

Supplementary Notes

A. Merging, Quality Control and Imputation

Using Ricopili²⁶, we developed and applied the following stringent and iterative procedure to match cohorts based on country and genome chip and perform quality control. We first evaluated potential merges of cohorts with cases of both phenotypes (“case-case” cohorts, with BD coded as “cases” and MDD coded as “controls”) based on matched genotyping platform and country. After performing the standard Ricopili QC pipeline, we evaluated each potential match based on nine criteria, to avoid technical artifacts, extremely unbalanced cohorts and inflation due to population structure, as well as to secure adequate number of good-quality variants: 1) the number of SNPs post-QC (fewer than 200,000 not considered) 2) the number of SNPs post-QC per platform (threshold is platform-dependent) 3) and 4) the number of MDD and BD cases (fewer than 100 not considered), 5) the number of participants lost relative to the total sample size (above 10% not considered) 6) number of individuals without phenotype (more than 10 not considered), 7) the ratio of cases failing sex-check (greater than 2.5% not considered) 8) lambda of post-QC GWAS without covariates (greater than 1.2 not considered) 9) number of genome-wide significant SNPs (not considered if any, given the small sample size of individual cohorts). Acceptable values for these criteria on all cohort-merges were obtained by iteratively excluding variants or individuals, while as few cohorts as possible were dropped altogether. We were able to match about 63% of BD cases and 65% of MDD cases of the total PGC collection, grouped into 13 case-case cohorts, and included 15,532 BD cases with 12,920 MDD cases from 22 and 19 cohorts respectively (Suppl. Tables 1, 2).

Principal component analysis of all the final merged cohorts as well as the final QC criteria can be found in Supp. Figures 1a, 1b and Supp. Tables 2 and 3.

Consistent with prior PGC analyses, QC was followed by imputation to the European subset of the Haplotype Reference Consortium (HRC) reference panel⁴⁰. Specifically, genotype imputation was performed using the pre-phasing/imputation stepwise approach implemented in EAGLE / MINIMAC3 (with a variable chunk size of 132 genomic chunks and default parameters). The imputation reference set consisted of

54,330 phased haplotypes with 36,678,882 variants from the publically available HRC reference. For subsequent analysis we imposed a filter of INFO>0.8 on imputation quality score.

B. Meta-regression framework

We use a meta-regression approach to model the effect size of each SNP (the coefficient of the SNP from the GWAS) as a function of a single fixed covariate: a binary indicator of phenotype (BD or MDD). Specifically, we used a random-effects meta regression, where group was treated as a random effect. In this random-effects model, a groups's observed effect size $\hat{\theta}_k$ deviates from θ because of sampling error ϵ_k and an additional error term ζ_k (variance τ^2) related to the variability of individual groups around their true effect sizes. This regression uses maximally 26 estimates of effect size (two for each of the 13 groups). We analyzed only SNPs where there were GWAS summary statistics for at least 10 of the 13 groups.

The variable τ^2 from the meta-regression estimates the variance of distribution of true SNP effect sizes after accounting for the fixed covariate (phenotype). We also performed a separate random effects meta-analyses of the BD and MDD GWAS summary statistics, obtaining two estimates of τ^2 , one for BD and one for MDD, to evaluate which phenotype appeared to have more heterogeneity in SNP effect sizes.

C. Comparison between the BDvsMDD and BD predictors with equal sample sizes

Since the BD GWAS has a larger sample size than the BDvsMDD GWAS, due to the fact that control individuals (included in the former) outnumber MDD individuals (included in the latter), we also calculated scores based on a subsampled version of the former, whereby the number of controls included in the BD GWAS is downsampled to the same number as that of MDD cases in the BDvsMDD GWAS, to provide a fair comparison between the two.

Our comparison of BD and BDvsMDD with similar sample sizes is shown in Supp. Figure 3A (also see Supp. Figure 6C,D). In this scenario, the performance difference is no longer significant ($p = 0.28$ for equal sample size, paired weighted t-test), meaning that the BDvsMDD predictor still does not outperform the BD predictor, even after accounting for sample size.

D. Full list of members of the Bipolar Disorder Working Group of the PGC, the Major Depressive Disorder Working Group of the PGC and the iPSYCH Consortium

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