nature portfolio

https://doi.org/10.1038/s41586-024-07019-6

Supplementary information

Genetic drivers of heterogeneity in type 2 diabetes pathophysiology

In the format provided by the authors and unedited

Genetic drivers of heterogeneity in type 2 diabetes pathophysiology

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SUPPLEMENTARY INFORMATION

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Summary of loci identified through recent large-scale multi-ancestry meta-analyses. Two recent partially overlapping multi-ancestry meta-analyses of T2D GWAS together account for 69.3% of the total effective sample size of the multi-ancestry meta-regression undertaken by the T2D Global Genomics Initiative (**Supplementary Figure 1**). First, the meta-analysis of GWAS from the Million Veteran Program[1], which includes 228,499 T2D cases and 1,178,783 controls. Second, the meta-analysis of GWAS from the DIAMANTE Consortium[2], which includes 180,834 cases and 1,159,055 controls. We aimed to provide a comprehensive overview of the genetic contribution to T2D by summarising loci reported in these multi-ancestry GWAS meta-analyses at the conventional genome-wide significance threshold ($P<5x10^{-8}$) and a more stringent multi-ancestry genome-wide significance threshold ($P<5x10^{-9}$) proposed by the DIAMANTE Consortium. We aggregated loci reported in each of the three meta-analyses, ensuring no overlap between adjacent loci. Taken together, the three studies report 636 non-overlapping loci spanning 835.5Mb, of which 536 (84.3%) meet stringent multi-ancestry genome-wide significance in at least one of the multi-ancestry meta-analyses (**Supplementary Table 25**).

We investigated the likelihood that loci reported at the conventional genome-wide significance threshold by the DIAMANTE Consortium meet the more stringent multi-ancestry threshold in the larger sample size afforded by the T2D Global Genomics Initiative. We focussed on comparing results from these two efforts because both used the same meta-regression approach (MR-MEGA) to aggregate association summary statistics across GWAS. Of 39 loci with association signals meeting $5x10^{-9}\leq P<5x10^{-8}$ reported by the DIAMANTE Consortium, 36 (92.3%) attained multi-ancestry genome-wide significance in the T2D Global Genomics Initiative (**Supplementary Table 25**). Of the three loci that did not meet the more stringent threshold, the signal at the *RASA1* locus was marginally more strongly associated (lead SNV rs11953892, $P=1.6x10^{-8}$ versus $P=1.9x10^{-8}$) in the T2D Global Genomics Initiative meta-analysis than in the DIAMANTE Consortium meta-analysis. However, association signals at the two remaining loci were weaker in the T2D Global Genomics Initiative than in the DIAMANTE Consortium, despite the increase in sample size. At the locus encompassing *CCDC39* and *FXR1*, the association signal was nominally significant in the Million Veteran Program (lead SNV rs4854992, $P=0.0081$) with the same direction of effect as in the DIAMANTE Consortium meta-analysis. However, at the *CFAP6* locus, there was no association in the Million Veteran Program (lead SNV rs7261425, $P=0.13$).

Taken together, these results indicate that index SNVs attaining the conventional threshold of $P<5x10^{-8}$ are unlikely to be false positive association signals but have modest effects that require larger sample sizes to meet multi-ancestry genome-wide significance.

Clusters are differentially associated with insulin-related endophenotypes. We assessed the association of index SNVs with insulin-related endophenotypes that were not used for clustering and derived from: hyperinsulinemic-euglycemic clamp assessments and oral glucose tolerance tests (OGTT) in up to 1,316 Mexican American participants without diabetes from the GUARDIAN Consortium[3]; and homeostatic model assessment measures of beta-cell function (HOMA-B) and insulin resistance (HOMA-IR) in up to 36,466 non-diabetic EUR individuals from MAGIC[4] (**Supplementary Methods**). We observed significant heterogeneity in the effects of T2D risk alleles at index SNVs between clusters on HOMA-B ($P_{HET}<2.2x10^{-16}$), HOMA-IR ($P_{HET}=4.1x10^{-15}$), insulin secretion (OGTT-derived area under the

curve for insulin normalised for glucose from baseline to 30 minutes, P_{HET}=0.0026), and insulin sensitivity (clamp-derived glucose infusion rate, P_{HET}=0.026). T2D risk alleles at index SNVs showed a gradient of effects on these correlated measures across clusters (**Extended Data Figure 4, Supplementary Tables 10 and 11**), representing a cline from insulin production and processing in the two beta-cell dysfunction clusters (increased insulin sensitivity; decreased insulin secretion, HOMA-B, and HOMA-IR) through to insulin resistance (decreased insulin sensitivity; increased insulin secretion, HOMA-B, and HOMA-IR) that was most extreme in the lipodystrophy cluster.

