs1798246[I]			TH, SRG & ST
TNNI1	rs3767548[I]		Striated muscle contraction	AM, TH & VT
GLT1D1	rs516034[I]			SRG, HT & STG
ESRRG	rs1833036[I]			CN, CU & TH
ZNF385B	rs10432487[I]			TH, CU & ST

[‡]Present in the top 100 genes in European-American (Accession: pha002858.1) genome wide study of bipolar disorder (58) from database of Genotypes and phenotypes (dbGap)

[§]Present in the top 100 genes in African-American (Accession: pha002863.1) genome wide study of bipolar disorder (58) from dbGap

[¶]Present in the top 100 genes in European-American (Accession: pha002857.1) genome wide study of schizophrenia (58) from dbGap

AD, Alzheimer's disease; ADHD, attention deficit-hyperactivity disorder; AL, alcoholism; AM, amygdala; ASD, autism spectrum disorder; CA, cell adhesion; Ca^{2+}, calcium ion; CAM, cell adhesion molecule; cAMP, cyclic adenosine monophosphate; CC, cerebellar cortex; CP, cell proliferation; CPD, cell proliferation and differentiation; CN, cerebellar nuclei; CNS; central

nervous system; CU, cuneus; D, depression; ECM, extracellular matrix; FO, frontal operculum; GP, globus pallidus; HC, histocompatibility complex; HP, hippocampal formation; HT, hypothalamus; I, intron; IFG, inferior frontal gyrus; IN, insula; IOG, inferior occipital gyrus; ITG, inferior temporal gyrus; IRG, inferior rostral gyrus; LC, locus coeruleus; M, missense; MAPK, mitogen-activated protein kinase; MDD, major depressive disorder; MIPBP, mood incongruent; MOG, medial orbital gyrus; MR, mental retardation; NA, nucleus accumbens; NAD, nicotinamide adenine dinucleotide; NGF, nerve growth factor; OCD, obsessive-compulsive disorder; P, personality traits; PBP, psychotic bipolar disorder; PD, parkinson disease; PG, parolfactory gyri, POG, posterior orbital gyrus; PT, pontine tegmentum; SFG, superior frontal gyrus; SG, subcallosal gyrus; SMG, ; SN, substantia nigra; SNP, single nucleotide polymorphism; SOG, superior occipital gyrus; STG, superior temporal gyrus; SRG, superior rostral gyrus; ST, striatum; SZ, schizophrenia; TH, thalamus; TP, temporal pole; Utr-3, three prime untranslated region; Utr-5 five prime untranslated region; VT, ventral tegmental area;

