**SUPPLEMENTARY MATERIAL: Figure and Tables, and study cohort details, authors and affiliations for h2-SNP data**

**Genetic influences on eight psychiatric disorders based on family data of 4 408 646 full and half siblings, and genetic data of 333 748 cases and controls**

**By Polderman et al.**

Table of contents Page

**SUPPLEMENTARY TABLES**

Table 1: ICD codes for all eight disorders as used for h2-national estimate 2

Table 2: Descriptives national sibling study 3

Table 3: Descriptives SNP-based studies 4

**SUPPLEMENTARY STUDY COHORT DETAILS** h2-SNP

Alcohol Dependence 5

Anorexia Nervosa 9

Attention Deficit/Hyperactivity Disorder 11

Autism Spectrum Disorder 12

Bipolar Disorder 14

Major Depressive Disorder 19

Obsessive Compulsive Disorder 20

Schizophrenia 22

References 25

Collaborating Authors and Affiliations 30

**Supplement Table 1. ICD codes for all eight disorders as used for h2-national estimates**

|  |
| --- |
| **Definition of psychiatric disorders** |
| Psychiatric disorder | ICD 8 (1969-1986) | ICD 9 (1987-1996) | ICD 10 (1997- ) |
| Alcohol Dependence | 291, 2910, 2911, 2912, 2913, 2919, 303, 3030, 3031, 3032, 3039, 5710 | 291, 291A, 291B, 291C, 291D, 291E, 291F, 291W, 291X, 303, 303A, 303X, 357F, 357G, 425F, 535D, 571A, 571B, 571C, 571D, 980, 980A, V11D | F10, F100, F101, F102, F103, F104, F105, F106, F107, F108, G621, G721, I426, K292, K70, K700, K701, K702, K703, K704, K709, K860 |
| Anorexia Nervosa | 3065, 7840 | 307B | F500, F501 |
| ADHD | --- | 314, 314A, 314B, 314C, 314J, 314W, 314X | F90, F900, F901, F908 |
| Autism Spectrum Disorder | --- | 299A | F840, F841, F845 |
| Bipolar Disorder | 2961, 2962, 2963 | 296A, 296C, 296D, 296E, 296F, 296G, 296H, 296W, 296X | F30, F301, F302, F308, F309, F31, F310, F311, F312, F313, F314, F315, F316, F317, F318, F319 |
| Major Depressive Disorder | 2960, 300E | 296B, 311 | F32, F320, F321, F322, F323, F328, F329, F33, F330, F331, F332, F333, F334, F338, F339, F34, F348, F349, F38, F380, F381, F388, F39 |
| Obsessive Compulsive Disorder  | 3003 | 300D | F42, F420, F421, F422, F428, F429 |
| Schizophrenia | 295, 2950, 2951, 2952, 2953, 2954, 2956, 2957, 2958, 2959 | 295, 295A, 295B, 295C, 295D, 295E, 295G, 295H, 295W, 295X | F20, F200, F201, F202, F203, F204, F205, F206, F208, F209, F25, F250, F251, F252, F258, F259 |

**Supplement Table 2. Descriptives national sibling study: Per disorder, the number of included full and half siblings, prevalences, tetrachoric sibling correlations and estimated heritability (h2), including SE**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **Full siblings** | **Maternal half siblings** | **Tetrachoric correlation (SE)** | **h2 (SE)** |
|  | **N affected** | **N unaffected** | **Prevalence****in sample** | **N affected** | **N unaffected** | **Prevalence****in sample** | **Full sibs** | **Maternal half sibs** |  |
| Alcohol Dependence | 89,526 | 3,567,472 | 2.4% | 11,094 | 187,766 | 5.58% | 0.26 (0.004) | 0.16 (0.012) | 0.41 (0.05) |
| Anorexia Nervosa | 9,104 | 3,647,894 | 0.25% | 723 | 198,137 | 0.36% | 0.24 (0.018) | 0.14 (0.074) | 0.41 (0.30) |
| ADHD | 18,698 | 1,047,880 | 1.75% | 4,274 | 51,762 | 7.63% | 0.48 (0.007) | 0.28 (0.018) | 0.80 (0.08) |
| Autism Spectrum Disorder | 9,347 | 1,057,231 | 0.88% | 1,114 | 54,922 | 1.99% | 0.43 (0.011) | 0.27 (0.036) | 0.64 (0.15) |
| Bipolar Disorder | 18,860 | 3,365,174 | 0.56% | 1,676 | 183,040 | 0.91% | 0.31 (0.009) | 0.18 (0.037) | 0.51 (0.15) |
| Major Depressive Disorder | 125,627 | 3,531,371 | 3.44% | 13,345 | 185,515 | 6.71% | 0.21 (0.003) | 0.14 (0.011) | 0.30 (0.05) |
| Obsessive Compulsive Disorder | 13,044 | 3,643,954 | 0.36% | 1,157 | 197,703 | 0.58% | 0.25 (0.013) | 0.15 (0.046) | 0.38 (0.19) |
| Schizophrenia | 15,203 | 3,368,831 | 0.45% | 1,111 | 183,605 | 0.60% | 0.33 (0.010) | 0.05 (0.058) | 0.58 (0.05) |

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Disorder**  | **Study**  | **N case/N control** | **Prevalence**  | **h2** |
| Alcohol Dependence | PGC AD 1 | 3,772/6,158 | 13,1% | 0.10 (0.05) |
| Anorexia Nervosa | PGC AN 1 | 3,495/10,982 | 0.9% | 0.20 (0.03) |
| ADHD | PGC + iPSYCH ADHD | 19,099/34,194 | 5% | 0.22 (0.01) |
| Autism Spectrum Disorder | PGC + iPSYCH ASD | 18,381/27,969 | 1% | 0.12 (0.01) |
| Bipolar Disorder | PGC BIP 2 | 20,352/31,358 | 1% | 0.21 (0.01) |
| Major Depressive Disorder | PGC MDD 2 | 16,823/25,632 | 10% | 0.12 (0.02) |
| Obsessive Compulsive Disorder  | PGC OCD/TS | 2,936/7,279 | 3% | 0.28 (0.04) |
| Schizophrenia | PGC2 + CLOZUK1 | 40,675/64,643 | 1% | 0.24 (0.01) |

**Supplement Table 3. Descriptives SNP-based studies: Samples, prevalences and estimated heritability (h2) including SE**

Note: PGC AD1= Substance Use Disorder Working Group of the PGC; PGC AN1= Eating Disorder Working Group of the PGC; PGC + iPSYCH ADHD= Attention Deficit/Hyperactivity Disorder Working Group of the iPSYCH-Broad-PGC Consortium; PGC + iPSYCH ASD= Autism Spectrum Disorder Working Group of the iPSYCH-Broad-PGC Consortium; PGC BIP2= Bipolar Disorder Working Group of the PGC; PGC MDD 2= Major Depressive Disorder Working Group of the PGC; PGC OCD/TS= Obsessive Compulsive Disorders and Tourette Syndrome Working Group of the PGC; CLOZUK= Schizophrenia CLOZUK

**STUDY COHORT DETAILS**

**ALCOHOL DEPENDENCE (AD)**

**SAMPLES**

All samples are part of the PGC Substance Use Disorder (PGC-SUD) Consortium.

Funding: PGC is funded by MH094421. We thank the National Institute of Drug Abuse (NIDA) for supporting us via an administrative supplement and the National Institute of Alcohol Abuse and Alcoholism (NIAAA) for support via the Collaborative Study on the Genetics of Alcoholism (AA008401). AA also acknowledges support from NIDA via DA032573.

**Gene-Environment-Development Initiative -GEDI – Duke University (GSMS)**

**Sample description**: The Duke arm of the NIDA-funded Gene-Environment-Development Initiative (GEDI) combined existing phenotypic and environmental data from two large prospective studies, the Great Smoky Mountains Study (GSMS) and the Caring for Children in the Community (CCC) study. For each of the two population-based contributing studies, genomewide genotyping was conducted using a common platform (Illumina Human660W-Quad v1), generating a total genotyped sample of ~1300 subjects. Further details of the GEDI-Duke sample are available in 1 and 2.

**Alcohol dependence measure**: Participants of both studies were assessed via structured interviewing using the Young Adult Psychiatric Assessment and its early life extension (i.e., YAPA and CAPA), yielding diagnoses and symptom scales for a wide range of substance use disorders (SUDs). Alcohol dependence was defined using DSM-IV criteria. For the purposes of these analyses, controls were defined as those who had a lifetime history of alcohol drinking but did not meet criteria for alcohol abuse or dependence. No other comorbid diagnoses were excluded.

**Acknowledgements:** This research was supported by the National Institute on Drug Abuse (U01DA024413, R01DA11301), the National Institute of Mental Health (R01MH063970, R01MH063671, R01MH048085, K01MH093731 and K23MH080230), NARSAD, and the William T. Grant Foundation. We are grateful to all the GSMS and CCC study participants who contributed to this work.

**German Study on the Genetics of Alcoholism (GESGA):**

**Sample description:** Patients were recruited from consecutive admissions to the psychiatry and addiction medicine departments of several German psychiatric hospitals participating in the German Addiction Research Network (for detailed description see 3 and 4). All patients were male and of self-reported German ancestry and fulfilled DSM-IV criteria for AD. Control subjects had been drawn from three population based cohort studies (KORA: <https://www.helmholtz-muenchen.de/kora>; popgen: <https://www.epidemiologie.uni-kiel.de/biobanking/biobank-popgen>; HNR: <https://www.uni-due.de/recall-studie>) in Germany and a Munich community sample. Control samples are mainly population based and can thus comprise alcohol dependent individuals.

**Alcohol dependence measure:** Alcohol dependence was assessed using DSM-IV criteria. Patients received a consensus diagnosis of two clinical psychiatrists and were assessed using one (dependent on recruiting center) of the following (semi-) structured interviews conducted by trained clinical staff members: Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA), Composite International Diagnostic Interview (CIDI) or Structured Clinical Interview for DSM-IV (SCID).

**Acknowledgements:** MR and MMN were supported by the German Federal Ministry of Education and Research (BMBF) through grants BMBF 01ZX1311A (to MR and MMN), and through grants 01ZX1314A (to MMN) and 01ZX1314G (to MR) within the e:Med research program.