Clusters are differentially associated with insulin resistance-related disorders. To understand the shared biological pathways driving genetic correlations with gestational diabetes mellitus (GDM) and polycystic ovary syndrome (PCOS), we extracted association summary statistics for each T2D index SNV from the largest available published GWAS for both disorders[5,6] (**Supplementary Methods**). We observed significant heterogeneity in the effects of T2D risk alleles at index SNVs between clusters for both disorders (**Extended Data Figure 5, Supplementary Table 12**): GDM (P_{HET}=7.0x10^{-16}) and PCOS (P_{HET}=0.00022). Index SNVs in the beta-cell +PI cluster demonstrated the strongest associations with GDM. This cluster includes T2D index SNVs that overlap with association signals previously reported for GDM, mapping to/near *MTNR1B*, *CDKAL1*, *TCF7L2*, and *CDKN2A-CDKN2B*, consistent with hyperglycaemia due to beta-cell dysfunction on a background of pregnancy-induced physiologic insulin resistance[7]. In contrast, PCOS is most strongly associated with index SNVs in the obesity cluster, consistent with previous Mendelian randomization studies that report a strong causal effect of higher BMI on increased PCOS risk[8].

Cluster-specific associations of index SNVs with circulating GLP-1 concentrations. The beta-cell -PI cluster was enriched in adult enterochromaffin cells, a type of enteroendocrine cell that plays an essential role in regulating intestinal motility and secretion in the gastrointestinal tract[9]. Enterochromaffin cells are a major target for GLP-1 and highly express GLP-1 receptor, whose agonists are widely used as medications for T2D[10]. Between clusters, we compared the associations of index SNVs with 2-hour and fasting circulating GLP-1 concentrations in up to 3,514 EUR individuals from the Malmo Diet and Cancer Study[11] and the PPP-Botnia Study[12] (**Supplementary Methods**). Whilst differences in the effects of index SNVs on these measures were not significant between clusters (P>0.05), T2D risk alleles for index SNVs in the beta-cell -PI cluster showed a trend of association with decreased 2-hour GLP-1, whilst those in other clusters showed a trend of association with increased fasting GLP-1 (**Supplementary Figure 13**). Additional analyses in GLP-1 GWAS with larger sample sizes will be required to validate this finding.

T2D association signals are differentially enriched for ancestry-correlated heterogeneity across mechanistic clusters. To understand better the impact of genetic diversity on differences in allelic effects between GWAS at T2D association signals, we assessed the contribution of each of the three axes of genetic variation to heterogeneity (**Methods**). For 118 (92.9%) of the 127 association signals with significant evidence of ancestry-correlated heterogeneity, allelic effect sizes were most strongly associated with the first two axes of genetic variation (**Extended Data Figure 1, Supplementary Table 16**). This may simply reflect greater power to detect heterogeneity because these two axes separate GWAS from the three ancestry groups (AFA, EAS, and EUR) that make the largest contributions to the

effective sample size of the multi-ancestry meta-analysis. The magnitude and direction of the association of index SNVs with these two axes reflected differences in allelic effect size between AFA/EUR and EAS GWAS on the AFA-EAS axis, and AFA/EAS and EUR GWAS on the AFA-EUR axis (**Extended Data Figure 6**). For example, the T2D association signal indexed by rs7766070 at the *CDKAL1* locus was positively associated with the AFA-EAS axis (P=4.2x10^{-14}), but not the AFA-EUR axis (P=0.74) and is therefore characterised by a larger allelic effect in EAS GWAS than in AFA and EUR GWAS. On the other hand, at the locus encompassing *CILP2*, *CRTC1*, and *TM6SF2*, the T2D association signal indexed by rs8107974 has a larger allelic effect in EUR GWAS than in AFA and EAS GWAS, consistent with a positive association with the AFA-EUR axis (P=3.7x10^{-10}), but not the AFA-EAS axis (P=0.72).

The most significant evidence of ancestry-correlated heterogeneity was observed for the T2D association signal at the *HNF1A* locus indexed by rs1169299 (P_{HET}=4.8x10^{-35}). This index SNV was negatively associated with the AFA-EAS axis (P_{HET}=2.7x10^{-11}), and positively associated with the AFA-EUR axis (P_{HET}=4.6x10^{-9}), corresponding to an AFA allelic effect (OR=1.02) that was intermediate between the EAS and EUR allelic effects (OR=0.95 and OR=1.05, respectively). In contrast, the association signal indexed by rs2237884, at the locus encompassing *INS*, *IGF2*, and *KCNQ1*, was not associated with either the AFA-EAS axis (P_{HET}=0.61) or AFA-EUR axis (P_{HET}=0.56), indicating no difference in allelic effects between AFA, EAS, and EUR GWAS (OR=1.03 for all three ancestry groups). Instead, the heterogeneity for this signal was driven by association with the third axis of genetic variation (P_{HET}=2.8x10^{-8}), which separates HIS and SAS GWAS (OR=1.09 and OR=0.97, respectively).