FIGURE S1. Schematic of the 64-Channel EEG Montage Used for Data Collection

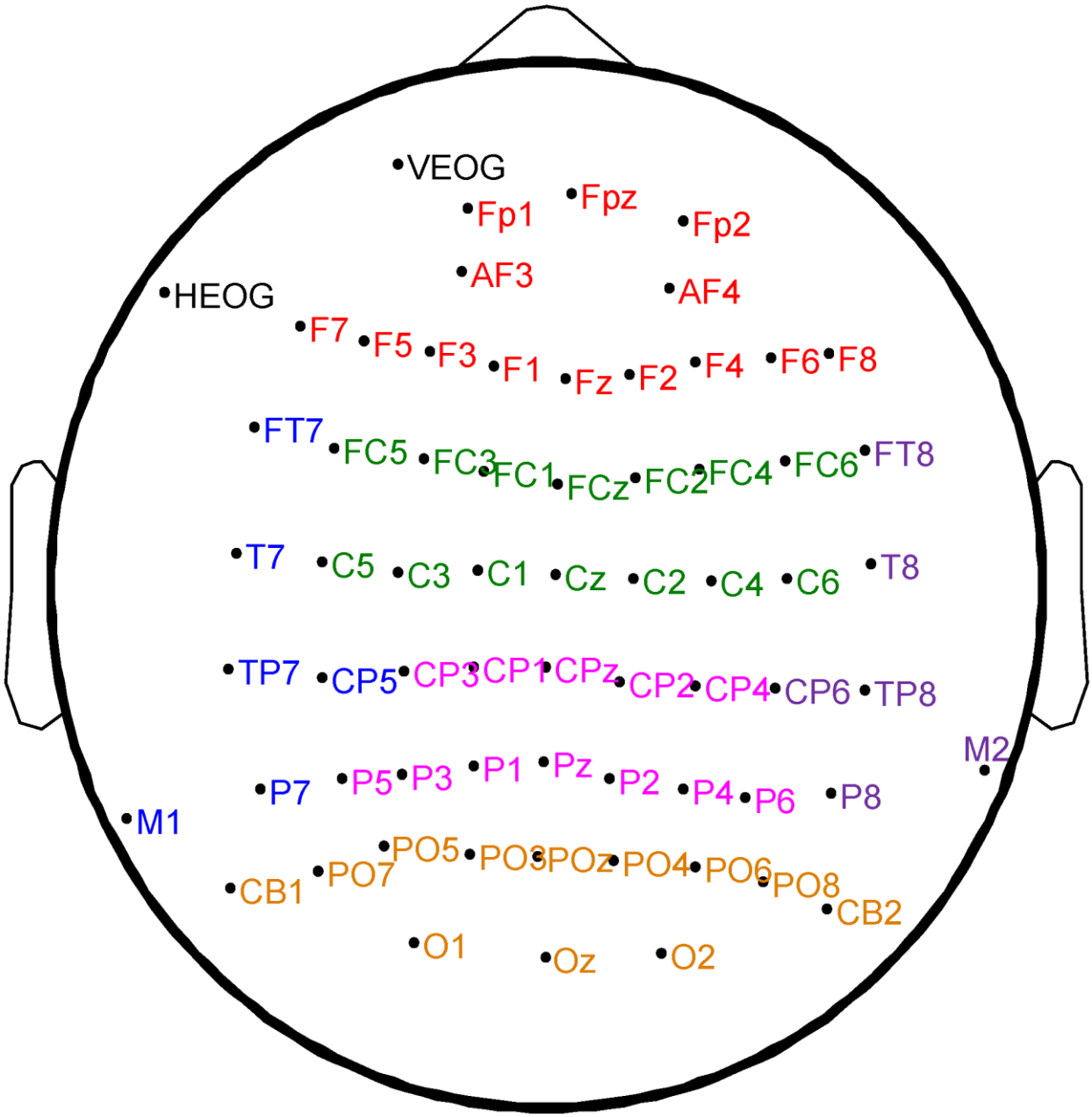
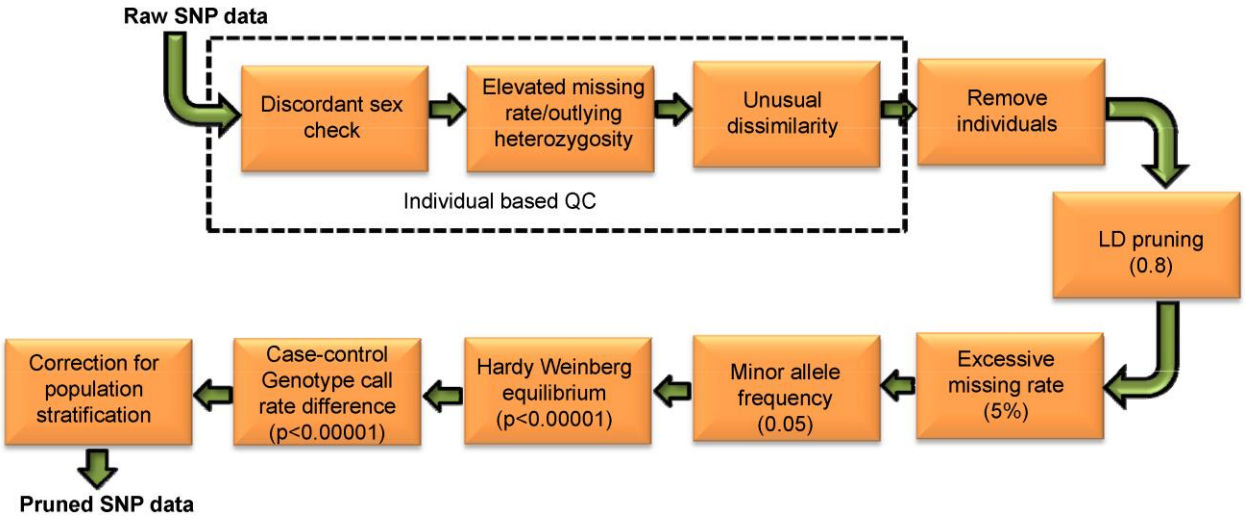