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**Phenomics and Genomics Sample (PAGES)**

**Sample description:** Individuals in this study were recruited as part of a large schizophrenia case control sample from the Munich greater area and consisted of stable schizophrenia inpatients or outpatients and healthy volunteers. All participants were genetically unrelated, schizophrenia patients were of Caucasian, psychiatrically healthy volunteers of German descent. Candidates with a history of head injury or neurological diseases were excluded.

**Alcohol dependence measure**: Detailed medical and psychiatric histories were collected, including a clinical interview using the Structured Clinical Interview for DSM-IV (SCID), to evaluate lifetime Axis I and II diagnoses. Alcohol dependence was assessed using DSM-IV criteria. Controls were defined as never-having had any alcohol or having a lifetime history of alcohol without meeting the criteria for alcohol abuse or dependence. No other comorbid diagnoses were excluded.

**Acknowledgements**: None

**Christchurch Health and Development study (CHDS)**

**Sample description**: The Christchurch Health and Development study (CHDS)5 is a longitudinal study of a birth cohort of 1265 children collected in mid-1977 from urban Christchurch, New Zealand. Data  on social circumstances, health, development and wellbeing of the participants was obtained from the cohort at birth, 4 months, 1 year, annually to age 16 years, and at 18, 21, 25, 30, and 35 years. All study information was collected on the basis of signed consent from study participants and all information is fully confidential. All aspects of the study have been approved by the Canterbury (NZ) Ethics Committee.

**Alcohol dependence measure**: At ages 18, 21, 25, 30 and 35 years cohort members were questioned about their substance use behaviours and problems associated with substance use since the previous assessment (alcohol, tobacco, cannabis, other illicit drugs), using the relevant sections of the Composite International Diagnostic Interview (CIDI) to assess DSM-IV symptom criteria for substance use disorders. Using this information, lifetime alcohol dependence was classified on the basis of whether the participant met DSM criteria for alcohol dependence at any assessment up to age 35.

**Acknowledgements:** The Christchurch Health and Development Study has been supported by funding from the Health Research Council of New Zealand, the National Child Health Research Foundation (Cure Kids), the Canterbury Medical Research Foundation, the New Zealand Lottery Grants Board, the University of Otago, the Carney Centre for Pharmacogenomics, the James Hume Bequest Fund, US NIH grant MH077874, and NIDA grant ‘‘A developmental model of gene-environment interplay in SUDs’’ (R01DA024413) 2007–2012.

**Yale-Penn studies**

**Sample description**: Yale-Penn subjects were recruited in the eastern US, predominantly in Connecticut and Pennsylvania. They were administered the Semi-Structured Assessment for Drug Dependence and Alcoholism (SSADDA)6,7 to derive DSM-IV diagnoses of lifetime alcohol dependence (and other major psychiatric traits). The study received IRB approval from all participating institutions and written informed consent was obtained from all study participants. Additional information is available in the relevant GWAS publications, e.g.8

**Alcohol dependence measure**: DSM-IV diagnoses from the SSADDA.

**Acknowledgements**: Yale-Penn was supported by National Institutes of Health Grants RC2 DA028909, R01 DA12690, R01 DA12849, R01 DA18432, R01 AA11330, and R01 AA017535 and the Veterans Affairs Connecticut and Philadelphia Veterans Affairs Mental Illness Research, Educational, and Clinical Centers.

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**Collaborative Study on the Genetics of Alcoholism (COGA case control)**

**Sample description**: COGA is a multi-site study of alcohol dependent probands and their family members. Alcohol dependent probands were recruited from inpatient and outpatient facilities. Community probands and their family members were also recruited from a variety of sources. A subset of alcohol dependent cases and genetically unrelated controls were genotyped using the Illumina HumanMap 1M BeadChip. The sample used here included 847 alcohol dependent cases and 552 controls of European-American descent. Additional details are available in9.

**Alcohol dependence measure**: All participants were assessed using the Semi-Structured Assessment for the Genetics of Alcoholism10. Cases met criteria for a lifetime history of DSM-IV alcohol dependence. Controls reported a history of alcohol drinking, but did not meet criteria for alcohol dependence, abuse or harmful use, nor did they meet criteria for abuse/dependence on illicit drugs.

**Acknowledgements**: We continue to be inspired by our memories of Henri Begleiter and Theodore Reich, founding PI and Co-PI of COGA, and also owe a debt of gratitude to other past organizers of COGA, including Ting-Kai Li, currently a consultant with COGA, P. Michael Conneally, Raymond Crowe, and Wendy Reich, for their critical contributions. This national collaborative study is supported by NIH Grant U10AA008401 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA). Funding support for GWAS genotyping, which was performed at the Johns Hopkins University Center for Inherited Disease Research, was provided by the National Institute on Alcohol Abuse and Alcoholism, the NIH GEI (U01HG004438), and the NIH contract "High throughput genotyping for studying the genetic contributions to human disease" (HHSN268200782096C). The authors thank Kim Doheny and Elizabeth Pugh from CIDR and Justin Paschall from the NCBI dbGaP staff for valuable assistance with genotyping and quality control in developing the dataset available at dbGaP.

**Study of Addiction: Genetics and Environment (SAGE) &**

**Collaborative Genetic Study of Nicotine Dependence (COGEND)**

**Sample description:** Subjects were selected from three large, complementary studies: COGA11, Family Study of Cocaine Dependence (FSCD12), and Collaborative Genetic Study of Nicotine Dependence (COGEND13). COGA participants were assessed using the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA). FSCD and COGEND participants were assessed using polydiagnostic instruments closely based on the SSAGA. Genotyping was conducted using the Illumina Human1Mv1\_C BeadChips. Further details of the SAGE sample are available in14.

**Alcohol dependence measure**: Cases reported a lifetime history of DSM-IV alcohol dependence. Genetically unrelated control subjects reported alcohol drinking but had no significant alcohol-dependence symptoms and did not meet criteria for a diagnosis of illicit drug dependence.

**Acknowledgements**: Support for the Study of Addiction: Genetics and Environment (SAGE) was provided through the NIH Genes, Environment and Health Initiative [GEI; U01 HG004422; dbGaP study accession phs000092.v1.p1]. SAGE is one of the genome-wide association studies funded as part of the Gene Environment Association Studies (GENEVA) under GEI. Assistance with phenotype harmonization and genotype cleaning, as well as with general study coordination, was provided by the GENEVA Coordinating Center [U01 HG004446]. Assistance with data cleaning was provided by the National Center for Biotechnology Information. Support for collection of datasets and samples was provided by the Collaborative Study on the Genetics of Alcoholism [COGA; U10 AA008401], the Collaborative Genetic Study of Nicotine Dependence [COGEND; P01 CA089392], and the Family Study of Cocaine Dependence [FSCD; R01 DA013423, R01 DA019963]. Funding support for genotyping, which was performed at the Johns Hopkins University Center for Inherited Disease Research (CIDR), was provided by the NIH GEI [U01HG004438], the National Institute on Alcohol Abuse and Alcoholism, the National Institute on Drug Abuse, and the NIH contract "High throughput genotyping for studying the genetic contributions to human disease" [HHSN268200782096C].

**Disclosures**: Laura J. Bierut is listed as an inventor on Issued U.S. Patent 8,080,371,“Markers for Addiction” covering the use of certain SNPs in determining the diagnosis, prognosis, and treatment of addiction.

**Comorbidity and Trauma Study (CATS)**

**Sample description**: This study consisted of opioid dependent individuals aged 18 and older recruited from opioid substitution therapy clinics in the greater Sydney area and genetically unrelated individuals with little or no lifetime opioid misuse from neighborhoods in geographic proximity to these clinics. All subjects were of European-Australian descent. Additional details are available in15.

**Alcohol dependence measure**: All participants were assessed using the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA). Alcohol dependence was defined using DSM-IV criteria. For the purposes of these analyses, controls were defined as those who had a lifetime history of alcohol drinking but did not meet criteria for alcohol abuse or dependence. No other comorbid diagnoses were excluded.

**Acknowledgements**: Funding support for the CATS (dbGAP accession number: phs000277.v1.p1) was provided by the National Institute on Drug Abuse (R01 DA17305); GWAS genotyping services at the CIDR at The Johns Hopkins University were supported by the National Institutes of Health (contract N01-HG-65403).

**Center on Antisocial Drug Dependence (CADD)**

**Sample description**: GWAS participants were drawn from several primary studies described elsewhere16,17,18,19. The current sample of 1,901 unrelated adolescents was over-selected for adolescent BD, with half of the participants ascertained specifically from high-risk populations (i.e. recruited through substance abuse treatment, special schools, or involvement with the criminal justice system; see Supplement for additional criteria for clinical probands). CADD GWAS participants had an average age of 16.5 (SD = 1.4, range = 13.0–19.9), 28.9 % were female, and 37.3 % of participants reported non-Caucasian ancestry.

**Alcohol dependence measure**: Lifetime Alcohol Dependence was assessed with the CIDI-SAM and defined as meeting alcohol dependence at any wave for this longitudinal study.

**Acknowledgements**: The following grants supported data collection and analysis: DA011015, DA012845, DA021913, DA021905, DA032555, and DA035804.

**METHODS**

Quality Control (QC), imputation, and analysis were performed for each of the 11 studies in the alcohol dependence meta-analysis using ricopili (<https://github.com/Nealelab/ricopili>).

**QC:** Initial sample QC criteria included filters for call rate (< 0.98) and excess or depleted heterozygosity (|*F*| > 0.20). Samples were also checked for concordance between reported and genetically-inferred sex, except where insufficient chromosome X markers were available (GSMS, CHDS, and CADD).Variant QC criteria included filters for call rate (< 0.98), differential missingness between cases and controls (> 0.02), and departure from Hardy-Weinberg equilibrium (HWE) in cases (*p* < 1e-10) or controls (*p* < 1e-6). Additional filtering on minor allele frequency (MAF) was performed after imputation.Next, population outliers and cryptic relatedness were identified for exclusion. Relatedness coefficients ($\hat{π}$) and principal components analysis (PCA) were computed using a strictly defined subset of approximately independent autosomal SNPs (i.e. MAF > 0.05, HWE *p* > 1e-3, exclude MHC and chromosome 8 inversion regions, exclude strand ambiguous SNPs, and LD prune to *r2* < 0.20). Samples were filtered for cryptic relatedness $\hat{π}$ > 0.20 and PCA outliers and individuals with substantial non-European ancestry were removed. PCA with 1000 Genomes reference samples were compared to verify the selection of samples with European ancestry.Where necessary, additional study-specific QC criteria were also applied. To protect against potential effects of genotyping batch in Yale-Penn, variants were excluded if substantial differences in allele frequency in cases were observed between the two genotyping locations (*p* < 1e-4). Strand ambiguous SNPs, and SNPs that were invariant in data from either genotyping location were also excluded from the Yale-Penn data. For GESGA, variants were similarly excluded if allele frequency differences were observed across the 7 sample collection sites, conditional on phenotype (*p* < 1e-4).

