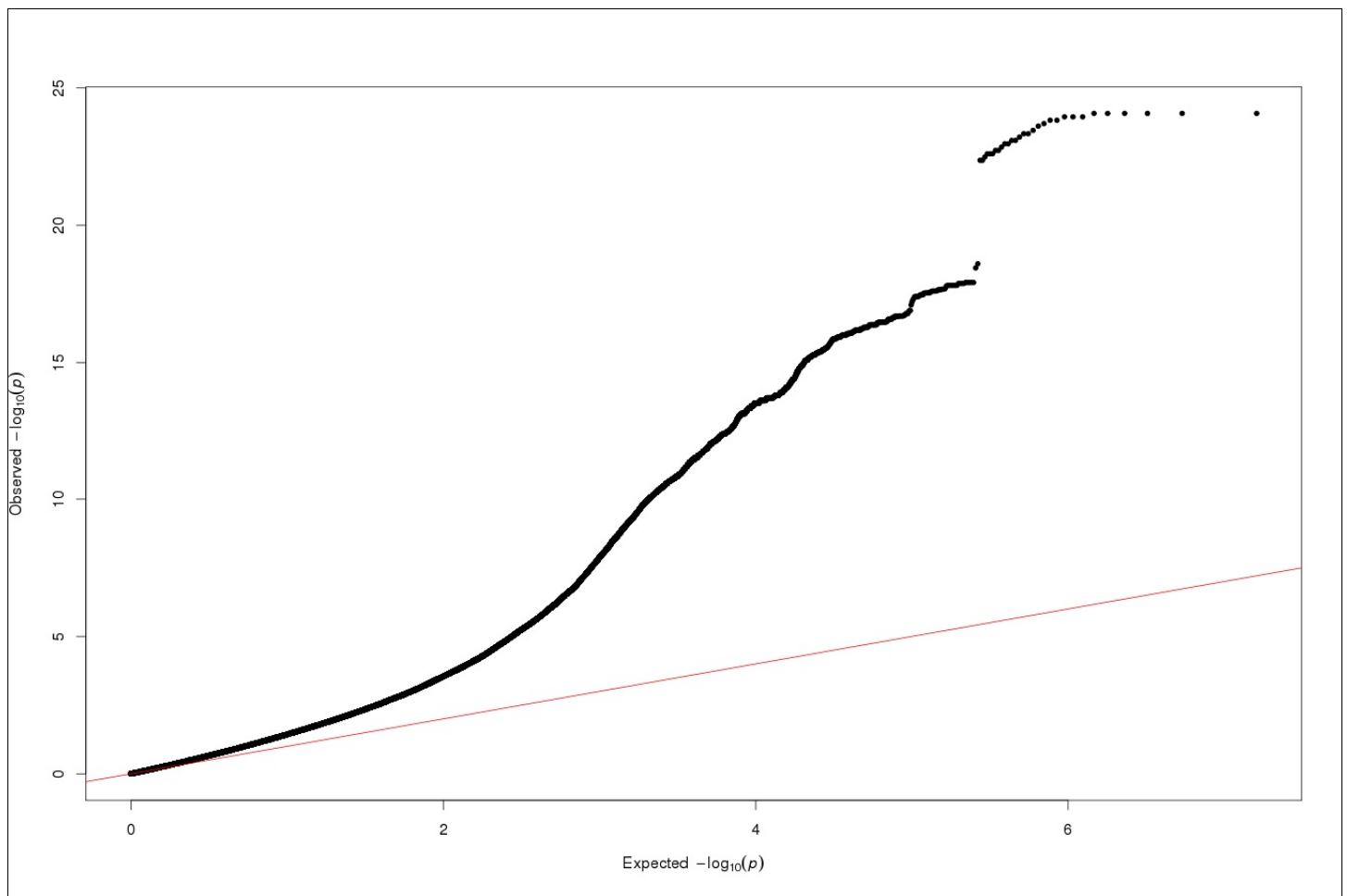


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Genome-wide meta-analysis of depression identifies 102 independent variants and highlights the importance of the prefrontal brain regions

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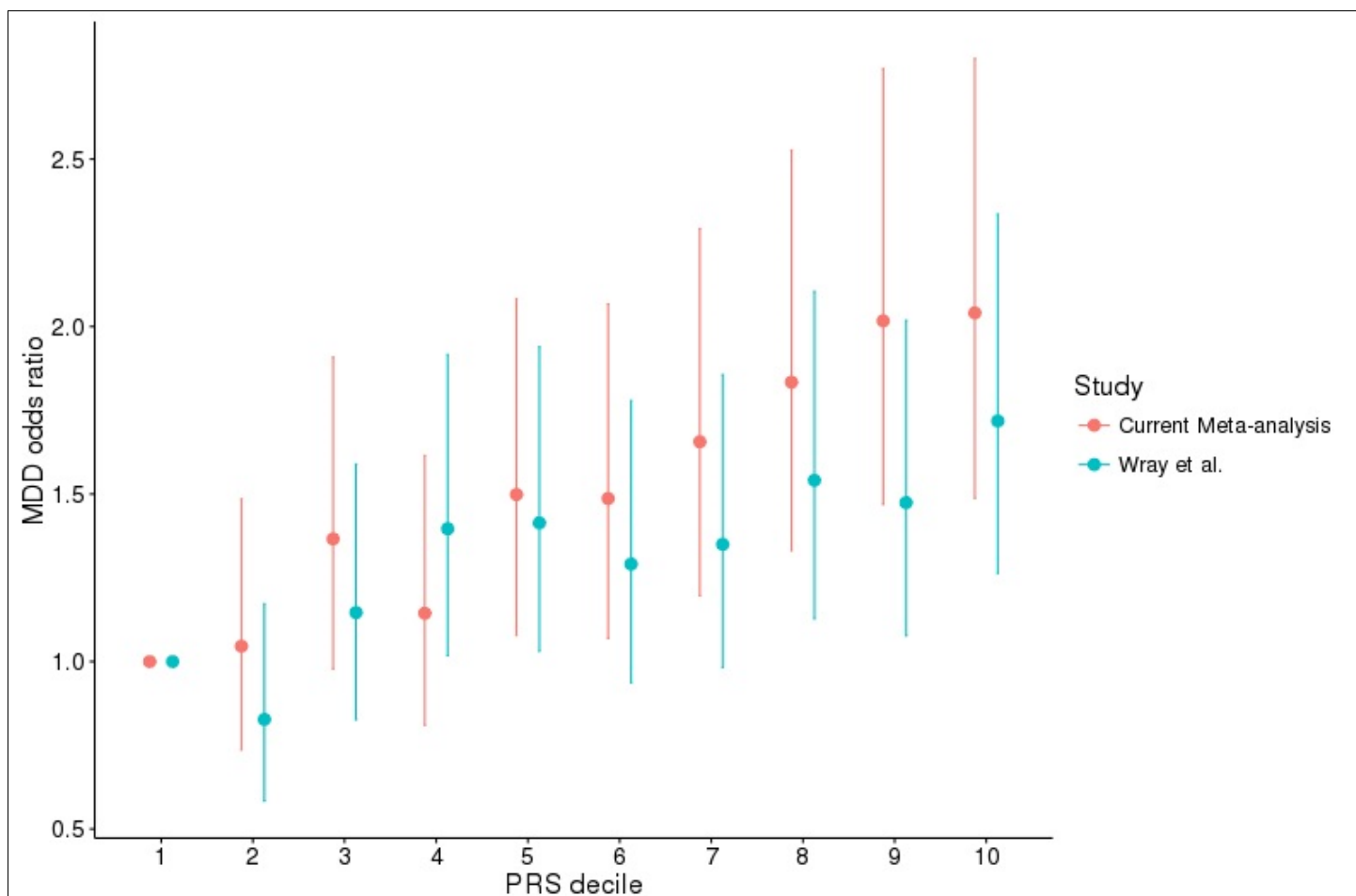
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Supplementary Figure 1