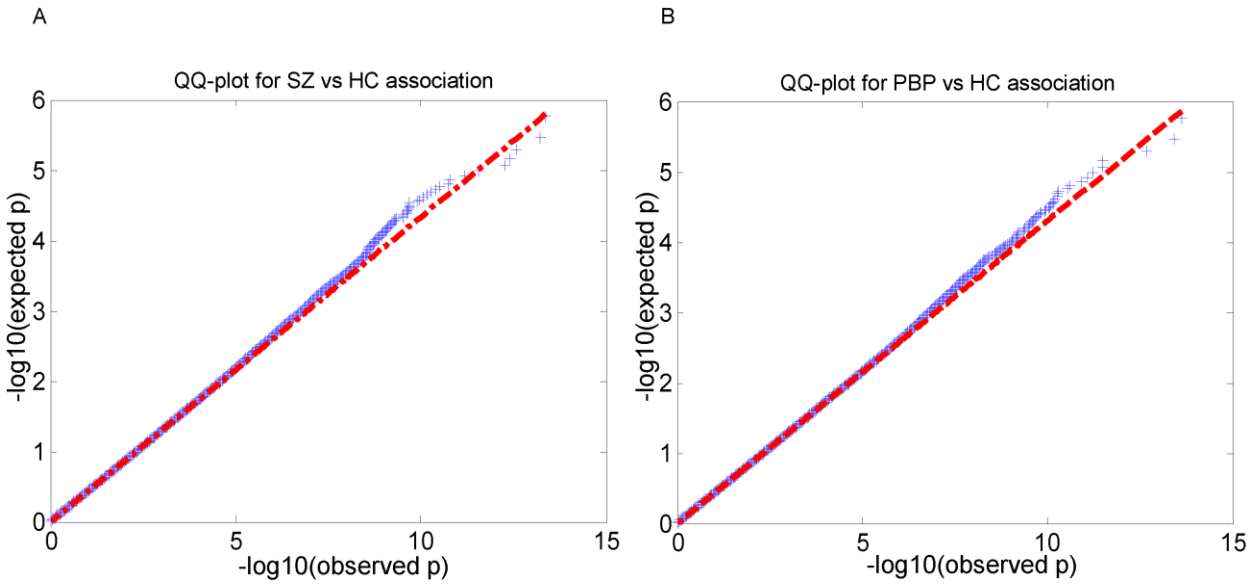
FIGURE S2. Schematic Depiction of the Processing Pipeline for Quality Control of Single-Nucleotide Polymorphism (SNP) Data[a]



[a] The process involved two stages: individual based followed by SNP-based quality control. Poorly genotyped individuals and bad SNPs were removed from the final regression analysis. SNP data were corrected for stratification bias by correcting for the top three eigen factors associated with self-reported ethnicity. LD=linkage disequilibrium.