**Imputation:** Prior to imputation, SNP locations were converted to genome build hg19 if required, and ricopili was used to verify alignment of each SNP’s chromosomal position to the 1000 Genomes phase 3 reference panel. SNPs were excluded from imputation if the reported alleles did not match the reference panel (without or without a strand flip), if the variant was strand ambiguous without a clear strand assignment from allele frequency, or if observed MAF differed substantially from the European allele frequency in 1000 Genomes.After alignment to the reference, samples were pre-phased using SHAPEIT. Imputation was then performed for each study using IMPUTE2 with the 1000 Genomes phase 3 reference panel.

**Association Analysis:** For each of the 11 studies of alcohol dependence, a GWAS for alcohol dependence status was performed using logistic regression with imputed dosages in PLINK2. The first five principal components were included as covariates within each study to control for population structure. Principal component covariates were computed using imputed best-guess genotypes filtered and LD pruned using the same criteria described in QC. Sex was also included as a covariate for all studies except GESGA (due to lack of female cases of alcohol dependence). Duplicated samples across studies were removed prior to analysis (*N*=16).The GWAS results from the 11 studies were then combined in an inverse-variance weighted meta-analysis using METAL. GWAS results were filtered for imputation INFO score (> 0.80), MAF (> 0.01), and MAF in cases (> 0.0025). Variants present in fewer than 7 of the 11 studies were also excluded. After filtering, the meta-analysis contains results for 8,524,330 variants with a sample size of *N*=11,424 (4,901 cases, 6,523 controls).

**Heritability Analysis:** LD score regression analysis was performed using HapMap3 SNPs and pre-computed LD scores from 1000 Genomes European reference samples (available from <https://data.broadinstitute.org/alkesgroup/LDSCORE/>)20. After filtering for MAF (> 0.05) and average imputation INFO score (> 0.90), and exclusion of structural variants and strand-ambiguous markers, 1,141,006 SNPs remained for LD score analysis. Conversion of $h\_{g}^{2}$estimates from observed to liability scale was performed assuming a population prevalence of 0.13 for alcohol dependence.

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**ANOREXIA NERVOSA (AN)**

**SAMPLES**

PGC-AN is a collaboration representing researchers and clinicians from around the world, founded with the goal of identifying the genetic risk factors involved in the etiology of anorexia nervosa (AN). The Freeze 1 sample (2016) comprises 3,495 individuals with AN and 10,982 controls from 12 separate cohorts.\* Cases met DSM-IV criteria for either lifetime AN (restricting or binge-purge subtype) or lifetime eating disorders ‘not otherwise specified’ AN-subtype (i.e., exhibiting the core features of AN)21. Detailed information on recruitment and case ascertainment can be found elsewhere22,23. Out of the 12 cohorts, the largest single contributor (1,031 cases and 3,627 controls post-QC) was the Children’s Hospital of Philadelphia/Price Foundation collection, and these samples were included in a previous AN GWAS publication22. The remaining 11 cohorts were wholly or in part from the Welcome Trust Case Control Consortium 3 (WTCCC3). The Welcome Trust Sanger Institute genotyped cases for all WTCCC3 AN samples as well as controls for two of the samples, and these cases and controls were included in a previous AN GWAS meta-analysis23. Two of the 11 included cohorts included matched controls. As the WTCCC3 did not fund genotyping of controls, ancestrally matched controls for 9 out of 11 remaining WTCCC3 AN samples were sourced from multiple independent and overlapping research groups, consortia (including PGC), and funding bodies.

**METHODS**

**QC:** Genotyping of cases and controls was performed using Illumina arrays (Illumina, Inc., San Diego, CA). QC was performed on each of the 12 individual datasets using the updated version of PLINK24. Exclusion criteria for SNP-level QC comprised: (1) missingness > 0.02; (2) minor allele frequency < 0.05; (3) differential missingness between cases and controls > 0.02; and (4) HWE cutoff of p < 1x10-6 for controls and p < 1x10-10 for cases. Exclusion criteria for sample-level QC comprised: (1) missingness > 0.02; (2) FHET > |0.2|; (3) failed sex check; and (4) $π̂$ > 0.2 for relatedness. Principal components analysis (PCA) was first performed within each dataset and then across all datasets using FastPCA25, as implemented in the PGC pipeline26.

**Imputation:** Imputation to the 1000 Genomes phase 127 reference was performed within the PGC pipeline using SHAPEIT28 for phasing and IMPUTE2 29 for imputation. Imputation was performed with a chunk size of 3Mb and using default parameters on the full set of 2,186 phased haplotypes (August 2012, 30,069,288 variants, release “v3.macGT1”).

**Analysis:** Analysis within datasets was performed using an additive model in PLINK24, with the first ten principal components as covariates. Fixed-effects meta-analysis across the 12 datasets was accomplished using METAL30, with inverse variance weighting. QC, imputation, and primary GWAS were performed following the Ricopili pipeline26 at the Broad Institute.

\*Note that the number of cohorts differs from the 15 cohorts presented in the original WTCCC3 publication by Boraska et al., 201423.The reasons for this difference are as follows. First, cases from the United States and Canada were combined and analysed with United States controls. QC indicated that this was appropriate. Second, Sweden had contributed 39 cases to the initial GWAS. This sample size was below the threshold for inclusion so that cohort was not included. Third, samples from Italy (North) were not included due to the inability to identify appropriate controls.

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**ATTENTION DEFICIT/HYPERACTIVITY DISORDER (ADHD)**

**SAMPLES**

The GWAS meta-analysis of ADHD results used in the SNP heritability analysis, include samples from the Danish iPSYCH initiative, and 10 samples from the Psychiatric Genomics Consortium (PGC). The iPSYCH ADHD sample is a nationwide population based case-cohort sample. Cases and controls were identified based on information in the Danish Psychiatric Central Research Register31 and controls were randomly selected from the same nationwide birth cohort and not diagnosed with ADHD. In order to obtain DNA for genotyping the identified cases and controls were linked to samples in the Danish Newborn Screening Biobank through the unique personal identification number, which is assigned to all live-born babies in Denmark.

The samples from the PGC were all of European Ancestry and consists of four trio samples and six case control samples. The four trio samples include: 1) The CHOP (Children’s Hospital of Philadelphia) ADHD trio sample (262 trios), which were recruited from pediatric and behavioral health clinics in the Philadelphia area32; 2) The IMAGE-I (International Multisite ADHD Genetics Project) trio samples33,34 (700 trios), collected from countries in and around Europe including Belgium, Germany, Ireland, the Netherlands, Spain, Switzerland, and the United Kingdom, and Israel; 3) the PUWMa (Pfizer-funded study from the University of California, Los Angeles (UCLA), Washington University, and Massachusetts General Hospital (MGH)) trio samples35 (563 trios), which were collected independently at those three sites using similar but slightly different methods; 4) The Toronto, Canadian ADHD trio sample36 (109 trios), which was drawn from an outpatient clinic in an urban pediatric hospital and included children who were referred for attention, learning and/or behavioral problems.

The six case control samples include:

1) The Barcelona sample37 (572 cases and 425 controls), which comprised ADHD cases recruited and evaluated at the Hospital Universitari Vall d’Hebron located in Barcelona (Spain). The control sample consisted of unrelated blood donors frequency-matched for gender with the ADHD cases and screened to exclude those with lifetime ADHD symptoms or diagnosis; 2) The Bergen, Norway sample38 (295 cases and 202 controls) consisted of patients recruited through a Norwegian national medical registry, as well as by psychologists and psychiatrists working at out-patient clinics. The controls were recruited through the Medical Birth Registry of Norway; 3) The Cardiff sample39 (721 cases, 5081 controls) consisted of cases recruited from community clinics in Cardiff, Wales, St. Andrews, Scotland and Dublin, Ireland. Controls were obtained from the Wellcome Trust Case Control Consortium–Phase 2; 4) The German (Würzburg) sample40 (487 cases and 1290 controls) comprised patients with ADHD recruited and phenotypically characterized in six psychiatric outpatient units for children and adolescents. The controls were drawn from three population based epidemiological studies: (a) the Heinz Nixdorf Recall (Risk Factors, Evaluation of Coronary Calcification, and Lifestyle) study 3, (b) PopGen, (c) KORA (Cooperative Health Research in the Region of Augsburg); 5) The IMAGE-II ADHD sample41 (624 cases, 1755 controls) included cases collected using similar but not identical methods as the IMAGE-I study. The controls were collected for an IRB approved GWAS of schizophrenia which have been described elsewhere 42; 6) The Yale-Penn sample (182 cases, 1315 controls) consists of small nuclear families and unrelated individuals, collected to study the genetics of substance dependence 84344. The case-control subjects were recruited from substance abuse treatment centers and through advertisements at the University of Connecticut Health Center, Yale University School of Medicine, the Medical University of South Carolina, the University of Pennsylvania, and McLean Hospital.

**METHODS**

Quality control, imputation and primary association analyses were done using the bioinformatic pipeline “Ricopili”26. The 10 PGC samples were processed separately and the iPSYCH sample was processed in 23 separate waves.

Stringent quality control was applied to each sample before imputation, following standard procedures for GWAS26. Imputing was done using SHAPEIT28 to estimate haplotypes and subsequently IMPUTE245 for imputing genotypes. Haplotypes the 1000 Genomes Project, phase 346,20 was used as reference data. Trio imputation was done with a case-pseudocontrol setup.