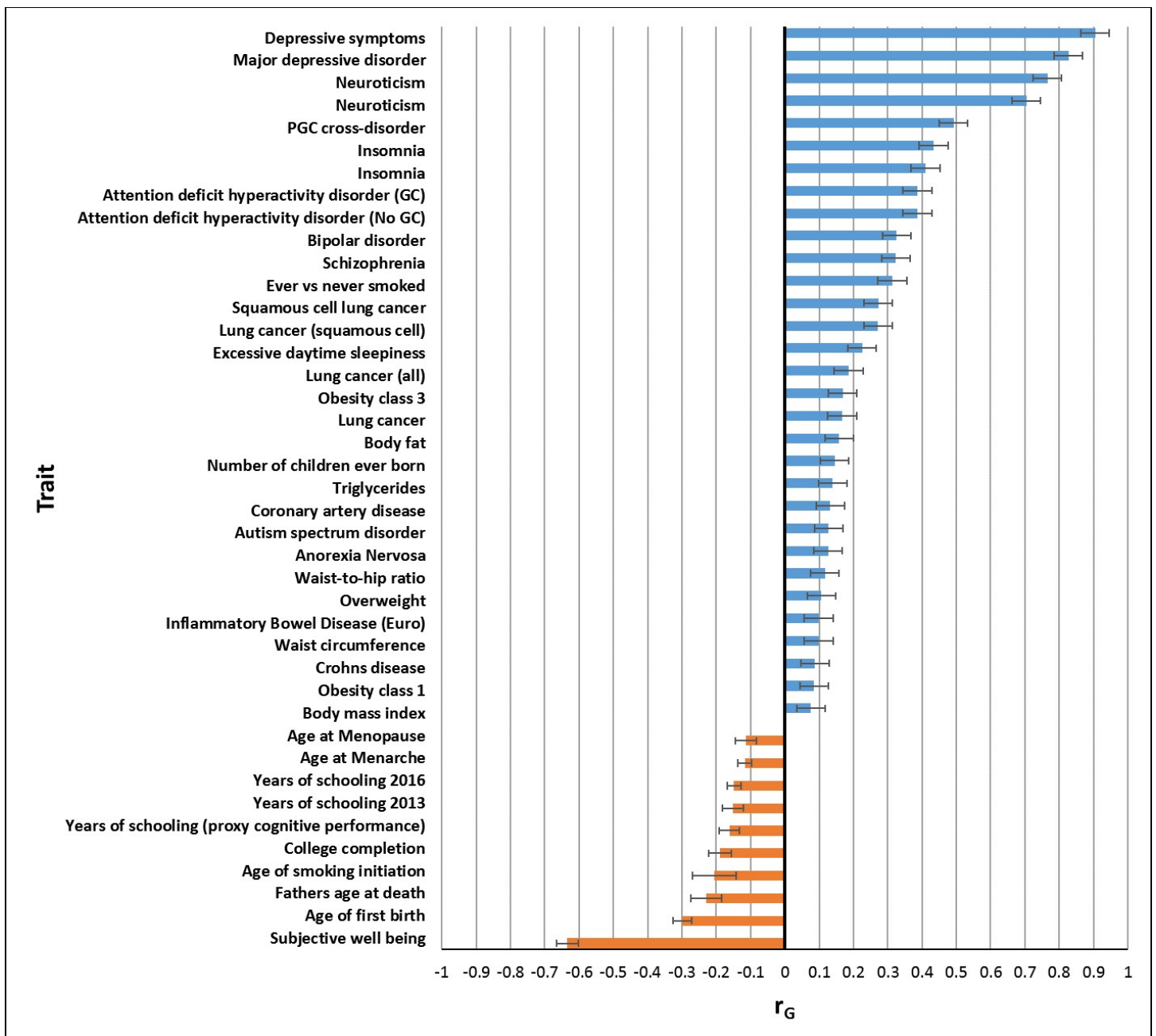
Quantile–quantile plot of the observed P values on those expected for each genetic variants association with depression in our meta-analysis ($n = 807,553$ individuals).

P -values were obtained from an inverse-variance weighted meta-analysis of summary statistics data from UK Biobank, Psychiatric Genomics Consortium and 23andMe. UK Biobank summary statistics were obtained using a linear association analysis with the results transformed to the logistic scale. Psychiatric Genomics Consortium and 23andMe data were analysed using an additive logistic model. All association analyses were two-sided tests.