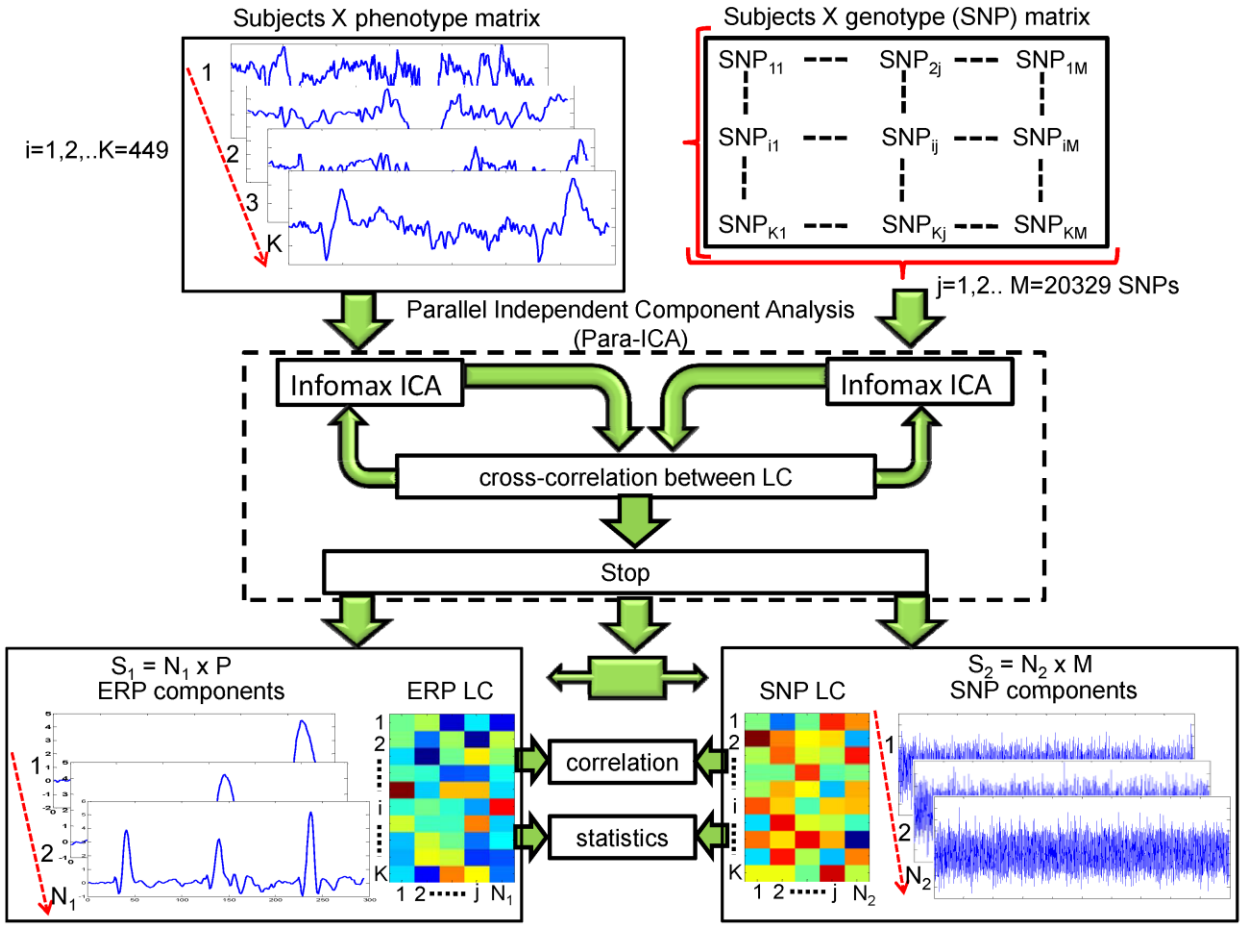
*p<0.05. **p<0.0001. ***p<<0.000000001.

FIGURE S3. Quantile-Quantile Plot of Theoretical and Empirical p Values From Logistic Regression for Schizophrenia Probands (SZ) vs Healthy Comparison Subjects (HC), and for Psychotic Bipolar Disorder Probands (PBP) vs. HC[a]



[a] Logistic regression was applied to individual markers in case-control fashion for both the SZ and PBP groups.

FIGURE S4. Schematic Illustration of the Parellel Independent Component Analysis (Para-ICA) for Genetic Association of Event-Related Potential (ERP) Data[a]



[a] Data were constructed as a matrix of subjects by SNP (K=449 × M=20,329) and subjects by reduced ERP waveforms (K=449 × p=294 [3 × 98 time points]). The number of components extracted from Para-ICA for the ERP and SNP data were N_1=8 and N_2=11, respectively. LC=loading coefficient; SNP=single-nucleotide polymorphism.