Related individuals and genetic outliers were identified based on a set of high quality markers (~30,000) pruned for linkage disequilibrium (LD). Related individuals were identified by an “identity by state analysis” using PLINK v1.924,47 and one individual was excluded in pairs of subjects with $\hat{π}$ > 0.2. Genetic outliers were identified and subsequently removed based on principal component analyses performed using smartPCA48. This was done separately for each of the PGC samples and on a merged set of genotypes for the iPSYCH sample.

Association analyses using dosage data were performed for the 10 PGC samples and the 23 waves in iPSYCH by an additive logistic regression model using PLINK v1.924,47 and relevant principal components as covariates. The results were meta-analysed using an inverse-weighted fixed effects mode implemented in the software METAL (version 2011-03-25)30. Only SNPs with imputation quality (INFO score) > 0.8 and maf > 0.01 were included. The meta-analysis included, in total 19,099 cases and 34,194 controls and 8,094,095 markers (only markers supported by an effective sample size (Neff = 2/(1/Ncases + 1/Ncontrols)49 greater than 70% were included).

**Heritability analysis**

SNP heritability was estimated using LD score regression50. Summary statistics from the ADHD GWAS meta-analysis and pre-computed LD scores for HapMap3 SNPs calculated based on 378-phased European-ancestry individuals from the 1000 Genomes Project were used in the analysis (LD scores available on <https://github.com/bulik/ldsc>). Only results for markers with an imputation INFO score > 0.90 were included in the analysis. After filtering and merging with the panel of high confidence HapMap3 SNPs 1,065,687 SNPs were included in the analysis. The SNP heritability for ADHD was calculated on the liability scale using a prevalence of ADHD of 5% in the population51.

**Acknowledgements**

The iPSYCH ADHD study was supported by The Lundbeck Foundation (grant no R102-A9118 and R155-2014-1724), Denmark; the Stanley Medical Research Institute; an Advanced Grant from the European Research Council (project no: 294838); the Stanley Center for Psychiatric Research at Broad Institute and Centre for Integrated Register-based Research at Aarhus University. This research has been conducted using the Danish National Biobank resource, supported by the Novo Nordisk Foundation.

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**AUTISM SPECTRUM DISORDER (ASD)**

**SAMPLES**

The ASD summary statistics are based on a meta-analysis of the results based on 5305 trios of European ancestry from the Autism Spectrum Disorder Working Group of the PGC Consortium and 13076 cases and 22664 controls of European ancestry from the iPSYCH autism sample.

A detailed description of the PGC sample is available on the PGC web site: <https://www.med.unc.edu/pgc/files/resultfiles/PGCASDEuro_Mar2015.readme.pdf>. Briefly, five cohorts provided genotypes (n denote the number of trios for which genotypes were available): The Geschwind Autism Center of Excellence (ACE; n = 391), the Autism Genome Project (AGP; n = 2272)52, the Autism Genetic Resource Exchange (AGRE; n = 974)53, the NIMH Repository (<https://www.nimhgenetics.org/available_data/autism/>), the Montreal/Boston Collection (MONBOS; n = 1396)54, and the Simons Simplex Collection (SSC; n = 2231)55. The trios were analyzed as cases and pseudo controls.

The iPSYCH ASD sample is a population based case-cohort sample extracted from the birth cohorts consisting of all children born in Denmark between May 1st 1981 and December 31st 2005. Eligible were singletons born to a known mother and resident in Denmark on their one-year birthday. Cases were defined from the Danish Psychiatric Central Research Register31 as those having an ASD diagnosis (ICD codes F84.0, F84.1, F84.5, F84.8 or F84.9) given no later than 2013. The controls constitute a random sample from the set of eligible children that did not have an ASD diagnosis by 2013. The samples were linked using the unique personal identification number to the Danish Newborn Screening Biobank. Genotypes are available on 14970 cases and 26125 controls.

**METHODS**

Data processing and QC was conducted according to the standards employed by the PGC Statistical Analysis Group and carried out using their pipeline Ricopili26. To minimize potential batch effects the data was processed separately in the 23 genotyping batches in the case of iPSYCH and for each cohort in the PGC sample. Phasing was achieved using SHAPEIT28 and imputation done by IMPUTE256 with haplotypes from the 1000 Genomes Project, phase 3 (1kGP3) as reference57. Trio samples were imputed as a case-pseudo-controls design.

Prior to principal component analysis (PCA), regions of high LD58 were excluded and genotypes were pruned down to a set of roughly 30k markers by pruning in a sliding window fashion using plink 1.947. Using Plink’s identity by state analysis, pairs of subjects were identified with $\hat{π}>0.2$ and one subject of each such pair excluded at random keeping cases when possible. PCA was carried out using smartPCA48. In iPSYCH a subsample of European ancestry was selected using a ellipsoid in the space of the first 3 principal components (PCs) centred and scaled using the mean and 8 standard deviation of the PCs restricted to the subsample whose parents and grandparents were all known to have been born in Denmark (n=31500). In the PGC sample a Euclidian distance measure weighted by the variance explain for each of the first 3 PCs. Individuals more distant than 10 standard deviations from the combined CEU and TSI HapMap reference populations were excluded. For both iPSYCH and PGC samples we conducted a secondary PCA to provide covariates for the association analyses.

Association analyses were done applying plink 1.9 to the imputed dosage data for each iPSYCH batch at a time and each PGC subcohort at a time. The results were subsequently meta analyzed using METAL30(July 2010 version) employing an inverse variance weighted fixed effect model59. Prior to release we filtered the summary statistics allowing only markers with an imputation info score $\geq 0.7$, maf $\geq 0.01$ and an effective sample size of at least 70% of the maximum. The effective sample size was estimated from the number of cases, Nca, and controls, Nco, contributing to the individual regression as 2\*Nca\*Nco/(Nca+Nco).

SNP heritability was estimated using LD score regression (LDSC)50. For LDSC we used precomputed LD scores based on the European ancestry samples of the 1000 Genomes Project57 restricted to HapMap3 SNPs60(downloaded from the <https://github.com/bulik/ldsc>). The summary stats with standard LDSC filtering were regressed onto these scores. For liability scale estimates, we used a population prevalence of 1.22%61.

**Acknowledgements:**

The iPSYCH ASD study was supported by The Lundbeck Foundation (grant no R102-A9118 and R155-2014-1724), Denmark; the Stanley Medical Research Institute; an Advanced Grant from the European Research Council (project no: 294838); the Stanley Center for Psychiatric Research at Broad Institute and Centre for Integrated Register-based Research at Aarhus University. This research has been conducted using the Danish National Biobank resource, supported by the Novo Nordisk Foundation.

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**BIPOLAR DISORDER (BIP)**

**SAMPLES**

This paper reports the results of genomic analyses on 20,352 bipolar disorder cases and 31,358 controls (51,710 subjects). All individual genotypes in the discovery GWAS were directly processed and analysed by the PGC-BIP2.

Case definitions

The sections below describe the bipolar disorder samples that were part of this report. We also describe the ascertainment procedures and diagnosis of the subjects comprising this report. As in our previous mega-analysis (PGC1)62, individuals with schizoaffective disorder bipolar type were included as cases since family history studies have shown coaggregation of these two disorders, diagnostic criteria separating them are subjective, and the inter-rater reliability is often low across research groups63,64.

The lead-PI of each sample warranted that their protocol was approved by their local Ethical Committee and that all subjects provided written informed consent.

All of these subjects are independent as confirmed using SNPs directly genotyped in all samples.

Most studies have been described in detail in the citations provided. The boldfaced first line for each sample is study PI, PubMed ID, country (study name), and the PGC internal tag or study identifier.

**Adolfsson, R | Not published | Umeå, Sweden | bip\_ume4\_eur**

Clinical characterization of the patients included the Mini-International Neuropsychiatric Interview (MINI65), the Diagnostic Interview for Genetic Studies (DIGS66), the Family Interview for Genetic Studies (FIGS67) and the Schedules for Clinical Assessment in Neuropsychiatry (SCAN68). The final diagnoses were made according to the DSM-IV-TR21 and determined by consensus of 2 research psychiatrists. The unrelated Swedish control individuals, consisting of a large population-based sample representative of the general population of the region, were randomly selected from the ‘Betula study’69.

**Alda, M; Smoller, J | Not published | Nova Scotia, Canada; I2B2 controls | bip\_hal2\_eur**

The case samples were recruited from patients longitudinally followed at specialty mood disorders clinics in Halifax and Ottawa (Canada). Cases were interviewed in a blind fashion with the Schedule of Affective Disorders and Schizophrenia-Lifetime version (SADS-L)70 and consensus diagnoses were made according to DSM-IV21 and Research Diagnostic Criteria (RDC)71. Protocols and procedures were approved by the local Ethics Committees and written informed consent was obtained from all patients before participation in the study. Control subjects were drawn from the I2B2 (Informatics for Integrating Biology and the Bedside) project 72. The study consists of de-identified healthy individuals recruited from a healthcare system in the Boston, MA, US area. The de-identification process meant that the Massachusetts General Hospital Institutional Review Board elected to waive the requirement of seeking informed consent as detailed by Code of Federal Regulations, Title 45, Part 46, Section 116 (46.116).

**Andreassen, O | 21926972 [PGC1] | Norway (TOP) | bip\_top7\_eur**

In the TOP study (Tematisk omrade psykoser), cases of European ancestry, born in Norway, were recruited from psychiatric hospitals in the Oslo region. Patients were diagnosed according to the SCID73 and further ascertainment details have been reported. Healthy control subjects were randomly selected from statistical records of persons from the same catchment area as the patient groups. The control subjects were screened by interview and with the Primary Care Evaluation of Mental Disorders (PRIME-MD)74. None of the control subjects had a history of moderate/severe head injury, neurological disorder, mental retardation or an age outside the age range of 18-60 years. Healthy subjects were excluded if they or any of their close relatives had a lifetime history of a severe psychiatric disorder. All participants provided written informed consent and the human subjects protocol was approved by the Norwegian Scientific-Ethical Committee and the Norwegian Data Protection Agency.

**Andreassen, O | Not published | Norway (TOP) | bip\_top8\_eur**

The TOP8 bipolar disorder cases and controls were ascertained in the same way as the bip\_top7\_eur (TOP7) samples described above.