Supplementary Figure 2

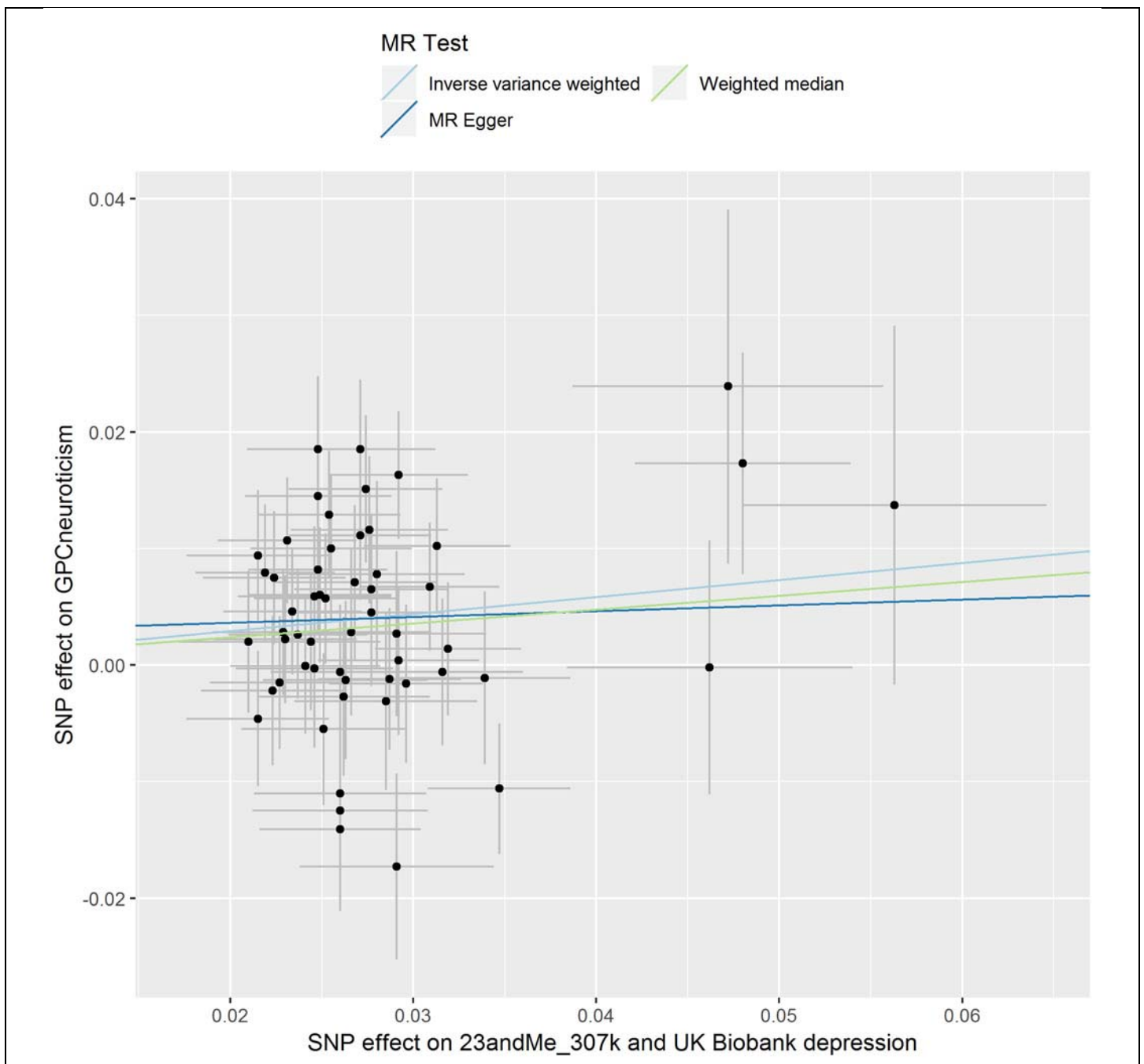
Odds ratios and 95% confidence intervals for major depressive disorder (MDD) in Generation Scotland ($n = 6,946$) based on polygenic risk score (PRS) deciles calculated from the current meta-analysis of depression ($n = 765,884$ individuals) and from summary statistics from the genome-wide association study of major depression conducted by Wray, et al.⁹ ($n = 453,779$ individuals).



Supplementary Figure 3

Significant genetic correlations (r_G ; $P < 0.01$, after false discovery rate correction for multiple testing) between depression ($n = 807,553$ individuals) and other behavioural and disease related traits using LD score regression.

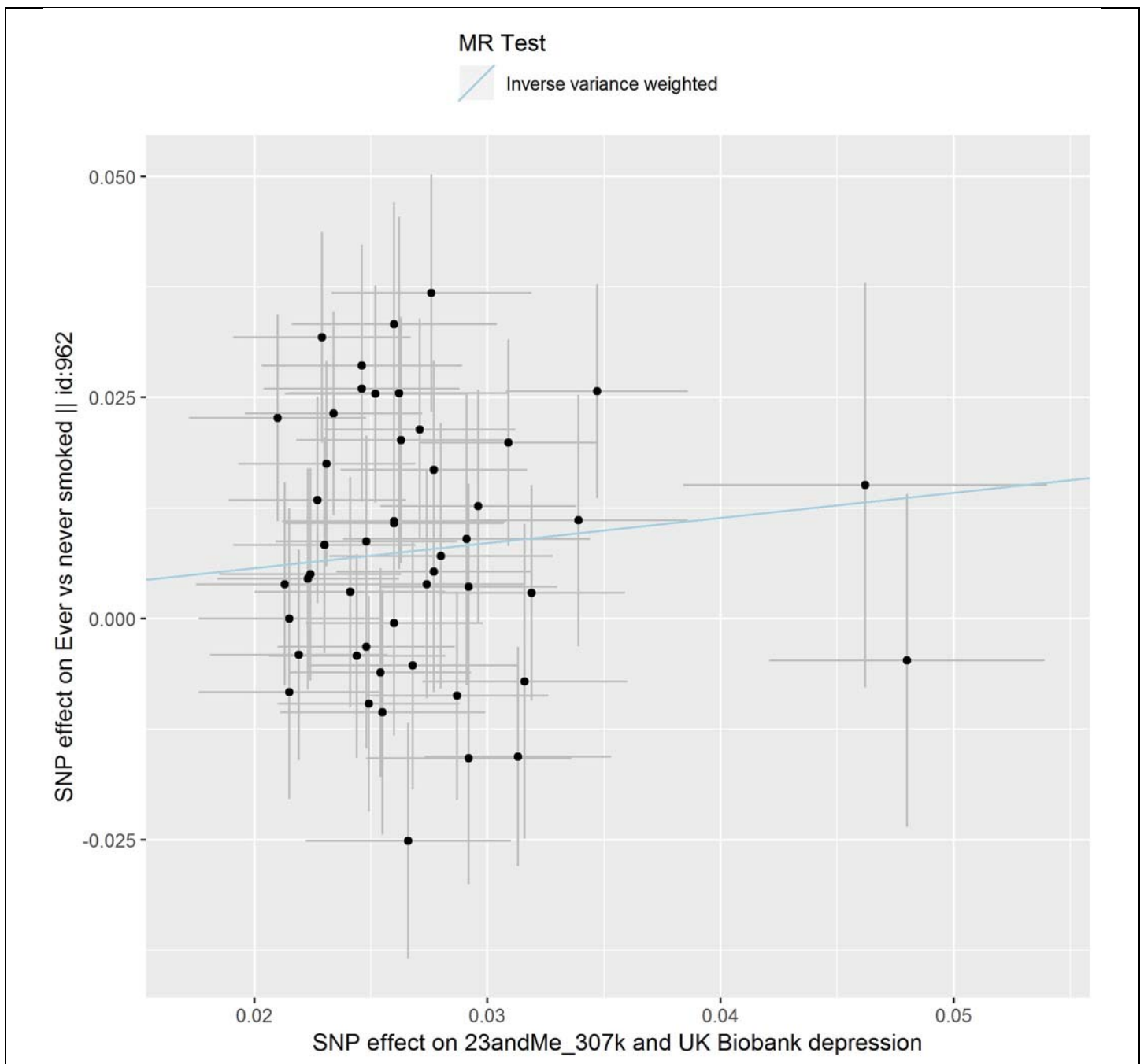
A negative r_G indicates that an earlier or lower value of a continuous trait (i.e. earlier father's age of death or lower subjective well being) was associated with depression. A positive r_G indicates that a later or higher value of a continuous trait (i.e. higher triglyceride level) was associated with depression. P -values were calculated by testing whether r_G was significantly different from 0 using a two-sided test. The centre values are the genetic correlation for each trait and the error bars are the respective standard error. LD score regression implemented in LD Hub software (<http://ldsc.broadinstitute.org/>).