We investigated whether the observed ancestry-correlated differences in allelic effects on T2D between ancestry groups varied across mechanistic clusters. To do this, we compared the magnitude and direction of association of index SNVs in each cluster with the first three axes of genetic variation (**Methods**). We observed significant differences in mean Z-scores for association between clusters for both the AFA-EAS axis (P=4.1x10^{-6}) and the AFA-EUR axis (P=1.5x10^{-6}), but not for the HIS-SAS axis (P=0.17), reflecting at least in part differences in sample size and therefore statistical power. Index SNVs in the two beta-cell clusters were most positively associated with the AFR-EAS axis, indicating allelic effects on T2D that were greater in EAS than in AFA and EUR GWAS (**Extended Data Figure 7, Supplementary Table 17**). In contrast, index SNVs in the lipodystrophy and obesity clusters were most positively associated with the AFA-EUR axis, indicating allelic effects on T2D that were greater in EUR GWAS than in EAS/AFA GWAS.

Impact of BMI on ancestry-correlated heterogeneity between GWAS. To investigate the impact of ancestry-correlated heterogeneity in allelic effects between GWAS, we extended the MR-MEGA meta-regression model to account for mean BMI in T2D cases and controls, in addition to axes of genetic variation (**Methods**). After adjustment for study-level mean BMI in T2D cases and in controls, only 24 association signals retained significant evidence of ancestry-correlated heterogeneity (P<3.9x10^{-5}), compared with 127 signals without adjustment (**Supplementary Table 18**). For example, at the *HNF1A* locus, the ancestry-correlated heterogeneity at the T2D association indexed by rs1169299 was attenuated after BMI adjustment (P=0.00016 versus P=4.8x10^{-35} without adjustment), which is consistent with the assignment of this signal to the beta-cell -PI cluster. In contrast, at the association signal indexed by rs2237884, at the locus encompassing *INS*, *IGF2*, and *KCNQ1*, which was assigned to the body fat cluster, ancestry-correlated heterogeneity was not meaningfully impacted by BMI adjustment (P=5.0x10^{-7} versus P=2.7x10^{-7} without adjustment). After

adjustment for BMI, significant differences in mean Z-scores for association between clusters for the AFA-EUR axis were maintained (P=3.2x10^{-5} versus P=1.5x10^{-6} without adjustment), whilst those for the AFA-EAS axis were not (P=0.18 versus P=4.1x10^{-6} without adjustment). Furthermore, after adjustment for BMI, the two beta-cell clusters were no longer strongly positively associated with the AFA-EAS axis (**Extended Data Figure 7, Supplementary Table 19**).

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Supplementary Methods

Cluster-specific associations of index SNVs with insulin-related endophenotypes and insulin resistance-related disorders. We extracted association summary statistics for measures of glucose homeostasis derived from hyperinsulinemic-euglycemic clamp assessments and oral glucose tolerance tests (OGTT) performed by the GUARDIAN Consortium[1], which were obtained from GWAS undertaken in up to 1,316 non-diabetic Mexican American participants from the Mexican American Coronary Artery Disease (MACAD) study[2] and the Hypertension and Insulin Resistance (HTN-IR) study[3]. The measures used were: insulin sensitivity (clamp-derived glucose infusion rate in 1,316 participants from MACAD and HTN-IR); insulin clearance (clamp-derived metabolic clearance rate of insulin in 1,261 participants from MACAD and HTN-IR); and insulin secretion (OGTT-derived area under the curve for insulin normalised for glucose from baseline to 30 minutes in 513 participants from MACAD). We also extracted association summary statistics for homeostatic model assessment measures of beta-cell function (HOMA-B) and insulin resistance (HOMA-IR) from published GWAS meta-analyses of up to 36,466 non-diabetic European ancestry individuals from MAGIC[4]. We also extracted association summary statistics for insulin resistance-related disorders from published GWAS meta-analyses of: (i) 5,485 GDM cases and 347,856 female controls of diverse ancestry from the GenDIP Consortium[5]; and (ii) 10,074 PCOS cases and 103,164 female controls of European ancestry[6].

For each endophenotype/disorder, we aligned the effect estimate to the T2D risk allele from the fixed-effects multi-ancestry meta-analysis, denoted β_j for the jth index SNV. We then calculated the Z-score, given by $Z_j = \beta_j / s_j$, where s_j is the standard error of the effect estimate of the jth index SNV. We tested for association of each endophenotype with index SNVs across clusters in a linear regression model, given by $E(Z_j) = \sum_k \gamma_k C_{jk}$, where C_{jk} is an indicator variable that takes the value "1" if the jth index SNV was assigned to the kth cluster and "0" otherwise. We tested for heterogeneity in cluster effects on each endophenotype by comparing the deviance of this model with that of $E(Z_j) = \gamma_0$. Regression models were fitted using the glm function in R.