**Biernacka, J | 27769005 | Mayo Clinic, USA | bip\_may1\_eur**

Bipolar cases were drawn from the Mayo Clinic Bipolar Biobank75. Enrolment sites included Mayo Clinic, Rochester, Minnesota; Lindner Center of HOPE/University of Cincinnati College of Medicine, Cincinnati, Ohio; and the University of Minnesota, Minneapolis, Minnesota. Enrolment at each site was approved by the local Institutional Review Board approval, and all participants consented to use of their data for future genetic studies. Participants were identified through routine clinical appointments, from in-patients admitted in mood disorder units, and recruitment advertising. Participants were required to be between 18 and 80 years old and be able to speak English, provide informed consent, and have DSM-IV-TR21 diagnostic confirmation of type I or II bipolar disorder or schizoaffective bipolar disorder as determined using the SCID73. Controls were selected from the Mayo Clinic Biobank76. Potential controls with ICD9 codes for bipolar disorder, schizophrenia or related diagnoses in their electronic medical record were excluded.

**Blackwood, D | 18711365 [PGC1] | Edinburgh, UK | bip\_edi1\_eur**

This sample comprised Caucasian individuals contacted through the inpatient and outpatient services of hospitals in South East Scotland. A BD-I diagnosis was based on an interview with the patient using the SADS-L70 supplemented by case note review and frequently by information from medical staff, relatives and care givers. Final diagnoses, based on DSM-IV21 criteria were reached by consensus between two trained psychiatrists. Ethnically-matched controls from the same region were recruited through the South of Scotland Blood Transfusion Service. Controls were not directly screened to exclude those with a personal or family history of psychiatric illness. The study was approved by the Multi-Centre Research Ethics Committee for Scotland and patients gave written informed consent for the collection of DNA samples for use in genetic studies.

**Corvin, A | 18711365 [PGC1] | Ireland | bip\_dubl\_eur**

Samples were collected as part of a larger study of the genetics of psychotic disorders in the Republic of Ireland, under protocols approved by the relevant IRBs and with written informed consent that permitted repository use. Cases were recruited from Hospitals and Community psychiatric facilities in Ireland by a psychiatrist or psychiatric nurse trained to use the SCID73. Diagnosis was based on the structured interview supplemented by case note review and collateral history where available. All diagnoses were reviewed by an independent reviewer. Controls were ascertained with informed consent from the Irish GeneBank and represented blood donors who met the same ethnicity criteria as cases. Controls were not specifically screened for psychiatric illness.

**Craddock, N | 17554300 | WTCCC | bip\_wtcc\_eur\_sr-qc**

Cases were all over the age of 16 years old, living in mainland UK and of European descent. Recruitment was undertaken throughout the UK and included individuals who had been in contact with mental health services and had a lifetime history of high mood. After providing written informed consent, participants were interviewed by a trained psychologist or psychiatrist using a semi-structured lifetime diagnostic psychiatric interview and available psychiatric medical records were reviewed. Using all available data, best-estimate life-time diagnoses were made according to the RDC71.   In the current study we included cases with a lifetime diagnosis of RDC bipolar I disorder, bipolar II disorder or schizo-affective disorder, bipolar type.

Controls were recruited from two sources: the 1958 Birth Cohort study and the UK Blood Service (blood donors) and were not screened for history of mental illness.

All cases and controls were recruited under protocols approved by the appropriate IRBs. All subjects gave written informed consent.

**Craddock, N; Jones, I; Jones, L | [ICCBD] | Cardiff, UK (ICCBD-BDRN) | bip\_icuk\_eur**

Cases were recruited via systematic and not systematic methods as part of the Bipolar Disorder Research Network project ([www.bdrn.org](http://www.bdrn.org)), provided written informed consent and were interviewed using a semi-structured diagnostic interview, the Schedules for Clinical Assessment in Neuropsychiatry, a life chart and detailed information about family history of psychiatric disorders. Based on the information gathered from the interview, case notes review and questionnaires, best-lifetime diagnosis was made according to DSM-IV21. Inter-rater reliability was formally assessed using 20 randomly selected cases (mean ĸ Statistic = 0.85). In the current study we included cases with a lifetime diagnosis of DSM-IV bipolar disorder or schizo-affective disorder, bipolar type. The BDRN study received approval from the Multi-Region and Local Research Ethics Committee in the United Kingdom.

Controls were part of the Wellcome Trust Case Control Consortium common control set, which comprised healthy blood donors recruited from the UK Blood Service and samples from the 1958 British Birth Cohort. Controls were not screened for a history of mental illness.

All cases and controls were recruited under protocols approved by the appropriate IRBs. All subjects gave written informed consent.

**Hauser, J; Lissowska, J; Forstner, AJ | 24618891 | BOMA-Poland | bip\_bmpo\_eur**

Cases were recruited at the Department of Psychiatry, Poznan University of Medical Sciences, Poznan, Poland. All cases received a lifetime diagnosis of BD according to the DSM-IV21 criteria on the basis of a consensus best-estimate procedure77 and structured diagnostic interviews using the SCID73. Controls were drawn from a population-based case-control sample recruited by the Cancer-Center and Institute of Oncology, Warsaw, Poland and a hospital-based case-control sample recruited by the Nofer Institute of Occupational Medicine, Lodz, Poland. The Polish controls were produced by the International Agency for Research on Cancer (IARC) and the Centre National de Génotypage (CNG) GWAS Initiative for a study of upper aerodigestive tract cancers 78. The controls were not screened for a history of mental illness. Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Fullerton, J.M.; Mitchell, P.B.; Schofield, P.R.; Martin N.G. | 24618891 | BOMA-Australia | bip\_bmau\_eur**

Cases were recruited at the Mood Disorder Unit, Prince of Wales Hospital in Sydney. All cases received a lifetime diagnosis of BD according to the DSM-IV21 criteria on the basis of a consensus best-estimate procedure77 and structured diagnostic interviews using the DIGS66, FIGS67, and the SCID73. Controls were parents of unselected adolescent twins from the Brisbane Longitudinal Twin Study79. The controls were not screened for a history of mental illness. Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Grigoroiu-Serbanescu, M; Nöthen, MM | 21353194 | BOMA-Romania | bip\_rom3\_eur**

Cases were recruited from consecutive admissions to the Obregia Clinical Psychiatric Hospital, Bucharest. Patients were administered the DIGS66 and FIGS67 interviews. Information was also obtained from medical records and close relatives. The diagnosis of BP-I was assigned according to DSM-IV21 criteria using the best estimate procedure77. All patients had at least two hospitalized illness episodes. Population-based controls were evaluated using the DIGS66 to exclude a lifetime history of major affective disorders, schizophrenia, schizoaffective disorders, and other psychoses, obsessive-compulsive disorder, eating disorders, and alcohol or drug addiction.

**Kelsoe, J | 21926972 [PGC1] | USA (GAIN) | bip\_gain\_eur**

*Genetic Association Information Network (GAIN)/ The Bipolar Genome Study (BiGS)* The BD sample was collected under the auspices of the NIMH Genetics Initiative for BD (<http://zork.wustl.edu/nimh/>), genotyped as part of GAIN and analyzed as part of a larger GWAS conducted by the BiGS consortium. Approximately half of the GAIN sample was collected as multiplex families or sib pair families (waves 1-4), the remainder were collected as individual cases (wave 5). Subjects were ascertained at 11 sites: Indiana University, John Hopkins University, the NIMH Intramural Research Program, Washington University at St. Louis, University of Pennsylvania, University of Chicago, Rush Medical School, University of Iowa, University of California, San Diego, University of California, San Francisco, and University of Michigan. All investigations were carried out after the review of protocols by the IRB at each participating institution. At all sites, potential cases were identified from screening admissions to local treatment facilities and through publicity programs or advocacy groups. Potential cases were evaluated using the DIGS66, FIGS67, and information from relatives and medical records. All information was reviewed through a best estimate diagnostic procedure by two independent and non-interviewing clinicians and a consensus best-estimate diagnosis was reached. In the event of a disagreement, a third review was done to break the tie. Controls were from the NIMH Genetic Repository sample obtained by Dr. P. Gejman through a contract to Knowledge Networks, Inc. Only individuals with complete or near-complete psychiatric questionnaire data who did not fulfill diagnostic criteria for major depression and denied a history of psychosis or BD were included as controls for BiGS analyses. Controls were matched for gender and ethnicity to the cases.

**Kelsoe, J; Sklar, P; Smoller, J | [PGC1 Replication] | USA (FAT2; FaST,** **BiGS, TGEN) | bip\_fat2\_eur**

Cases were collected from individuals at the 11 U.S. sites described for the GAIN sample. Eligible participants were age 18 or older meeting DSM-IV criteria for BD-I or BD-II by consensus diagnosis based on interviews with the Affective Disorders Evaluation (ADE)80 and MINI65. All participants provided written informed consent and the study protocol was approved by IRBs at each site. Collection of phenotypic data and DNA samples were supported by NIMH grants MH063445 (JW Smoller); MH067288 (PI: P Sklar), and MH63420 (PI: V Nimgaonkar). The control samples were NIMH controls that were using the methods described in that section. The case and control samples were independent of those included in the GAIN sample.

**Kirov, G | 25055870 | Bulgarian trios | bip\_butr\_eur**

All cases were recruited in Bulgaria from psychiatric inpatient and outpatient services. Each proband had a history of hospitalisation and was interviewed with an abbreviated version of the SCAN68. Consensus best-estimate diagnoses were made according to DSM-IV21 criteria by two researchers. All participants gave written informed consent and the study was approved by local ethics committees at the participating centers.

**Kirov, G | 25055870 | UK trios | bip\_uktr\_eur**

The BD subjects were recruited and interviewed in person by a senior psychiatrist, using abbreviated version of the SCAN68. Consensus best-estimate diagnoses were made based on the interview and hospital notes. Ethics committee approval for the study was obtained from the relevant research ethics committees and all individuals provided written informed consent for participation.

**Landen, M | [ICCBD] | Sweden (ICCBD) | bip\_swa2\_eur**

The BD subjects were identified using the Swedish Hospital Discharge Register

including subjects with at least two hospitalizations with a BD diagnosis and confirmatory diagnostic review in a subset of subjects. Additional subjects were recruited from the Affective Center at St Goran Hospital in Stockholm, Sweden, following physician's referral for BD. The diagnostic instrument used was a Swedish adaptation of the ADE80 which includes the affective module of the SCID73. Further BD cases were recruited from the Stockholm County catchment area. Diagnoses were made according to the DSM-IV criteria, and cases were not reported previously. The control subjects used were the same as for the SCZ analyses described above. All ascertainment procedures were approved by the Regional Ethical Committees in Sweden.