Supplementary Figure 4

Mendelian randomization test for a putative causal effect of depression ($n = 629,934$ individuals; 56 instrumental variables) on neuroticism ($n = 29,496$ twin pairs) using inverse weighted regression, MR Egger and a weighted median test.

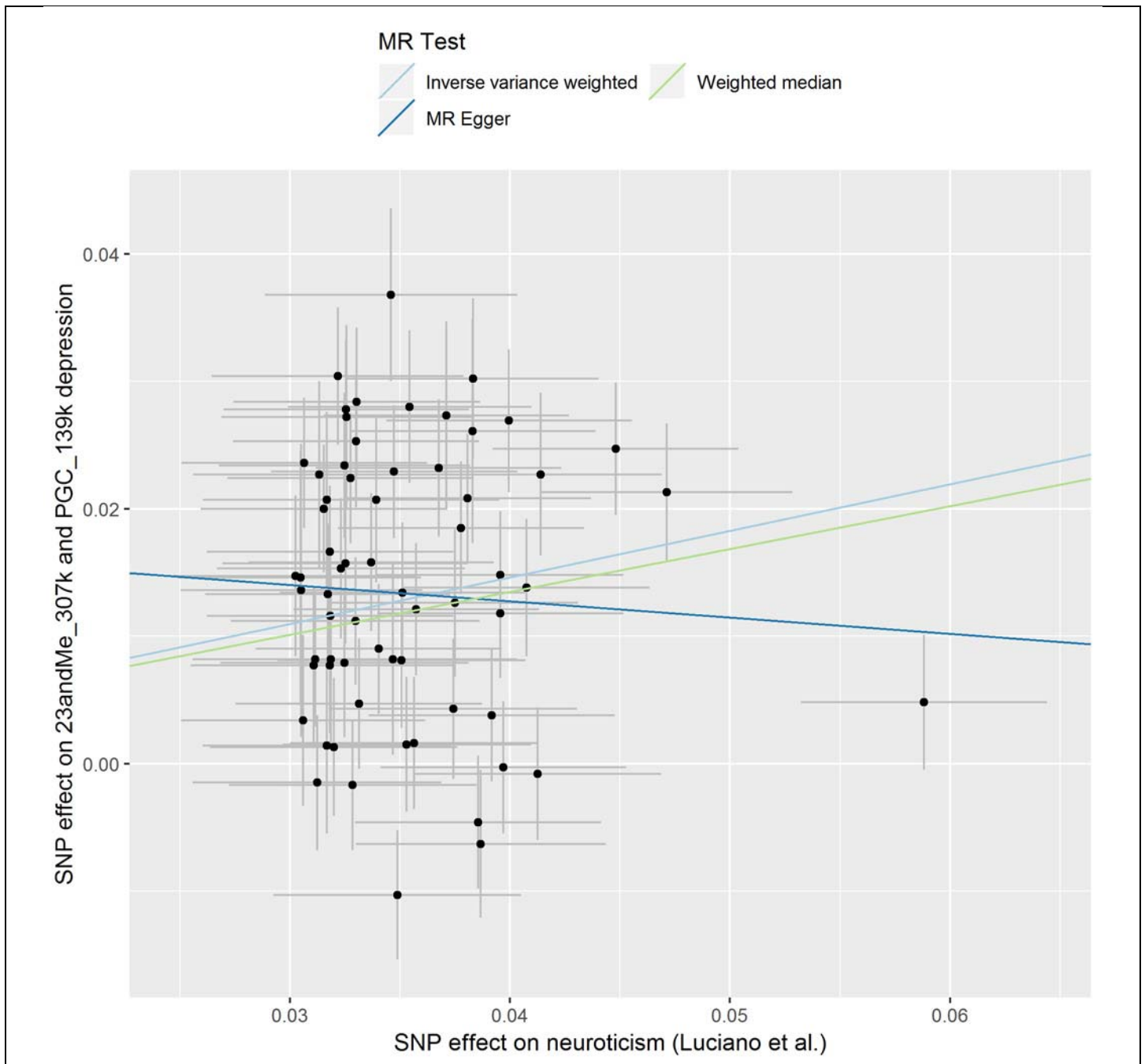
Centre values reflect the effect of each instrumental variable on each trait with the respective error bars reflecting the standard error of the variable's effect on each trait.



Supplementary Figure 5

Mendelian randomization analysis for a putative causal effect of depression ($n = 629,934$ individuals; 49 instrumental variables) on ever vs. never smoked ($n = 69,409$ individuals) using inverse weighted regression.