Cluster-specific associations of index SNVs with circulating GLP-1 concentrations. The Malmo Diet and Cancer Study (MDCS) is a prospective population-based cohort study that includes 31088 men and women aged 44 to 74 who completed a baseline examination between 1991 and 1996 and lived in Malmo[7]. A random subset was invited to a reinvestigation starting in 2007, where GLP-1 was measured[8]. Individuals with diabetes were excluded from the analysis. An overnight fast was followed by the administration of 75g OGTT for diabetes free individuals. Blood samples were analyzed for GLP-1 concentrations at 0 and 120 minutes. Total plasma GLP-1 concentrations, including intact GLP-1 and the metabolite GLP-1 9-36 amide, were determined radioimmunologically with an in-house anti-serum (no. 89390; sensitivity <1 pmol/l)[9,10].

The Prevalence, Prediction and Prevention of type 2 diabetes (PPP)-Botnia Study is a population-related study that began in 2004 in Finland. Participants were randomly selected from the National Finnish Population Registry, representing 6%-7% of the 18-75 age population. Of the original 5,208 participants, 3,850 (77%) attended the first follow-up study in 2011-2015, where GLP-1 was measured[11]. A 75g OGTT was conducted after overnight 10-12 hours fasting with blood samples drawn at 0, 30, and 120 minutes. GLP-1 was measured at 0 and 120 minutes. GLP-1 was measured using GLP-1 (total) radioimmunoassay (GLP1T-

36HK, EMD Millipore) with high specificity to GLP-1 (GLP-2, glucagon, and exendin <0.2%). The range was 3–333 pmol/l. Serum insulin was measured by an AutoDelfia fluoroimmunometric assay (B080-101, PerkinElmer)[11].

MDCS was genotyped at the Broad genotyping facility using the Infinium OmniExpressExome v1.0 B Beadchip array (Illumina). PPP-Botnia genotyping was performed on a FinnGen ThermoFisher Axiom custom array[12] at the Thermo Fisher genotyping service facility in San Diego. Standard quality control filters were applied to filter SNvs and samples before imputation. SNVs were excluded for monomorphism, low call rate, or Hardy-Weinberg deviation. Samples with duplications or low call rates, unexpected relatives, sex mismatches, heterozygosity outliers, ancestral outliers (non-EUR) were excluded. For MDCS, genotype imputation for autosomal chromosomes was performed using the Haplotype Reference Consortium version 1.0.3 on the Michigan Server. For PPP-Botnia, genotype imputation was carried out using the population-specific SISu v3 reference panel[12] with Beagle 4.1[13]. In both studies, GLP-1 hormone levels were log-transformed before analysis. SNPTEST v.2.5.6[14] was used for genome-wide association analyses, using frequentist score method adjusted for age, sex and first four principal components. The results were filtered based on MAF >0.01, Hardy-Weinberg equilibrium $P>5x10^{-7}$, and imputation info >0.4. A fixed effect meta-analysis (inverse-variance weighting) was performed using GWAMA[15]. The final analysis included 3,514 individuals with fasting GLP-1 and 3,511 individuals with 2-hour GLP-1.

All of Us Research Program (AoURP) cohort description, sequencing, quality control, and phenotype derivation. We considered participants with whole-genome sequencing (WGS) and electronic health record (EHR) data from the AoURP Controlled Tier Dataset v7[16,17]. Details of the generation and quality control of the genomic data can be found in the AoURP Genomic Quality Report release C2022Q4R9 (https://support.researchallofus.org/hc/en-us/article_attachments/17973653017236). Briefly, we used computed genetic ancestries and removed related individuals in the maximal independent set (kinship score >0.1). To reduce the computational burden of the WGS dataset, we considered only high-quality SNVs (as defined in the AoURP Genomic Quality Report release C2022Q4R9) with MAF >1% or MAC >100 in at least one of the computed genetic ancestries. To correct for population structure, within each computed genetic ancestry, we derived principal components using the smartpca function from EIGENSOFT v7.2.1 with the “fastmode” option enabled[18]. In the principal component calculations, we excluded SNVs that were not present in the 1000 Genomes Project (phase 3, October 2014 release) reference panel[19]. We also excluded SNVs with MAF <1%, that deviated from Hardy-Weinberg equilibrium ($P<10^{-6}$), or were located in the major histocompatibility complex and regions of high LD. Subsequently, we extracted autosomal LD-pruned SNVs ($r^2<0.05$) using PLINK v2.0[20]. Cases of T2D, T2D-related macrovascular outcomes, and microvascular complications were derived from the combination of diagnosis codes (ICD-9-CM and ICD-10-CM), drug exposures, and LOINC codes for laboratory test results, extracted from EHR data. Age of T2D onset was defined by age at the first diagnosis code or age at the first drug exposure code.