**Landen, M | [ICCBD] | Sweden (ICCBD) | bip\_swei\_eur**

The cases and controls in the bip\_swei\_eur sample were recruited using the same ascertainment methods described for the bip\_swa2\_eur sample.

**Leboyer, M | [PGC1 replication] | France | bip\_fran\_eur**

Cases with BD-I or BD-II and control samples were recruited as part of a large study of genetics of BD in France (Paris-Creteil, Bordeaux, Nancy) with a protocol approved by relevant IRBs and with written informed consent. Cases were of French descent for more than 3 generations were assessed by a trained psychiatrist or psychologist with the DIGS66 and the FIGS67. Diagnoses were based on structured interviews supplemented by medical case notes, mood scales and self-rating questionnaire assessing dimensions.

**Li, Q | 24166486; 27769005** **| USA (Janssen), SAGE controls | bip\_jst5\_eur**

The study included unrelated patients with bipolar I disorder from 6 clinical trials (IDs: NCT00253162, NCT00257075, NCT00076115, NCT00299715, NCT00309699, and NCT00309686)81,82,83,84,85,86. Participant recruitment was conducted by Janssen Research & Development, LLC (formerly known as Johnson & Johnson Pharmaceutical Research & Development, LLC) to assess the efficacy and safety of risperidone. Bipolar cases were diagnosed according to DSM-IV-TR 21 criteria. The diagnosis of bipolar disorder was confirmed by the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL)87 in NCT00076115, by the SCID73 in NCT00257075 and NCT00253162, or by the MINI65 in NCT00299715 and NCT00309699, and NCT00309686, respectively. Additional detailed descriptions of these clinical trials can be found at ClinicalTrials.gov. Only patients of European ancestry with matching controls were included in the current analysis. Controls subjects were drawn from the Study of Addiction: Genetics and Environment (SAGE, dbGaP Study Accession: phs000092.v1.p1). Control subjects did not have alcohol dependence or drug dependence diagnoses; however, mood disorders were not an exclusion criterion14.

**McQuillin, A; Gurling, H | 18317468 [PGC1] | UCL (University College London), London, UK | bip\_uclo\_eur**

The UCL sample comprised Caucasian individuals who were ascertained and received clinical diagnoses of bipolar disorder according to UK National Health Service (NHS) psychiatrists at interview using the categories of the International Classification of Disease version 10. In addition bipolar subjects were included only if both parents were of English, Irish, Welsh or Scottish descent and if three out of four grandparents were of the same descent. All volunteers read an information sheet approved by the Metropolitan Medical Research Ethics Committee who also approved the project for all NHS hospitals. Written informed consent was obtained from each volunteer. The UCL control subjects were recruited from London branches of the National Blood Service, from local NHS family doctor clinics and from university student volunteers. All control subjects were interviewed with the SADS-L70 to exclude all psychiatric disorders.

**Ophoff, RA | Not Published | Netherlands | bip\_ucla\_eur**

The case sample consisted of inpatients and outpatients recruited through psychiatric hospitals and institutions throughout the Netherlands88. Cases with DSM-IV21 bipolar disorder were included in the analysis. Controls were collected in parallel at different sites in the Netherlands and were volunteers with no psychiatric history. Ethical approval was provided by UCLA and local ethics committees and all participants gave written informed consent.

**Paciga, S | [PGC1] | USA (Pfizer) | bip\_pf1e\_eur**

This sample comprised Caucasian individuals recruited into one of three Geodon (ziprasidone) clinical trials (NCT00141271, NCT00282464, NCT00483548). Subjects were diagnosed by a clinician with a primary diagnosis of Bipolar I Disorder, most recent episode depressed, with or without rapid cycling, without psychotic features, as defined in the DSM-IV-TR21 (296.5x) and confirmed by the MINI65 (version 5.0.0).  Subjects also were assessed as having a HAM-D-17 total score of >20 at the screening visit.  The trials were conducted in accordance with the protocols, International Conference on Harmonization of Good Clinical Practice Guidelines, and applicable local regulatory requirements and laws.  Patients gave written informed consent for the collection of blood samples for DNA for use in genetic studies.

**Pato, C | [ICCBD] | Los Angeles, USA (ICCBD-GPC)| bip\_usc2\_eur**

Genomic Psychiatry Consortium (GPC) cases and controls were collected via the University of Southern California healthcare system, as previously described89. Using a combination of focused, direct interviews and data extraction from medical records, diagnoses were established using the OPCRIT and were based on DSM-IV-TR criteria21. Age and gender-matched controls were ascertained from the University of Southern California health system and assessed using a validated screening instrument and medical records.

**Reif, A; Nöthen, MM; Forstner, AJ | 24618891 | BOMA-Germany II | bip\_bmg2\_eur**

Cases were recruited from consecutive admissions to psychiatric in-patient units at the University Hospital Würzburg. All cases received a lifetime diagnosis of BD according to the DSM-IV21 criteria using a consensus best-estimate procedure77 based on all available information, including semi-structured diagnostic interviews using the Association for Methodology and Documentation in Psychiatry (AMDP)90, medical records and the family history method. In addition, the OPCRIT91 system was used for the detailed polydiagnostic documentation of symptoms.

Control subjects were unaffected subjects were drawn from the population-based Heinz Nixdorf Recall (HNR) Study92. The controls were not screened for a history of mental illness. Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Rietschel, M; Nöthen, MM, Cichon, S | 21926972 [PGC1] | BOMA-Germany I | bip\_bonn\_eur**

Cases for the BOMA-Bipolar Study were ascertained from consecutive admissions to the inpatient units of the Department of Psychiatry and Psychotherapy at the University of Bonn and at the Central Institute for Mental Health in Mannheim, University of Heidelberg, Germany. DSM-IV lifetime diagnoses of bipolar I disorder were assigned using a consensus best-estimate procedure, based on all available information, including a structured interview with the SCID73 and SADS-L70, medical records, and the family history method. In addition, the OPCRIT91 checklist was used for the detailed polydiagnostic documentation of symptoms. Controls were ascertained from three population-based studies in Germany (PopGen, KORA, and Heinz-Nixdorf-Recall Study). Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Rietschel, M; Nöthen, MM; Rivas, F; Mayoral, F; Kogevinas, M; others | 24618891 | BOMA-Spain | bip\_bmsp\_eur**

Cases were recruited at the mental health departments of the following five centers in Andalusia, Spain: University Hospital Reina Sofia of Córdoba, Provincial Hospital of Jaen; Hospital of Jerez de la Frontera (Cádiz); Hospital of Puerto Real (Cádiz); Hospital Punta Europa of Algeciras (Cádiz); and Hospital Universitario San Cecilio (Granada). Diagnostic assessment was performed using the SADS-L70; the OPCRIT91; a review of medical records; and interviews with first and/or second degree family members using the Family Informant Schedule and Criteria (FISC) . Consensus best estimate BD diagnoses were assigned by two or more independent senior psychiatrists and/or psychologists, and according to the RDC71, and the DSM-IV21. Controls were Spanish subjects drawn from a cohort of individuals recruited in the framework of the European Community Respiratory Health Survey (ECRHS, http://www.ecrhs.org/). The controls were not screened for a history of mental illness. Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Rietschel, M; Nöthen, MM; Schulze, TG; Bauer, M; Forstner, AJ; Müller-Myhsok, B | 24618891 | BOMA-Germany III | bip\_bmg3\_eur**

Cases were recruited at the Central Institute of Mental Health in Mannheim, University of Heidelberg, and other collaborating psychiatric hospitals in Germany. All cases received a lifetime diagnosis of BD according to the DSM-IV21 criteria using a consensus best-estimate procedure77 based on all available information including structured diagnostic interviews using the AMDP90, Composite International Diagnostic Screener (CID-S)93, SADS-L70 and/or SCID73, medical records, and the family history method. In addition, the OPCRIT91 system was used for the detailed polydiagnostic documentation of symptoms.

Controls were selected randomly from a Munich-based community sample and recruited at the Max-Planck Institute of Psychiatry. They were screened for the presence of anxiety and mood disorders using the CID-S93. Only individuals without mood and anxiety disorders were collected as controls. Study protocols were reviewed and approved in advance by Institutional Review Boards of the participating institutions. All subjects provided written informed consent.

**Scott, L | [PGC1] | Michigan, USA (Pritzker and NIMH) | bip\_mich\_eur**

The Pritzker Neuropsychiatric Disorders Research Consortium (NIMH/Pritzker) case and controls samples were from the NIMH Genetics Initiative Genetics Initiative Repository. Cases were diagnosed according to DMS-III or DSM-IV criteria using the DIGS66 or FIGS67 and/or medical record review. Cases with low confidence diagnoses were excluded. From each wave 1-5 available non-Ashkenazi European-origin family, two BD-I siblings were included when possible and the proband was preferentially included if available (n=946 individuals in 473 sibling pairs); otherwise a single BD-I case was included (n=184). The bipolar sibling pairs were retained within the NIMH/Pritzker sample when individuals in more than one study were uniquely assigned to a study set. Controls had non-Ashkenazi European-origin, were aged 20-70 years and reported no diagnosis with or treatment for BD or schizophrenia, and that they had not heard voices that others could not hear. Individuals with suspected major depression were excluded based on answers to questions related to depressive mood. NIMH controls were further selected as the best match(es) to NIMH cases based on self-reported ancestry in the DIGS66.

**Sklar, P; Smoller, J | 18317468 [PGC1] | USA (STEP1) | bip\_stp1\_eur**

The Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) was a seven-site, national U.S., longitudinal cohort study designed to examine the effectiveness of treatments and their impact on the course of BD that enrolled 4,361 participants who met DSM-IV criteria for BD-I, BD-II, bipolar not otherwise specified (NOS), schizoaffective manic or bipolar type, or cyclothymic disorder based on diagnostic interviews. From the parent study, 2,089 individuals who were over 18 years of age with BD-I and BD-II diagnoses consented to the collection of blood samples for DNA. BD samples with a consensus diagnosis of BD-I on both the ADE80 and MINI65 were selected for inclusion in STEP1. Two groups of controls samples from the NIMH repository were used. One comprised DNA samples derived from US Caucasian anonymous cord blood donors. The second were controls who completed the online self-administered psychiatric screen and were ascertained as described above, by Knowledge Networks Inc. For the second sample of controls only those without history of schizophrenia, psychosis, BD or major depression with functional impairment were used.