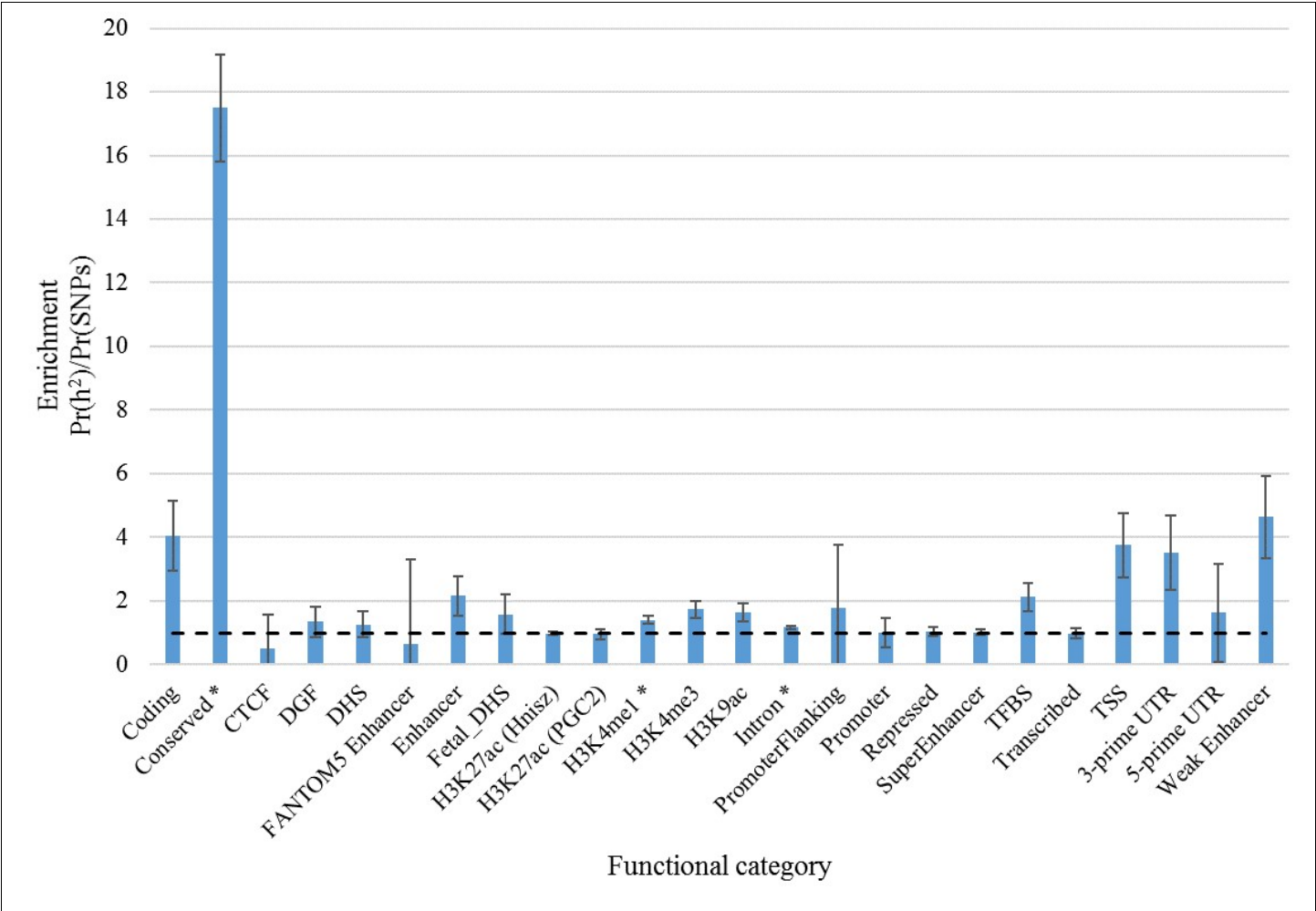
Centre values reflect the effect of each instrumental variable on each trait with the respective error bars reflecting the standard error of the variable's effect on each trait.



Supplementary Figure 6

Mendelian randomization test for a putative causal effect of neuroticism ($n = 329,821$ individuals; 65 instrumental variables) on depression ($n = 537,701$ individuals) using inverse weighted regression, MR Egger and a weighted median test.

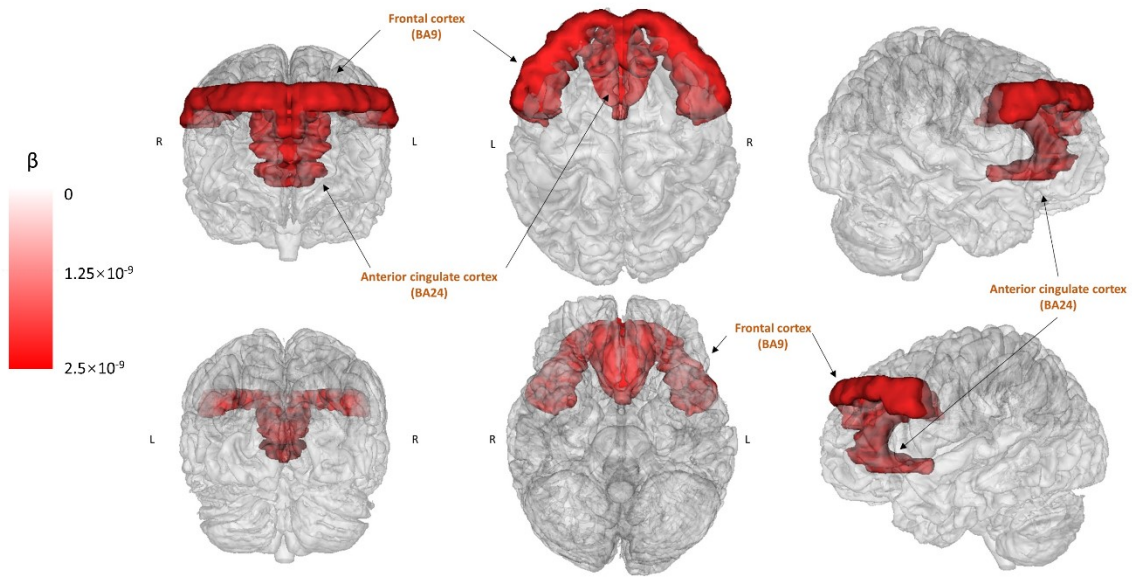
Centre values reflect the effect of each instrumental variable on each trait with the respective error bars reflecting the standard error of the variable's effect on each trait.



Supplementary Figure 7

Contribution of functional annotation of 24 categories to the heritability of depression ($n = 807,553$ individuals) based on the variants within each category.