Supplementary References

1. First MB, Spitzer RL, Gibbs P, Williams NM: Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) Version 2.0. New York, Biometrics Research Department, New York State Psychiatric Institute; 1998.
2. Strasser HC, Lilyestrom J, Ashby ER, Honeycutt NA, Schretlen DJ, Pulver AE, Hopkins RO, Depaulo JR, Potash JB, Schweizer B, Yates KO, Kurian E, Barta PE, Pearlson GD. Hippocampal and ventricular volumes in psychotic and nonpsychotic bipolar patients compared with schizophrenia patients and community control subjects: a pilot study. Biol Psychiatry 2005; 57:633-639.
3. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. Schizophr Bull 1987; 13:261-276.
4. Young RC, Biggs JT, Ziegler VE, Meyer DA. A rating scale for mania: reliability, validity and sensitivity. Br J Psychiatry 1978; 133:429-435.
5. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. Br J Psychiatry 1979; 134:382-389.
6. Arnold SJ, Ivleva EI, Gopal TA, Reddy AP, Jeon-Slaughter H, Sacco CB, Francis AN, Tandon N, Bidesi AS, Witte B, Poudyal G, Pearlson GD, Sweeney JA, Clementz BA, Keshavan MS, Tamminga CA. Hippocampal Volume Is Reduced in Schizophrenia and Schizoaffective Disorder But Not in Psychotic Bipolar I Disorder Demonstrated by Both Manual Tracing and Automated Parcellation (FreeSurfer). Schizophr Bull 2014.
7. Dale AM, Fischl B, Sereno MI. Cortical surface-based analysis. I. Segmentation and surface reconstruction. Neuroimage 1999; 9:179-194.
8. Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J Neurosci Methods 2004; 134:9-21.
9. Ethridge LE, Hamm JP, Shapiro JR, Summerfelt AT, Keedy SK, Stevens MC, Pearlson G, Tamminga CA, Boutros NN, Sweeney JA, Keshavan MS, Thaker G, Clementz BA. Neural activations during auditory oddball processing discriminating schizophrenia and psychotic bipolar disorder. Biol Psychiatry 2012; 72:766-774.
10. Clementz BA, Blumenfeld LD. Multichannel electroencephalographic assessment of auditory evoked response suppression in schizophrenia. Exp Brain Res 2001; 139:377-390.
11. Dien J, Khoe W, Mangun GR. Evaluation of PCA and ICA of simulated ERPs: Promax vs. Infomax rotations. Hum Brain Mapp 2007; 28:742-763.
12. Anderson CA, Pettersson FH, Clarke GM, Cardon LR, Morris AP, Zondervan KT. Data quality control in genetic case-control association studies. Nat Protoc 2010; 5:1564-1573.
13. Purcell S, Neale B, Todd-Brown K, Thomas L, Ferreira MA, Bender D, Maller J, Sklar P, de Bakker PI, Daly MJ, Sham PC. PLINK: a tool set for whole-genome association and population-based linkage analyses. Am J Hum Genet 2007; 81:559-575.
14. Meda SA, Narayanan B, Liu J, Perrone-Bizzozero NI, Stevens MC, Calhoun VD, Glahn DC, Shen L, Risacher SL, Saykin AJ, Pearlson GD. A large scale multivariate parallel ICA method reveals novel imaging-genetic relationships for Alzheimer's disease in the ADNI cohort. Neuroimage 2012; 60:1608-1621.
15. Liu J, Demirci O, Calhoun VD. A parallel independent component analysis approach to Investigate genomic influence on brain function. IEEE Signal Process Lett 2008; 15:413-416.
16. Rissanen J. A universal prior for integers and estimation by minimum description length. The Annals of Statistics 1983; 11:16.
17. Calhoun VD, Adali T, Pearlson GD, Pekar JJ. A method for making group inferences from functional MRI data using independent component analysis. Hum Brain Mapp 2001; 14:140-151.
18. Chen J, Calhoun VD, Liu J. ICA order selection based on consistency: application to genotype data. Conf Proc IEEE Eng Med Biol Soc 2012; 2012:360-363.
19. Grant A, Fathalli F, Rouleau G, Joober R, Flores C. Association between schizophrenia and genetic variation in DCC: a case-control study. Schizophr Res 2012; 137:26-31.