Derivation of T2D cases and controls. For T2D cases, we used a previously developed method (https://phekb.org/phenotype/type-2-diabetes-mellitus). Briefly, we considered participants as T2D cases if they fit the following criteria: (a) at least one T2D diagnosis code and at least one drug exposure for T2D medications, unless at least one type 1 diabetes (T1D) diagnosis code; (b) at least one T2D diagnosis code, at least two drug exposures for

T1D and T2D medications with a T2D drug exposure occurring at least one day before T1D drug exposure, unless at least one T1D diagnosis code; (c) at least two T2D diagnosis codes and at least one drug exposure for T1D medication, unless at least one T1D diagnosis code; or (d) at least one drug exposure for T2D medications and at least one abnormal laboratory test result (random glucose, fasting glucose, or HbA1c), unless at least one T1D diagnosis codes. For controls, we considered those participants that were free of all diabetes diagnosis codes, including T2D, T1D, and other forms of diabetes. Additionally, we excluded participants that matched criteria (d) from the T2D definition. Age of T2D onset was defined by age at the first diagnosis code under criteria (a-c), and by age at the first drug exposure code under criteria (d).

For T2D, we used diagnosis codes 250.00, 250.02, 250.20, 250.22, 250.30, 250.32, 250.40, 250.42, 250.50, 250.52, 250.60, 250.62, 250.70, 250.72, 250.80, 250.82, 250.90, 250.92 from ICD-9-CM and E11.00, E11.01, E11.21, E11.29, E11.311, E11.319, E11.36, E11.39, E11.40, E11.51, E11.618, E11.620, E11.621, E11.622, E11.628, E11.630, E11.638, E11.641, E11.649, E11.65, E11.69, E11.8, E11.9 from ICD-10-CM. For T2D drug exposures, we used the following medications: acarbose, acetohexamide, albiglutide, alogliptin, canagliflozin, chlorpropamide, colesevelam, dapagliflozin, dulaglutide, empagliflozin, exenatide, glimepiride, glipizide, glyburide, linagliptin, liraglutide, lixisenatide, metformin, miglitol, nateglinide, pioglitazone, repaglinide, rosiglitazone, saxagliptin, semaglutide, sitagliptin, tolazamide, and troglitazone. Finally, we considered the following abnormal lab results: random glucose (LOINC codes: 2339-0, 2345-7) > 200mg/dl, fasting glucose (LOINC code: 1558-6) ≥ 125mg/dl, and HbA1c (LOINC codes: 4548-4, 17856-6, 4549-2, 17855-8) ≥ 6.5%. For T1D, we used diagnosis codes 250.01, 250.03, 250.11, 250.13, 250.21, 250.23, 250.31, 250.33, 250.41, 250.43, 250.51, 250.53, 250.61, 250.63, 250.71, 250.73, 250.81, 250.83, 250.91, 250.93 from ICD-9-CM and E10.10, E10.11, E10.21, E10.29, E10.311, E10.319, E10.36, E10.39, E10.40, E10.51, E10.618, E10.620, E10.621, E10.622, E10.628, E10.630, E10.638, E10.641, E10.649, E10.65, E10.69, E10.8, E10.9 from ICD-10-CM. For T1D drug exposures, we used the following medications: insulin, insulin NPH, insulin aspart, insulin degludec, insulin detemir, insulin glargine, insulin glulisine, insulin lispro, pramlintide. For other forms of diabetes, we used diagnosis codes 249*, 648.0*, 648.8* in ICD-9-CM and E08*, E09*, E13*, O24* in ICD-10-CM.

Derivation of cases and controls for T2D-related clinical outcomes. For each T2D-related clinical outcome, we used previously-defined ICD-9-CM and ICD-10-CM diagnosis codes from EHR data to identify cases and controls[21-24]. For macrovascular outcomes (CAD, ischemic stroke, and peripheral artery disease), we defined cases and controls as participants with and without, respectively, the relevant diagnosis codes, irrespective of T2D status. For CAD, we used 410*, 411*, 412*, 413* in ICD-9-CM and I20*, I21*, I22*, I23*, I24*, I25* in ICD-10-CM. For ischemic stroke, we used 433*, 434* in ICD-9-CM and I63* in ICD-10-CM. For peripheral artery disease, we used 4400, 4402, 4438, 4439 in ICD-9-CM and I70.0, I70.00, I70.01, I70.2, I70.20, I70.21, I70.8, I70.80, I70.9, I70.90, I73.8, I73.9 in ICD-10-CM. For microvascular complications (ESDN and proliferative diabetic retinopathy), we considered only T2D cases. ESDN cases were defined with relevant diagnosis codes for both diabetic nephropathy and end-stage kidney disease (ESKD), and ESDN controls were defined as being free of any diagnosis code for diabetic nephropathy, defined using the AoURP cohort builder. For ESKD, we used 403.01, 403.11, 403.91, 404.02, 404.03, 404.12, 404.13, 404.92, 404.93, 585.6 in ICD-9-CM and I12.0, I13.11, I13.2, N18.6 in ICD-10-CM. For DN, we used E11.21 in ICD-10-CM. Proliferative diabetic retinopathy cases were defined with

relevant diagnosis codes. Proliferative diabetic retinopathy controls were defined as being free of any diagnosis code for diabetic retinopathy. For proliferative diabetic retinopathy, we used 362.02 in ICD-9-CM and E08.35*, E09.35*, E10.35*, E11.35*, E13.35* in ICD-10-CM. For diabetic retinopathy, we used 362.0* in ICD-9-CM and E08.31*, E08.32*, E08.33*, E08.34*, E08.35*, E09.31*, E09.32*, E09.33*, E09.34*, E09.35*, E10.31*, E10.32*, E10.33*, E10.34*, E10.35*, E11.31*, E11.32*, E11.33*, E11.34*, E11.35*, E13.31*, E13.32*, E13.33*, E13.34*, E13.35* in ICD-10-CM.