**Sklar, P; Smoller, J | 18711365 [PGC1] | USA (STEP2) | bip\_stp2\_eur**

The STEP2 sample included BD-1 and BD-2 samples from the STEP-BD study described above along with BD-2 subjects from UCL study also described above. The controls samples for this study were from the NIMH repository as described above for the STEP1 study.

**METHODS**

Individual genotype data for all samples were processed using the PGC “ricopili” pipeline (URLs) for standardized quality control, imputation, and analysis26.

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**MAJOR DEPRESSIVE DISORDER (MDD)**

**SAMPLES**

Reference94 and PGC MDD (submitted) have full details for the MDD samples. Briefly, this analysis is based on a GWAS mega-analysis of 29 samples of European-ancestry totaling 16,823 MDD cases and 25,632 controls including all samples from the prior PGC MDD report94. The majority of these samples passed a structured methodological review by MDD assessment experts (DL and KSK). Cases were required to meet international consensus criteria95,96,21 for a lifetime diagnosis of MDD established using structured diagnostic instruments from assessments by trained interviewers, clinician-administered checklists, or medical record review. Most cases were clinically ascertained, and most controls were randomly selected from the population and screened for the absence of lifetime MDD.

**METHODS**

Individual genotype data for all samples were processed using the PGC “ricopili” pipeline (URLs) for standardized quality control, imputation, and analysis26.

**Acknowledgements**

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**OBESSIVE COMPULSIVE DISORDER (OCD)**

**SAMPLES**

All samples are part of the International OCD Foundation Genomics Consortium (IOCDF-GC) or the Obsessive Compulsive Disorder Collaborative Genetics Association Study (OCGAS).

**IOCDF-GC:** Cases included 1,817 individuals and 663 trio probands diagnosed with OCD according to DSM-IV21 criteria and recruited predominantly from OCD specialty clinics. Screened controls were recruited from Bonn, Germany and unscreened controls from Capetown, South Africa. For study inclusion, all cases and trio probands were required to have a DSM-IV diagnosis of OCD. The controls from Bonn had an absent lifetime history of all axis I disorders and the South African controls were diagnostically unscreened. This work was approved by the relevant IRBs at all participating sites, and all participants provided written informed consent. Additionally, 5,654 unscreened controls, genotyped on two different Illumina SNP arrays, came from: 1) the Study of Addiction: Genes and Environment (SAGE) cohort (1,288 individuals)13,97; 2) the HYPERGENES Consortium Milan, Italy (501 individuals); 3) the Illumina ‘iControl’ Genotype Control Database (3,212 individuals,); and 4) a cohort of Dutch ancestry (653 individuals)12.

**OCGAS:** A total of 1 065 families were included in the OCGAS study (comprising 1,406 patients with OCD and 2,895 individuals in total). The sample comprised of 460 complete trios (including an affected proband and both parents); 155 pedigrees with a proband and an unaffected sibling and 450 families with another structure (complex family structure). An additional 192 probands without an additional family member present in the study (singletons) were included. For study inclusion, probands were required to meet DSM-IV criteria for OCD21 with onset of obsessions and/or compulsions before the age of 18 years (mean = 9.4 years; SD=6.35). Each case was evaluated by a PhD-level clinical psychologist, using the Structured Clinical Interview for DSM-IV (SCID), modified and extended to include additional symptom and diagnostic information. Final diagnostic status was assigned based on the consensus of two psychiatrists or psychologists reviewing the case independently. To increase the power of the study to detect significant association, we also included 1 1194 screened controls from the Genomic Psychiatry Cohort (GPC). Individuals with a self-reported or diagnosed neuropsychiatric disorder at the time of enrollment were excluded from the present study.

**METHODS**

**Genotyping and QC (IOCDFGC):** Subjects were genotyped on a combination of platforms including the Illumina Human610-Quadv1\_B SNP array, the Illumina Hap1M, and the Illumina Hap550k\_v1. Platform-specific QC includes removing SNPs and samples with low call rate (<98%), samples with ambiguous genomic sex or discordance between genomic and phenotypic sex, monomorphic SNPs and copy number variation targeted SNPs, SNPs with MAF<0.01, and strand-ambiguous SNPs or SNPs with allele frequency significantly different from HapMap CEU reference data. The batch effect was investigated on the samples genotyped at CGA, and no evidence for batch effect was found. Three SNPs with p<10-5 in the batch effect regression analysis were flagged, and none of these appeared in the top 580 SNPs in the case-control meta-analysis or in the top 584 SNPs in the final case-control and trio meta-analysis. Any SNPs detected with low concordance rate among different platforms were removed from OCD GWAS dataset. At this stage in the QC process, all samples were merged into a single dataset using PLINK. Platforms were merged after platform-specific cleaning was performed and 23 SNPs were either mismatched or tri-allelic and were removed. SNP allele frequencies in controls were compared among each platform and any SNP with an absolute allele frequency difference >0.15 between two platforms were flagged. SNPs with difference of missing rate in cases versus controls >0.02 were excluded. SNPs with Mendelian error rate >1% among 400 complete trios (IOTR) were removed. Multidimensional scaling (MDS) analyses were performed on cases, controls, and trio probands, and non-European descent samples and trios and population outliers were removed. Remaining European descent cases and controls were separated into three strata (IOEU for European, IOAJ for Ashkenazi Jewish, and IOSA for South African) based on observed genetic ancestry and source population. Within each of the subpopulations, samples with extreme inbreeding coefficient |F|>0.05 were removed. SNPs with Hardy Weinberg Equilibrium (HWE) p<10-10 in controls or p<10-6 in cases were removed, and those with HWE p<10-5 in controls were flagged. Lastly, any SNP not in common between the cleaned Hap1M, Hap610 and Hap550 platforms were removed, leaving 463,924, 456,734, 548,732, and 557,624 cleaned SNPs in IOEU, IOAJ, IOSA, and IOTR subpopulation, respectively, for subsequent analyses.

**Genotyping and QC (OCGAS):** Genotyping of recruited samples was performed at the Johns Hopkins SNP Center using Illumina’s HumanOmniExpress bead chips (Illumina Inc., San Diego, CA, USA). GPC controls were genotyped at USC using Illumina HumanOmniExpress 12 v 1.0 bead chips. Pedigree samples were converted to independent trios and unrelated cases. The GPC controls were used to match the cases based on MDS analysis. Quality control was applied in trios (OCTR) and case/control cohort (OCCC) separately, including removing SNPs and samples with call rate <98%, samples with ambiguous genomic sex or discordance between genomic and phenotypic sex, samples with discordance between genetic relationship and relationship on the clinical record, monomorphic SNPs and copy number variation targeted SNPs, strand-ambiguous SNPs, and SNPs with MAF<0.01. SNPs with HWE p<10-10 in controls or p<10-6 in cases were removed. MDS was carried out on case/control cohort and trio probands, and non-European ancestry samples and population outliers were removed. In case/control cohort, SNPs with difference of missing rate in cases versus controls >0.02 were excluded, leaving 545,630 cleaned SNPs for subsequent analyses. In trios, samples with Mendelian errors >10,000 and SNPs with Mendelian errors >4 were removed, leaving 581,862 cleaned SNPs for subsequent analyses.

Genotyped data of IOCDF-GC, OCGAS, and GPC samples were merged into one data set, SNPs with different strand orientation were flipped using 1000 Genomes Project (Phase I integrated variant set release; NCBI build 37 (hg19) as the reference. Pair-wise identity-by-descent (IBD) matrix was generated on all samples using linkage disequilibrium pruned (r2<0.15) SNPs, and overlapped or related samples (pi\_hat>0.2) between any two data sets were identified, and one of each duplicate/relative pairs were removed so that only independent samples and trios were kept for GWAS and meta-analysis. The final analysis dataset comprise of 1,429 cases, 5089 controls, and 285 trios from IOCDF-GC, 344 cases and 630 trios from OCGAS, and 1033 GPC samples matching for OCGAS cases.

**GWAS and meta-analysis:** GWAS genotype data from the IOCDF-GC (except the Dutch samples, which were imputed separately, see below), OCGAS and GPC samples using IMPUTE256 and reference haplotypes from the 1000 Genomes Project (Phase I integrated variant set release; NCBI build 37 (hg19) were constructed with SHAPEIT228. SNPs were excluded if IMPUTE2 info was <0.6, IMPUTE2 certainty was <0.9, or MAF<0.01. Separate genome-wide association analyses were conducted for each case-control subpopulation (IOCDF-GC European (IOEU), IOCDF-GC Ashkenazi Jewish (IOAJ), IOCDF-GC South African (IOSA), OCGAS case-control (OCCC)) and trio sample (IOCDF-GC trios (IOTR) and OCGAS trios (OCTR); as probands versus pseudo-controls) using PLINK to perform logistic association correcting for the top 10 principal components. Due to more stringent data sharing restrictions for Dutch cases, imputation and GWAS for the Dutch cases and population-matched controls (IODU) were calculated separately by the site investigators, following the same imputation and quality control procedures. An inverse variance meta-analysis was then performed using the summary statistics of all case-control subpopulations (including IODU) and trio samples using the software package METAL30.

**Heritability Analysis:** The LD score regression (LDSC) method50 was applied to 1,159,580 imputed and directly genotyped SNPs included in the OCD meta-analysis that also overlapped with a panel of high confidence HapMap3 SNPs. Regression weights were calculated using the HapMap European reference sample provided by Bulik-Sullivan and colleagues. Heritability was then calculated and the datasetchecked for residual population stratification (based on the LDSC intercept), followed by calculation of the genetic correlation between the two consortium sample collections. All 10,215 (2,936 cases and 7,279 controls) individuals in the GWAS meta-analysis comprised the sample for heritability estimation and the test for population stratification. To transform from the observed heritability scale to the liability scale, a population prevalence of 2.5% was used.