20. Kato T, Iwayama Y, Kakiuchi C, Iwamoto K, Yamada K, Minabe Y, Nakamura K, Mori N, Fujii K, Nanko S, Yoshikawa T. Gene expression and association analyses of LIM (PDLIM5) in bipolar disorder and schizophrenia. Mol Psychiatry 2005; 10:1045-1055.
21. Zhao T, Liu Y, Wang P, Li S, Zhou D, Zhang D, Chen Z, Wang T, Xu H, Feng G, He L, Yu L. Positive association between the PDLIM5 gene and bipolar disorder in the Chinese Han population. J Psychiatry Neurosci 2009; 34:199-204.
22. Squassina A, Manchia M, Manconi F, Piccardi M, Ardau R, Chillotti C, Severino G, Del Zompo M. A case-control association study of the PDLIM5 gene and bipolar disorder in a Sardinian sample. Psychiatr Genet 2008; 18:128-132.
23. Liu Z, Liu W, Xiao Z, Wang G, Yin S, Zhu F, Wang H, Cheng J, Wang X, He X, Li W. A major single nucleotide polymorphism of the PDLIM5 gene associated with recurrent major depressive disorder. J Psychiatry Neurosci 2008; 33:43-46.
24. Lang B, Alrahbeni TM, Clair DS, Blackwood DH, McCaig CD, Shen S. HDAC9 is implicated in schizophrenia and expressed specifically in post-mitotic neurons but not in adult neural stem cells. Am J Stem Cells 2011; 1:31-41.
25. Goes FS, Zandi PP, Miao K, McMahon FJ, Steele J, Willour VL, Mackinnon DF, Mondimore FM, Schweizer B, Nurnberger JI, Jr., Rice JP, Scheftner W, Coryell W, Berrettini WH, Kelsoe JR, Byerley W, Murphy DL, Gershon ES, Bipolar Disorder Phenome G, Depaulo JR, Jr., McInnis MG, Potash JB. Mood-incongruent psychotic features in bipolar disorder: familial aggregation and suggestive linkage to 2p11-q14 and 13q21-33. Am J Psychiatry 2007; 164:236-247.
26. Djurovic S, Le Hellard S, Kahler AK, Jonsson EG, Agartz I, Steen VM, Hall H, Wang AG, Rasmussen HB, Melle I, Werge T, Andreassen OA. Association of MCTP2 gene variants with schizophrenia in three independent samples of Scandinavian origin (SCOPE). Psychiatry Res 2009; 168:256-258.
27. McAuley EZ, Blair IP, Liu Z, Fullerton JM, Scimone A, Van Herten M, Evans MR, Kirkby KC, Donald JA, Mitchell PB, Schofield PR. A genome screen of 35 bipolar affective disorder pedigrees provides significant evidence for a susceptibility locus on chromosome 15q25-26. Mol Psychiatry 2009; 14:492-500.
28. Verma R, Holmans P, Knowles JA, Grover D, Evgrafov OV, Crowe RR, Scheftner WA, Weissman MM, DePaulo JR, Jr., Potash JB, Levinson DF. Linkage disequilibrium mapping of a chromosome 15q25-26 major depression linkage region and sequencing of NTRK3. Biol Psychiatry 2008; 63:1185-1189.
29. Chagnon YC. Shared susceptibility region on chromosome 15 between autism and catatonia. Int Rev Neurobiol 2006; 72:165-178.
30. Chagnon YC. Shared chromosomal susceptibility regions between autism and other mental disorders. Int Rev Neurobiol 2005; 71:419-443.
31. Thiselton DL, Maher BS, Webb BT, Bigdeli TB, O'Neill FA, Walsh D, Kendler KS, Riley BP. Association analysis of the PIP4K2A gene on chromosome 10p12 and schizophrenia in the Irish study of high density schizophrenia families (ISHDSF) and the Irish case-control study of schizophrenia (ICCSS). Am J Med Genet B Neuropsychiatr Genet 2010; 153B:323-331.
32. Ma X, Deng W, Liu X, Li M, Chen Z, He Z, Wang Y, Wang Q, Hu X, Collier DA, Li T. A genome-wide association study for quantitative traits in schizophrenia in China. Genes Brain Behav 2011; 10:734-739.
33. Walss-Bass C, Soto-Bernardini MC, Johnson-Pais T, Leach RJ, Ontiveros A, Nicolini H, Mendoza R, Jerez A, Dassori A, Chavarria-Siles I, Escamilla MA, Raventos H. Methionine sulfoxide reductase: a novel schizophrenia candidate gene. Am J Med Genet B Neuropsychiatr Genet 2009; 150B:219-225.
34. Walss-Bass C, Escamilla MA, Raventos H, Montero AP, Armas R, Dassori A, Contreras S, Liu W, Medina R, Balderas TG, Levinson D, Pereira R, Pereira M, Atmella I, Nesmith L, Leach R, Almasy L. Evidence of genetic overlap of schizophrenia and bipolar disorder: linkage disequilibrium