Biobank Japan (BBJ) cohort description, genotyping, quality control, and phenotype derivation. BBJ is a multi-institutional hospital-based registry that comprises DNA and medical records from individuals of Japanese ancestry[25,26]. The first BBJ cohort comprises approximately 200,000 participants with at least one of 47 common diseases collected between 2003 and 2007. The second BBJ cohort comprises approximately 67,000 participants with at least one of 38 common diseases collected between 2013 and 2017. Physicians of 66 cooperating hospitals determined the eligibility of cases. Only those individuals who were not included in the multi-ancestry meta-analysis were considered for testing of the partitioned GRS.

Genomic DNA was prepared following standard protocols from peripheral blood samples and genotyped using the Illumina Asian Screening Array, following the manufacturer's instructions. We excluded individuals with call rate <98% and outliers from the cluster of East Asian populations based on principal component analysis with reference individuals from Phase II HapMap[27]. We excluded SNVs with call rate <99%, MAC <5, exact Hardy-Weinberg equilibrium $P<10^{-10}$, and >5% difference in MAF when compared with Japanese whole-genome sequence data[28,29] and the Tohoku Medical Megabank Project[30]. After quality control, we performed pre-phasing using SHAPEIT4[31]. Phased haplotypes were imputed to the combined reference panel of 1000 Genomes Project Phase 3 and Japanese whole-genome sequencing data from 1,037 individuals[28,29] using Minimac4[32]. We subsequently excluded individuals with a mismatch between inferred genetic sex and sex registered in clinical information, who were not in a set of unrelated individuals defined by using PLINK with KING-cutoff <0.09375, or were outliers of heterozygosity rates (more than 5 SD from the mean). To correct for population structure, we derived principal components using PLINKv2.0[20], calculated from a set of autosomal LD-pruned SNVs ($r^2<0.1$) with MAF ≥0.5% after excluding the major histocompatibility complex region.

We selected participants of at least 18 years of age for PS analyses. We defined T2D cases as participants with a diagnosis of T2D, made by physicians at participating hospitals, but not type 1 diabetes, mitochondrial diabetes, maturity-onset diabetes of the young, or any other type of diabetes[33]. We extracted cases of microvascular complications from medical records in which diagnosis was made by physicians at participating hospitals. We defined controls for microvascular complications as T2D cases without any diagnosis of diabetic nephropathy or diabetic retinopathy. We defined CAD as a composite of stable angina, unstable angina, and myocardial infarction. These conditions, in addition to ischemic stroke and peripheral artery disease, were diagnosed by physicians at collaborating hospitals based on general medical practices following relevant guidelines. Age of T2D onset was defined from a questionnaire of medical history.

Genes & Health (G&H) cohort description, genotyping, quality control, and phenotype derivation. G&H is a UK-based cohort of British Pakistani and Bangladeshi individuals

recruited and consented for lifelong electronic health record access and genetic analysis[34]. Medical records are linked to ICD-10-CM, OPCS and SNOMED diagnosis and procedural codes across inpatient and hospital settings as well as clinical laboratory measurements, and a baseline questionnaire containing demographic information. Individuals were genotyped using the Illumina Infinium Global Screening Array. Full details of quality control have been reported previously[35]. KING was used to calculate kinship metrics[36] and individuals with at least second-degree relatedness were subsequently removed. Ancestry outliers based on principal component analysis were also excluded. Individuals were imputed to the TOPMed r2 reference panel[37]. Cases of T2D, T2D-related macrovascular outcomes, and microvascular complications were derived from the combination of diagnosis codes (ICD-10-CM), drug exposures, and laboratory test results, extracted from EHR data. Age of T2D onset was defined as the date a diagnosis was made (ICD-10-CM), or a medication was prescribed, or an abnormal laboratory test was recorded, whichever occurred first.

Derivation of T2D cases and controls. We considered participants as T2D cases if they fit the following criteria: (a) at least one T2D diagnosis code and at least one drug exposure for T2D medications, unless at least one type 1 diabetes (T1D) diagnosis code; (b) at least one T2D diagnosis code, at least two drug exposures for T1D and T2D medications with a T2D drug exposure occurring at least one day before T1D drug exposure, unless at least one T1D diagnosis code; (c) at least two T2D diagnosis codes and at least one drug exposure for T1D medication, unless at least one T1D diagnosis code; or (d) at least one drug exposures for T2D medications and at least one abnormal laboratory test result (random glucose, fasting glucose, or HbA1c), unless at least one T1D diagnosis codes. For controls, we considered those participants that were free of all diabetes diagnosis codes, including T2D, T1D, and other forms of diabetes. Additionally, we excluded participants that matched criteria (d) from the T2D definition.