**Acknowledgements**

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**SCHIZOPHRENIA (SZC)**

**SAMPLES**

Case sample:

Samples from treatment-resistant schizophrenia (TRS) patients were collected in the UK through the blood-monitoring system used by consumers of the antipsychotic clozapine. The CLOZUK1 sample was initially assembled in collaboration with Novartis (Basel, Switzerland). The company, through their proprietary Clozaril® Patient Monitoring Service (CPMS), provided whole-blood samples and anonymised phenotypic information for 6,882 clozapine-takers. In parallel, the CLOZUK2 sample was assembled in collaboration with Leyden Delta (Nijmegen, Netherlands). The company, through their proprietary Zaponex® Treatment Access System (ZTAS), provided whole-blood samples and anonymised phenotypic information for 7,417 clozapine-takers. Both Clozaril® and Zaponex® are bioequivalent brands of clozapine licensed for marketing in the UK98. As both samples are intrinsically related, the term “CLOZUK” will be used throughout this manuscript to describe them together. As stated in guidelines by the UK National Institute for Clinical Excellence (NICE), prescription of clozapine is only initiated in schizophrenia cases after therapeutic failure of two trials of common antipsychotics. This strict criteria ensures that patients sampled for CLOZUK conform to a standardised description of TRS99, consistent to that used in other initiatives to elucidate its genetic architecture 100101102.

Additionally, a cohort of patients with schizophrenia recruited in the UK (CardiffCOGS) was also included as part of the study. This cohort it’s a conventional sample of those with schizophrenia recruited via secondary care, mainly outpatient, mental health services in Wales and England. Thus, it includes patients that were not taking clozapine at the moment of their recruitment, and do not conform to a TRS classification. However, the recruitment procedure assured that all of its members have been specifically screened for neuropsychiatric disorders using standardized approaches and case-note reviews, as previously reported103.

Genotyping for the cases was performed by the Broad Institute (Massachusetts, USA) for the CLOZUK1 sample and 512 CardiffCOGS cases, using OmniExpress-12 and OmniExpressExome-8 chips as described elsewhere103. The remaining 247 CardiffCOGS cases and the CLOZUK2 sample were genotyped by deCODE Genetics (Reykjavík, Iceland), using OmniExpress-12 chips.

Control sample:

Control samples were collected from publicly available sources (EGA) or through collaboration with the dataset holders. Individual datasets were curated with the same specifications as the case-only datasets. In order to maximize the numbers of individuals that could be effectively included in the GWAS without introducing confounders, these datasets were chosen in the basis of having recruited individuals with self-reported British ancestry (either exclusively or primarily) and having been genotyped on Illumina chips.

**METHODS**

**QC:** Given the many data sources used and the variety of genotyping chips available, a stringent quality control (allowing only 2% of missing SNP and individual data) was performed separately in each individual dataset, using PLINK v1.947 and following standard procedures104. To facilitate merging and to avoid common sources of batch effects105, all SNPs in each dataset were also aligned to the plus strand of the human genome (build 37p13), removing strand-ambiguous markers in the process. As most control datasets lacked any markers in the X and Y-chromosomes, or in the mitochondrial DNA, every SNP from these regions was discarded. The final merge of all case and control datasets left 203,436 overlapping autosomal SNPs.

All individuals were imputed simultaneously in the Cardiff University high-performance computing cluster RAVEN106, using the SHAPEIT/IMPUTE2 algorithms 5628. As reference panels, a combination of the 1000 Genomes phase 3 (1KGPp3) and UK10K datasets was used, as this has previously been shown to increase the accuracy of imputation for individuals of British ancestry, particularly for rare variants107. After imputation, a principal component analysis (PCA) of variants with minor allele frequency (MAF) higher than 5% was carried out to obtain a general summary of the population structure of the sample, using the EIGENSOFT v6 toolset108. In order to ameliorate population stratification in the association analysis109, any individuals not falling into an area delimited by the mean and 3 standard deviations of the two first principal components of the control samples were excluded from further analyses. The sample was further pruned by removing all individuals with inbreeding coefficients (F) higher than 0.2, and leaving only a random member of each pair with a relatedness coefficient ($\hat{π}$) higher than 0.2. To ensure the independence of our analyses with previous GWASes conducted by the Schizophrenia Working Group of the Psychiatric Genomics Consortium (SCZ-PGC), relatedness coefficients of CLOZUK individuals were also calculated with all the individual datasets included in the latest PGC study (PGC2)26. Detected genetic relatives were excluded as aforementioned. After this imputation and curation process, 35802 samples (11260 cases + 24542 controls) with 9.65 million imputed markers (INFO > 0.3 and MAF > 0.001) remained in the dataset.

**PGC2 meta-analysis:** The CLOZUK GWAS results were combined with a version of PGC2 in which the CLOZUK1 sample had been previously removed. Meta-analysis was performed by using the fixed-effects procedure in METAL30 and weights derived from standard errors. For consistency with the PGC2 mega-analysis, additional filters (INFO > 0.6 and MAF > 0.01) were applied to the CLOZUK and PGC2noCLOZUK summary statistics, leaving 8 million markers in the final meta-analysis results. The same procedure as above was used in order to report independent loci from this analysis. As raw SCZ-PGC2 genotypes were not available for using in the LD-clumping procedure, 1KGPp3 was used as a reference.

**Heritability Analysis:** We used the software LD-Score v1.050 to analyse the aforementioned summary statistics, and obtain an estimate of SNP-based heritability (h2), in the CLOZUK+PGC2 meta-analysis, the largest combined sample we had available (40,675 cases and 64,643 controls). Heritability estimates were transformed to the liability scale using a prevalence of 0.1%, as estimated from population registry studies110. As population structure and sample overlap were explicitly accounted during the curation process of both the CLOZUK and PGC2 studies, the intercept of the LD-score regression was constrained to 1111.

**Acknowledgements**:

Case sample:

We thank the participants and clinicians who took part in the CardiffCOGS study. We acknowledge Sophie Bishop and Amy Lynham, from Cardiff University, for their work in recruitment, interviewing and rating of participants. For the CLOZUK2 sample we thank Leyden Delta, for supporting the sample collection, anonymisation and data preparation (particularly Marinka Helthius, John Jansen and Karel Jollie), Magna Laboratories, UK (Andy Walker) and, for CLOZUK1, Novartis and The Doctor’s Laboratory staff for their guidance and cooperation. We acknowledge Kiran Mantripragada, Lesley Bates, Catherine Bresner and Lucinda Hopkins, at Cardiff University, for laboratory sample management.

Control sample:

A full list of the investigators who contributed to the generation of the Wellcome Trust Case Control Consortium (WTCCC) data is available from www.wtccc.org.uk. Funding for the project was provided by the Wellcome Trust (WT) under award 076113. Venous blood collection for the 1958 Birth Cohort (NCDS) was funded by the UK’s Medical Research Council (MRC) grant G0000934, peripheral blood lymphocyte preparation by Juvenile Diabetes Research Foundation (JDRF) and WT and the cell-line production, DNA extraction and processing by WT grant 06854/Z/02/Z. Genotyping was supported by WT (083270) and the European Union (EU; ENGAGE: HEALTH-F4-2007- 201413). The UK Blood Services Common Controls (UKBS-CC collection) was funded by WT (076113/C/04/Z) and by the National Institute for Health Research (NIHR) programme grant to NHS Blood and Transplant authority (NHSBT; RP-PG-0310-1002). NHSBT also made possible the recruitment of the Cardiff Controls, from participants who provided informed consent. Generation Scotland received core funding from the Chief Scientist Office of the Scottish Government Health Directorates CZD/16/6 and the Scottish Funding Council HR03006. Genotyping of the GS:SFHS samples was carried out by the Genetics Core Laboratory at the WT Clinical Research Facility, Edinburgh, Scotland and was funded by the MRC. The Type 1 Diabetes Genetics Consortium (T1DGC) is a collaborative clinical study sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institute of Allergy and Infectious Diseases (NIAID), National Human Genome Research Institute (NHGRI), National Institute of Child Health and Human Development (NICHD), and JDRF. The People of the British Isles (POBI) project is supported by WT (072974/Z/03/Z, 088262/Z/09/Z, 075491/Z/04/Z, 075491/Z/04/A, 075491/Z/04/B, 090532/Z/09/Z, 084818/Z/08/Z, 095552/Z/11/Z, 085475/Z/08/Z, 098387/Z/12/Z, 098386/Z/12/Z), the Academy of Finland (257654) and the Australian National Health and Medical Research Council (APP1053756). TwinsUK is funded by WT, MRC, EU, NIHR-funded BioResource, Clinical Research Facility and Biomedical Research Centre based at Guy’s and St Thomas’ NHS Foundation Trust in partnership with King’s College London. Funding for the QIMR samples was provided by the Australian National Health and Medical Research Council (241944, 339462, 389875, 389891, 389892, 389927, 389938, 442915, 442981, 496675, 496739, 552485, 552498, 613602, 613608, 613674, 619667), the Australian Research Council (FT0991360, FT0991022), the FP-5 GenomEUtwin Project (QLG2-CT- 2002-01254) and the US National Institutes of Health (NIH; AA07535, AA10248, AA13320, AA13321, AA13326, AA14041, MH66206, DA12854, DA019951), and the Center for Inherited Disease Research (Baltimore, MD, USA). TEDS is supported by a program grant from the MRC [G0901245-G0500079], with additional support from the NIH [HD044454; HD059215]. In the GERAD Consortium, Cardiff University was supported by WT, MRC, Alzheimer’s Research UK (ARUK) and the Welsh Government. Kings College London acknowledges support from the MRC. The University of Belfast acknowledges support from ARUK, Alzheimer's Society, Ulster Garden Villages, N.Ireland R&D Office and the Royal College of Physicians/Dunhill Medical Trust. Washington University was funded by NIH grants, Barnes Jewish Foundation and the Charles and Joanne Knight Alzheimer's Research Initiative. The Bonn group was supported by the German Federal Ministry of Education and Research (BMBF), Competence Network Dementia and Competence Network Degenerative Dementia, and by the Alfried Krupp von Bohlen und Halbach-Stiftung.

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**ATTENTION DEFICIT/HYPERACTIVITY DISORDER (ADHD)**

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