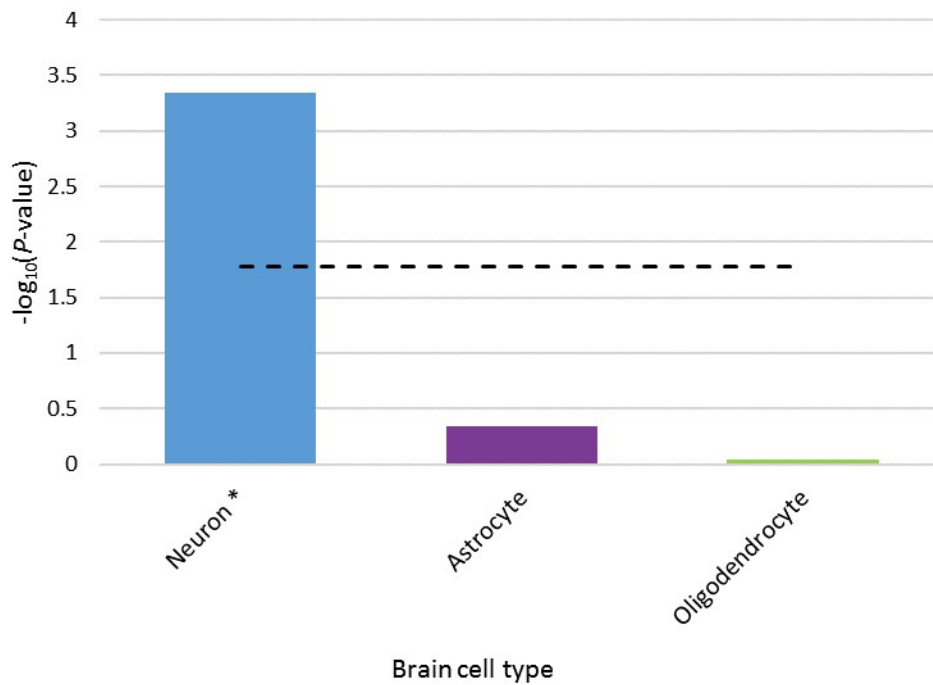
The enrichment of each functional category, shown on the y-axis, is calculated as the proportion of heritability assigned to a functional categories divided by the proportion of variants in that category ($\text{Pr}(h^2)/\text{Pr}(\text{SNPs})$). Error bars represent jackknife standard errors for each the estimate of enrichment, and an asterisk indicates significant enrichment after Bonferroni correction for multiple testing (Conserved P -value = 2.55×10^{-17} ; H3K4me1 P -value = 0.0015; Intron P -value = 0.0014). P -values were calculated using a one-sided test and tested whether there the estimate of enrichment was significantly different from zero enrichment. The dashed line represents the threshold for no enrichment.



Supplementary Figure 8

Enrichment estimate (β) of significantly enriched brain cell regions using GTEx overlaid on physical representation of brain anatomy ($n = 807,553$ individuals).

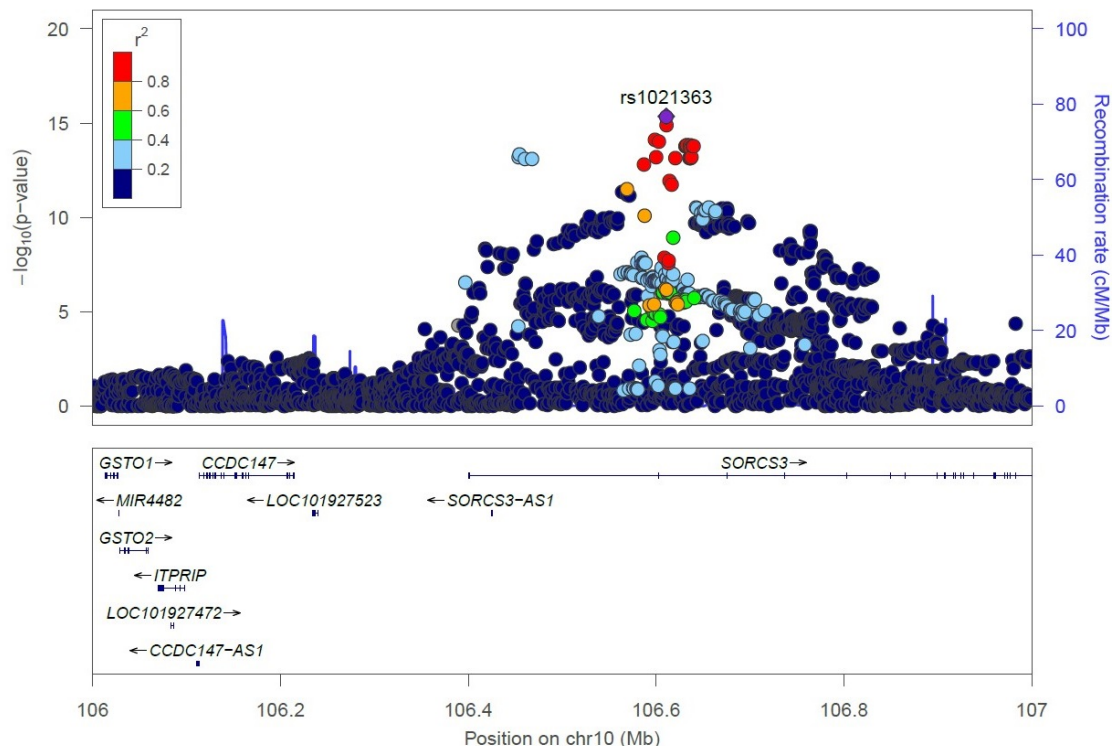
The pseudo-coloring highlights the coefficients of the brain regions in red that were significantly enriched ($P < 0.05$) for depression variants compared to no enrichment using a one-sided test and after Bonferroni correction for multiple testing.



Supplementary Figure 9

Stratified LD score regression analyses showing significance of enrichment estimates for 3 brain cell types in depression ($n = 807,553$ individuals).