For T2D, we used diagnosis codes E11.00, E11.01, E11.21, E11.29, E11.311, E11.319, E11.36, E11.39, E11.40, E11.51, E11.618, E11.620, E11.621, E11.622, E11.628, E11.630, E11.638, E11.641, E11.649, E11.65, E11.69, E11.8, E11.9 from ICD-10-CM. For T2D drug exposures, we used the following medications: acarbose, acetohexamide, albiglutide, alogliptin, canagliflozin, chlorpropamide, colesevelam, dapagliflozin, dulaglutide, empagliflozin, exenatide, glimepiride, glipizide, glyburide, linagliptin, liraglutide, lixisenatide, metformin, miglitol, nateglinide, pioglitazone, repaglinide, rosiglitazone, saxagliptin, semaglutide, sitagliptin, tolazamide, and troglitazone. Finally, we considered the following abnormal lab results: random glucose > 200mg/dl, fasting glucose ≥ 125mg/dl, and HbA1c ≥ 6.5%. For T1D, we used diagnosis codes E10.10, E10.11, E10.21, E10.29, E10.311, E10.319, E10.36, E10.39, E10.40, E10.51, E10.618, E10.620, E10.621, E10.622, E10.628, E10.630, E10.638, E10.641, E10.649, E10.65, E10.69, E10.8, E10.9 from ICD-10-CM. For T1D drug exposures, we used the following medications: insulin, insulin NPH, insulin aspart, insulin degludec, insulin detemir, insulin glargine, insulin glulisine, insulin lispro, pramlintide. For other forms of diabetes, we used diagnosis codes E08*, E09*, E13*, O24* in ICD-10-CM.

Derivation of cases and controls for T2D-related clinical outcomes. For macrovascular outcomes (CAD, ischemic stroke, and peripheral artery disease), we defined cases and controls as participants with and without, respectively, the relevant diagnosis codes, irrespective of T2D status. For CAD, we used I20*, I21*, I22*, I23*, I24*, I25* in ICD-10-CM. For ischemic stroke, we used I63* in ICD-10-CM. For peripheral artery disease, we used I70.0, I70.00, I70.01, I70.2, I70.20, I70.21, I70.8, I70.80, I70.9, I70.90, I73.8, I73.9 in ICD-10-CM. For microvascular complications (ESDN and proliferative diabetic retinopathy), we

considered only T2D cases. ESDN cases were defined with relevant diagnosis codes for both diabetic nephropathy and end-stage kidney disease (ESKD), and ESDN controls were defined as being free of any diagnosis code for diabetic nephropathy. For ESKD, we used I12.0, I13.11, I13.2, N18.6 in ICD-10-CM. For DN, we used E11.21 in ICD-10-CM. Proliferative diabetic retinopathy cases were defined with relevant diagnosis codes. Proliferative diabetic retinopathy controls were defined as being free of any diagnosis code for diabetic retinopathy. For proliferative diabetic retinopathy, we used E08.35*, E09.35*, E10.35*, E11.35*, E13.35* in ICD-10-CM. For diabetic retinopathy, we used E08.31*, E08.32*, E08.33*, E08.34*, E08.35*, E09.31*, E09.32*, E09.33*, E09.34*, E09.35*, E10.31*, E10.32*, E10.33*, E10.34*, E10.35*, E11.31*, E11.32*, E11.33*, E11.34*, E11.35*, E13.31*, E13.32*, E13.33*, E13.34*, E13.35* in ICD-10-CM. No cases with proliferative diabetic retinopathy were identified in the G&H cohort.

Clinical trials from the Thrombolysis in Myocardial Infarction (TIMI) Study. ENGAGE AF-TIMI 48 was a 3-arm trial comparing two doses of the Factor Xa inhibitor edoxaban to warfarin in patients with atrial fibrillation and CHADS2 risk score of 2 or higher, where co-morbidities included diabetes (38%), stroke (28%), and heart failure (57%). SOLID-TIMI 52 was a trial of the lipoprotein-associated phospholipase A2 inhibitor darapladib versus placebo in patients with recent acute coronary syndrome on optimal background medical therapy, where co-morbidities included hypertension (73%), hyperlipidemia (64%), and diabetes (35%). SAVOR-TIMI 53 was a trial of the DPP4 inhibitor saxagliptin in patients with T2D, where co-morbidities included atherosclerosis (78%) and hypertension (81%). PEGASUS-TIMI 54 was a trial of the antiplatelet drug ticagrelor in patients with prior myocardial infarction, where co-morbidities included smoking (17%), hypertension (78%), diabetes (32%), prior percutaneous coronary intervention (83%), and prior coronary artery bypass graft (5%). FOURIER-TIMI 59 was a trial of the PCSK9 inhibitor evolocumab in patients with myocardial infarction, stroke, or peripheral artery disease, where co-morbidities included hypertension (80%), diabetes (37%), and prior myocardial infarction (81%). DECLARE-TIMI 58 was a trial of the SGLT-2 inhibitor dapaglifozin in patients with T2D, where co-morbidities included established atherosclerotic cardiovascular disease (40%) or multiple risk factors for atherosclerotic cardiovascular disease (60%).