analysis of chromosome 18 in the Costa Rican population. Am J Med Genet B Neuropsychiatr Genet 2005; 139B:54-60.
35. Gabbita SP, Aksenov MY, Lovell MA, Markesbery WR. Decrease in peptide methionine sulfoxide reductase in Alzheimer's disease brain. J Neurochem 1999; 73:1660-1666.
36. Menashe I, Larsen EC, Banerjee-Basu S. Prioritization of Copy Number Variation Loci Associated with Autism from AutDB-An Integrative Multi-Study Genetic Database. PLoS One 2013; 8:e66707.
37. Griggs BL, Ladd S, Saul RA, DuPont BR, Srivastava AK. Dedicator of cytokinesis 8 is disrupted in two patients with mental retardation and developmental disabilities. Genomics 2008; 91:195-202.
38. Hu X, Zhang J, Jin C, Mi W, Wang F, Ma W, Ma C, Yang Y, Li W, Zhang H, Du B, Li K, Liu C, Wang L, Lu T, Lv L, Zhang D, Yue W. Association study of NRXN3 polymorphisms with schizophrenia and risperidone-induced bodyweight gain in Chinese Han population. Prog Neuropsychopharmacol Biol Psychiatry 2013; 43:197-202.
39. Vaags AK, Lionel AC, Sato D, Goodenberger M, Stein QP, Curran S, Ogilvie C, Ahn JW, Drmic I, Senman L, Chrysler C, Thompson A, Russell C, Prasad A, Walker S, Pinto D, Marshall CR, Stavropoulos DJ, Zwaigenbaum L, Fernandez BA, Fombonne E, Bolton PF, Collier DA, Hodge JC, Roberts W, Szatmari P, Scherer SW. Rare deletions at the neurexin 3 locus in autism spectrum disorder. Am J Hum Genet 2012; 90:133-141.
40. Hishimoto A, Liu QR, Drgon T, Pletnikova O, Walther D, Zhu XG, Troncoso JC, Uhl GR. Neurexin 3 polymorphisms are associated with alcohol dependence and altered expression of specific isoforms. Hum Mol Genet 2007; 16:2880-2891.
41. Liu Y, Zhang H, Ju G, Zhang X, Xu Q, Liu S, Yu Y, Shi J, Boyle S, Wang Z, Shen Y, Wei J. A study of the PEMT gene in schizophrenia. Neurosci Lett 2007; 424:203-206.
42. Bi XH, Zhao HL, Zhang ZX, Zhang JW. PEMT G523A (V175M) is associated with sporadic Alzheimer's disease in a Chinese population. J Mol Neurosci 2012; 46:505-508.
43. Li JM, Lu CL, Cheng MC, Luu SU, Hsu SH, Chen CH. Genetic analysis of the DLGAP1 gene as a candidate gene for schizophrenia. Psychiatry Res 2013; 205:13-17.
44. Stewart SE, Yu D, Scharf JM, Neale BM, Fagerness JA, Mathews CA, Arnold PD, Evans PD, Gamazon ER, Osiecki L, McGrath L, Haddad S, Crane J, Hezel D, Illman C, Mayerfeld C, Konkashbaev A, Liu C, Pluzhnikov A, Tikhomirov A, Edlund CK, Rauch SL, Moessner R, Falkai P, Maier W, Ruhrmann S, Grabe HJ, Lennertz L, Wagner M, Bellodi L, Cavallini MC, Richter MA, Cook EH, Jr., Kennedy JL, Rosenberg D, Stein DJ, Hemmings SM, Lochner C, Azzam A, Chavira DA, Fournier E, Garrido H, Sheppard B, Umana P, Murphy DL, Wendland JR, Veenstra-Vanderweele J, Denys D, Blom R, Deforce D, Van Nieuwerburgh F, Westenberg HG, Walitza S, Egberts K, Renner T, Miguel EC, Cappi C, Hounie AG, Conceicao do Rosario M, Sampaio AS, Vallada H, Nicolini H, Lanzagorta N, Camarena B, Delorme R, Leboyer M, Pato CN, Pato MT, Voyiaziakis E, Heutink P, Cath DC, Posthuma D, Smit JH, Samuels J, Bienvenu OJ, Cullen B, Fyer AJ, Grados MA, Greenberg BD, McCracken JT, Riddle MA, Wang Y, Coric V, Leckman JF, Bloch M, Pittenger C, Eapen V, Black DW, Ophoff RA, Strengman E, Cusi D, Turiel M, Frau F, Macciardi F, Gibbs JR, Cookson MR, Singleton A, Arepalli S, Dillman A, Ferrucci L, Hernandez DG, Johnson R, Longo DL, Nalls MA, R OB, Traynor B, Troncoso J, van der Brug M, Zielke HR, Zonderman A, Hardy J, Hardy JA, Ryten M, Smith C, Trabzuni D, Walker R, Weale M, Crenshaw AT, Parkin MA, Mirel DB, Conti DV, Purcell S, Nestadt G, Hanna GL, Jenike MA, Knowles JA, Cox N, Pauls DL. Genome-wide association study of obsessive-compulsive disorder. Mol Psychiatry 2013; 18:788-798.
45. Ishiguro H, Saito T, Shibuya H, Toru M, Arinami T. Mutation and association analysis of the Fyn kinase gene with alcoholism and schizophrenia. Am J Med Genet 2000; 96:716-720.
46. Shirazi SK, Wood JG. The protein tyrosine kinase, fyn, in Alzheimer's disease pathology. Neuroreport 1993; 4:435-437.
47. Wang KS, Liu X, Zhang Q, Aragam N, Pan Y. Parent-of-origin effects of FAS and PDLIM1 in attention-deficit/hyperactivity disorder. J Psychiatry Neurosci 2012; 37:46-52.