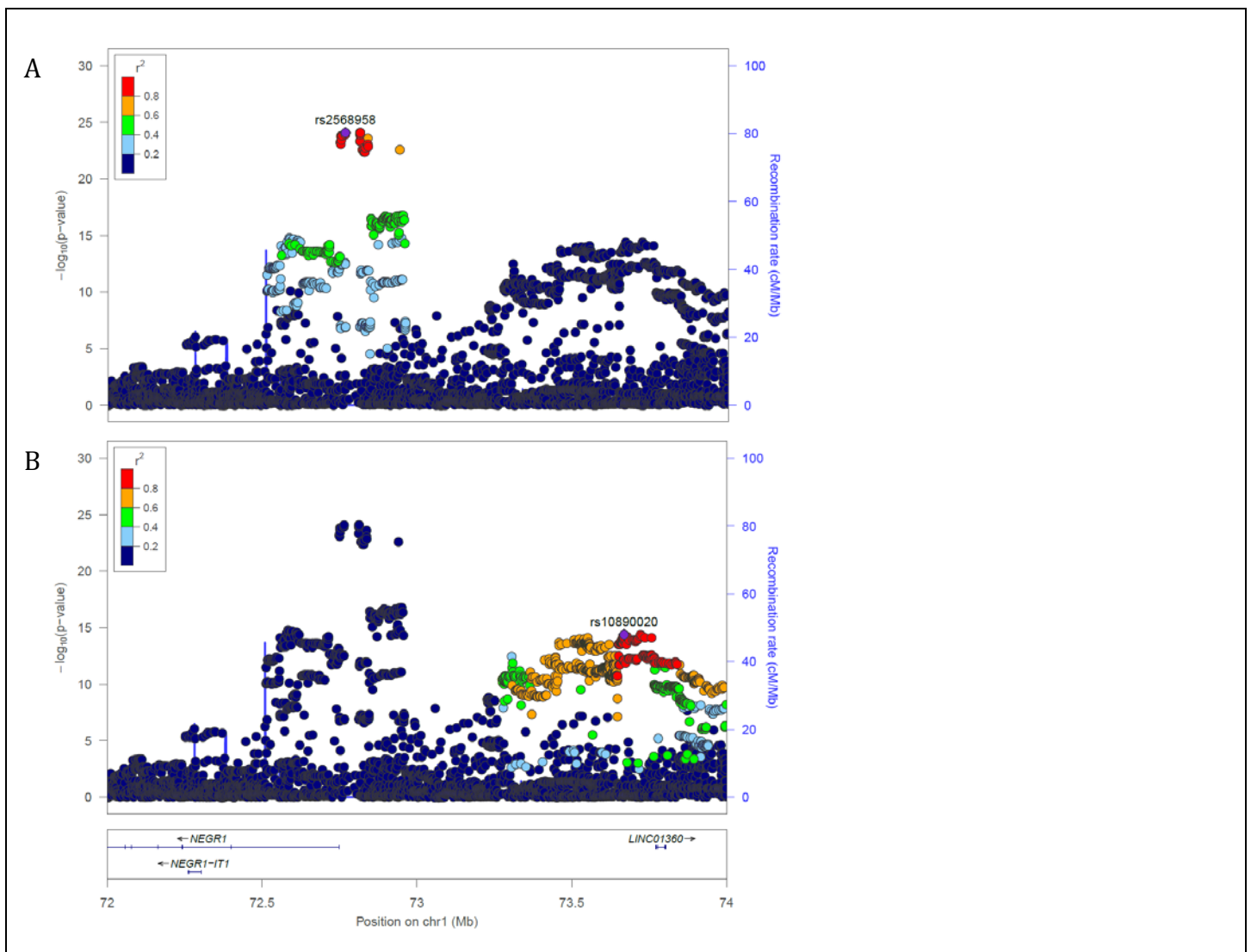
The dashed line represents the Bonferroni threshold for significance for multiple testing correction ($P = 0.0167$) and * indicates significant enrichment for that brain cell type (Neuron $P\text{-value} = 4.52 \times 10^{-4}$). $P\text{-values}$ were calculated based on evidence of enrichment compared to no enrichment using a one-sided test.



Supplementary Figure 10

Regional visualization plot centred on the independently-associated variant, rs1021363, close to the Sortilin-related VPS10 domain containing receptor 3 (SORCS3) gene on chromosome 10 ($n = 807,553$ individuals)

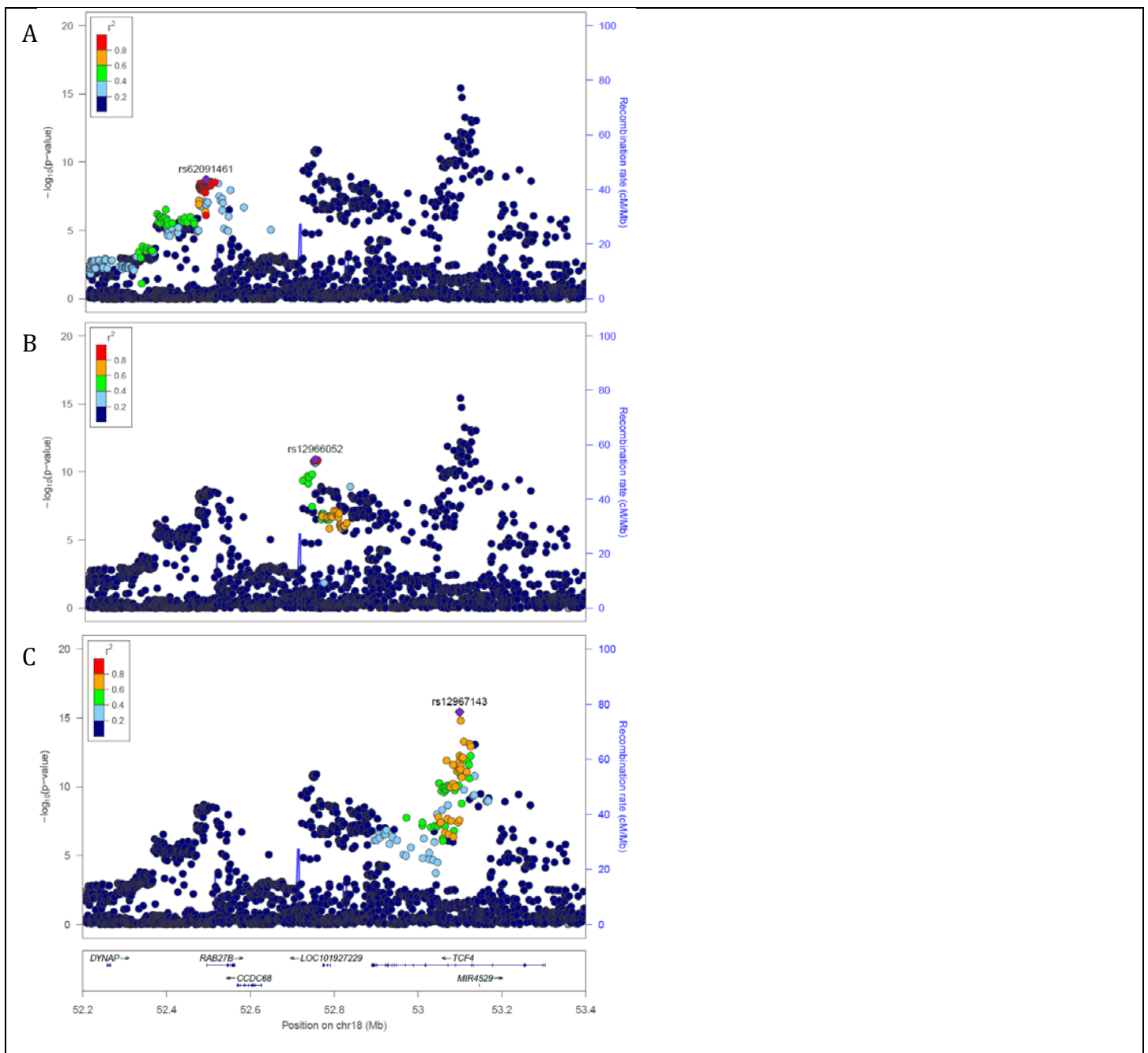
P-values were obtained from an inverse-variance weighted meta-analysis of summary statistics data from UK Biobank, Psychiatric Genomics Consortium and 23andMe. UK Biobank summary statistics were obtained using a linear association analysis with the results transformed to the logistic scale. Psychiatric Genomics Consortium and 23andMe data were analysed using an additive logistic model. All association analyses were two-sided tests. Recombination rates used in the plots are based on the European 1000 Genomes panel from November 2014.