48. Grupe A, Li Y, Rowland C, Nowotny P, Hinrichs AL, Smemo S, Kauwe JS, Maxwell TJ, Cherny S, Doil L, Tacey K, van Luchene R, Myers A, Wavrant-De Vrieze F, Kaleem M, Hollingworth P, Jehu L, Foy C, Archer N, Hamilton G, Holmans P, Morris CM, Catanese J, Sninsky J, White TJ, Powell J, Hardy J, O'Donovan M, Lovestone S, Jones L, Morris JC, Thal L, Owen M, Williams J, Goate A. A scan of chromosome 10 identifies a novel locus showing strong association with late-onset Alzheimer disease. Am J Hum Genet 2006; 78:78-88.
49. Chapuis J, Hot D, Hansmannel F, Kerdraon O, Ferreira S, Hubans C, Maurage CA, Huot L, Bensemain F, Laumet G, Ayral AM, Fievet N, Hauw JJ, DeKosky ST, Lemoine Y, Iwatsubo T, Wavrant-Devrieze F, Dartigues JF, Tzourio C, Buee L, Pasquier F, Berr C, Mann D, Lendon C, Alperovitch A, Kamboh MI, Amouyel P, Lambert JC. Transcriptomic and genetic studies identify IL-33 as a candidate gene for Alzheimer's disease. Mol Psychiatry 2009; 14:1004-1016.
50. Sim SC, Nordin L, Andersson TM, Virding S, Olsson M, Pedersen NL, Ingelman-Sundberg M. Association between CYP2C19 polymorphism and depressive symptoms. Am J Med Genet B Neuropsychiatr Genet 2010; 153B:1160-1166.
51. Ishii G, Suzuki A, Oshino S, Shiraishi H, Otani K. CYP2C19 polymorphism affects personality traits of Japanese females. Neurosci Lett 2007; 411:77-80.
52. Wang KS, Liu XF, Aragam N. A genome-wide meta-analysis identifies novel loci associated with schizophrenia and bipolar disorder. Schizophr Res; 124:192-199.
53. Ji W, Li T, Pan Y, Tao H, Ju K, Wen Z, Fu Y, An Z, Zhao Q, Wang T, He L, Feng G, Yi Q, Shi Y. CNTNAP2 is significantly associated with schizophrenia and major depression in the Han Chinese population. Psychiatry Res 2013; 207:225-228.
54. Terracciano A, Sanna S, Uda M, Deiana B, Usala G, Busonero F, Maschio A, Scally M, Patriciu N, Chen WM, Distel MA, Slagboom EP, Boomsma DI, Villafuerte S, Sliwerska E, Burmeister M, Amin N, Janssens AC, van Duijn CM, Schlessinger D, Abecasis GR, Costa PT, Jr. Genome-wide association scan for five major dimensions of personality. Mol Psychiatry 2010; 15:647-656.
55. Li X, Hu Z, He Y, Xiong Z, Long Z, Peng Y, Bu F, Ling J, Xun G, Mo X, Pan Q, Zhao J, Xia K. Association analysis of CNTNAP2 polymorphisms with autism in the Chinese Han population. Psychiatr Genet 2010; 20:113-117.
56. Alarcon M, Abrahams BS, Stone JL, Duvall JA, Perederiy JV, Bomar JM, Sebat J, Wigler M, Martin CL, Ledbetter DH, Nelson SF, Cantor RM, Geschwind DH. Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. Am J Hum Genet 2008; 82:150-159.
57. Elia J, Gai X, Xie HM, Perin JC, Geiger E, Glessner JT, D'Arcy M, deBerardinis R, Frackelton E, Kim C, Lantieri F, Muganga BM, Wang L, Takeda T, Rappaport EF, Grant SF, Berrettini W, Devoto M, Shaikh TH, Hakonarson H, White PS. Rare structural variants found in attention-deficit hyperactivity disorder are preferentially associated with neurodevelopmental genes. Mol Psychiatry 2010; 15:637-646.
58. Mailman MD, Feolo M, Jin Y, Kimura M, Tryka K, Bagoutdinov R, Hao L, Kiang A, Paschall J, Phan L, Popova N, Pretel S, Ziyabari L, Lee M, Shao Y, Wang ZY, Sirotkin K, Ward M, Kholodov M, Zbicz K, Beck J, Kimelman M, Shevelev S, Preuss D, Yaschenko E, Graeff A, Ostell J, Sherry ST. The NCBI dbGaP database of genotypes and phenotypes. Nat Genet 2007; 39:1181-1186.