Supplementary Figure 11

Regional visualization plots centred on independently-associated variants (A. rs2568958 and B. rs10890020) close to the neuronal growth regulator 1 (*NEGR1*) gene on chromosome 1 ($n = 807,553$ individuals).

P-values were obtained from an inverse-variance weighted meta-analysis of summary statistics data from UK Biobank, Psychiatric Genomics Consortium and 23andMe. UK Biobank summary statistics were obtained using a linear association analysis with the results transformed to the logistic scale. Psychiatric Genomics Consortium and 23andMe data were analysed using an additive logistic model. All association analyses were two-sided tests. Recombination rates used in the plots are based on the European 1000 Genomes panel from November 2014.



Supplementary Figure 12

Regional visualization plots centred on independently-associated variants (A. rs62091461, B. rs12966052, and C. rs12967143) close to the transcription factor 4 (*TCF4*) and *RAB27B* genes on chromosome 18 ($n = 807,553$ individuals).

P-values were obtained from an inverse-variance weighted meta-analysis of summary statistics data from UK Biobank, Psychiatric Genomics Consortium and 23andMe. UK Biobank summary statistics were obtained using a linear association analysis with the results transformed to the logistic scale. Psychiatric Genomics Consortium and 23andMe data were analysed using an additive logistic model. All association analyses were two-sided tests. Recombination rates used in the plots are based on the European 1000 Genomes panel from November 2014.

General directionality agreement of variants that have previously been associated with depression

Comparison with Hyde, et al. ¹

There were 17 variants associated with depression in the joint analysis conducted by Hyde, et al. ¹. All had an effect in the same direction in UK Biobank and 16 variants had an effect in the same direction in PGC_139k. In the current meta-analysis (which included the discovery cohort used by Hyde et al. ¹) all 17 variants had an effect in the same direction as that reported by Hyde et al. ¹ with 14 variants maintaining $P < 5 \times 10^{-8}$ and 13 variants providing stronger evidence of an effect based on the observed lower P -values compared to Hyde et al. ¹. All 17 of the Hyde et al. ¹ associated variants were within $\pm 1\text{Mb}$ of a significant variant ($P < 5 \times 10^{-8}$) in the current meta-analysis.

Comparison with Howard, et al. ²

There were 17 associated variants reported for depression in UK Biobank by Howard, et al. ² of which 16 were available in the current meta-analysis. Fifteen had an effect in the same direction in 23andMe_307k and 14 had an effect in the same direction in PGC_139k. In the current meta-analysis (which includes the broad depression phenotype from Howard, et al. ²) the direction of effect for all 16 variants matched that observed by Howard, et al. ². Nine of the 16 variants maintained $P < 5 \times 10^{-8}$ in the meta-analysis and 8 variants had lower P -values for an association with depression compared to Howard, et al. ². Twelve out of the 16 variants (excluding the MHC region) identified in UK Biobank by Howard, et al. ² were within $\pm 1\text{Mb}$ of a significant variant ($P < 5 \times 10^{-8}$) in the meta-analysis.

Comparison with Wray, et al. ³

The Wray, et al. ³ analysis of PGC identified 44 variants associated with depression of which 38 were available in the current meta-analysis. The Wray, et al. ³ study included an earlier release of the UK Biobank data, 23andMe_307k and PGC_139k and it is not surprising that the direction of effect was consistent across all these studies and the current meta-analysis. There were 28 variants that maintained $P < 5 \times 10^{-8}$ in the meta-analysis and 22 variants had lower P -values compared to those reported by Wray, et al. ³. Thirty six of the 43 significant variants (MHC region excluded) from Wray, et al. ³ were within $\pm 1\text{Mb}$ of a significant variant ($P < 5 \times 10^{-8}$) in the meta-analysis.

Cohort information for the 23andMe replication dataset

All participants within the replication dataset were drawn from the customer base of 23andMe, Inc. Saliva samples were used for DNA extraction and genotyping which was undertaken by the National Genetics Institute, a Clinical Laboratory Improvement Amendments licensed clinical laboratory and a subsidiary of Laboratory Corporation of America. Samples were genotyped on one of four platforms:

- The V1 and V2 platforms were versions of the Illumina HumanHap550+ BeadChip, with approximately 25,000 additional custom SNPs chosen by 23andMe, providing a total of about 560,000 SNPs
- The V3 platform had a total of about 950,000 SNPs and was based on the Illumina OmniExpress+ BeadChip, with custom SNPs selected to improve overlap with the V2 array.
- The V4 platform is a fully customized array of about 570,000 SNPs, including a lower redundancy subset of V2 and V3 SNPs and with additional coverage of lower allele frequency coding variation.

Those samples that had a call rate $\leq 98.5\%$ were re-analysed and additional samples were obtained from individuals whose samples repeatedly failed on this criterion.

To prepare the genotype data for imputation, the data was phased using Finch which implements the Beagle haplotype graph-based phasing algorithm⁴. Prior to phasing each chromosome was split into chunks containing a maximum of 300,000 variants with an overlap of 10,000 variants on either side. The Minimac3⁵ imputation model parameters were estimated for each chunk using single batches of 10,000 individuals. A combined imputation panel based on the May 2015 release of the 1000 Genomes Phase 3 haplotypes⁶ and the UK10K imputation reference panel⁷ was used to improve imputation accuracy. The phased data was imputed against the chunked combined imputation panel using Minimac3.

Individuals were then excluded that had $\leq 97\%$ European ancestry based on haplotype classification⁸ and a segmental identity-by-descent (IBD) estimation algorithm⁹ was used to identify a maximal set of unrelated individuals who were no closer than first cousins in an outbred population and unrelated to the 23andMe_307k cohort. The phenotype was the same as that used in the 23andMe_307k cohort and reported in Hyde, et al.¹ and was based on responses to web-based surveys with individuals that self-reported as having received a clinical diagnosis or treatment for depression classified as cases. This provided a total of 1,306,354

individuals (414,055 cases and 892,299 controls) with which to conduct an association analysis of the 102 variants found to be significant in the meta-analysis of PGC_139k, 23andMe_307k and UK Biobank. The association analysis was conducted using logistic regression and assuming additive allelic effects using imputed dosages. Age, sex, the top five principal components (to account for residual population structure), and genotype platform were fitted as covariates in the model. *P*-values were calculated using a likelihood ratio test.

All individuals in this replication cohort provided informed consent. The online surveys used to collect data by 23andMe, Inc. follow their human subjects protocol which has been reviewed and approved by Ethical & Independent Review Services, a private institutional review board (<http://www.eandireview.com>).

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Version 5. 2018-07-17