Genotyping was performed on the Infinium Global Array chip (FOURIER-TIMI 59), Affymetrix Biobank Array (SOLID-TIMI 52), Infinium Global Screening Array MD (DECLARE-TIMI 58) and Illumina Multi-Ethnic Genotyping Array (ENGAGE AF-TIMI 48, PEGASUS-TIMI 54 and SAVOR-TIMI 53). PLINK v2.0[20] was used for pre-imputation quality control, which included mapping to hg38 coordinates, removing SNVs and individuals with missingness >0.2 (first round) and >0.02 (second round), removing individuals with sex discrepancies based on X-chromosome F-values (<0.2 for females and >0.8 for males) and heterozygosity more than 3 SD from the mean, and removing SNVs with MAF <1% and extreme deviation from Hardy-Weinberg equilibrium ($P<10^{-6}$). Imputation was performed on the Michigan Imputation Server using Eagle v2.4[38] for phasing and Minimac4[32] on TOPMed Freeze 5 reference panel[37] with imputation quality filter $r^2>0.3$. Cryptic relatedness was assessed using identity by descent, and a pi-hat threshold of 0.2 was used to identify related samples. EUR individuals were identified using the 1000 Genomes phase 3 v5 reference panel and the ADMIXTURE tool[39] (cutoff for European ancestry was set at 0.8) and were retained for analysis.

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Acknowledgements and Funding

Anti-aging Study Cohort (AASC) is supported by the Grant-in-Aid for Scientific Research (20018020, 19659163, 20390185, 23659382, 24390084, 23659352, 25293141, 26670313, 17H04123) from the Ministry of Education, Culture, Sports, Science and Technology of Japan, research grant from the Japan Atherosclerosis Prevention Found, National Cardiovascular Research Grants, and Research Promotion Award from Ehime University.

All Of Us Research Program (AOURP) is supported by the National Institutes of Health, Office of the Director: Regional Medical Centers: 1 OT2 OD026549; 1 OT2 OD026554; 1 OT2 OD026557; 1 OT2 OD026556; 1 OT2 OD026550; 1 OT2 OD 026552; 1 OT2 OD026553; 1 OT2 OD026548; 1 OT2 OD026551; 1 OT2 OD026555; IAA #: AOD 16037; Federally Qualified Health Centers: HHSN 263201600085U; Data and Research Center: 5 U2C OD023196; Biobank: 1 U24 OD023121; The Participant Center: U24 OD023176; Participant Technology Systems Center: 1 U24 OD023163; Communications and Engagement: 3 OT2 OD023205; 3 OT2 OD023206; and Community Partners: 1 OT2 OD025277; 3 OT2 OD025315; 1 OT2 OD025337; 1 OT2 OD025276. In addition, the All of Us Research Program would not be possible without the partnership of its participants.

Atherosclerosis Risk in Communities (ARIC) study has been funded in whole or in part with Federal funds from the National Heart, Lung, and Blood Institute, National Institutes of Health, Department of Health and Human Services (contract numbers HHSN268201700001I, HHSN268201700002I, HHSN268201700003I, HHSN268201700004I and HHSN268201700005I), R01HL087641, R01HL059367 and R01HL086694; National Human Genome Research Institute contract U01HG004402; and National Institutes of Health contract HHSN268200625226C. The authors thank the staff and participants of the ARIC study for their important contributions. Infrastructure was partly supported by Grant Number UL1RR025005, a component of the National Institutes of Health and NIH Roadmap for Medical Research.

BioBank Japan (BBJ). This study was funded by the BioBank Japan project, which is supported by the Ministry of Education, Culture, Sports, Sciences and Technology (MEXT) of Japanese government and the Japan Agency for Medical Research and Development (AMED, grant ID JP21km0605001). AMED GRIFIN Diabetes Initiative Japan was supported by Japan Agency for Medical Research and Development (JP20km0405202, JP21tm0424218). Scarda was supported by AMED under Grant Number 223fa627011.

Beijing Eye Study (BES) was supported by National Natural Science Foundation of China (grant 81570835).

BioMe Biobank (BIOME) is supported by The Andrea and Charles Bronfman Philanthropies and in part by funding of the NIH (U01HG007417; R56HG010297; X01HL134588). BIOME thanks all participants in the Mount Sinai Biobank, and also thanks all the recruiters who have assisted and continue to assist in data collection and management. BIOME is grateful for the computational resources and staff expertise provided by Scientific Computing at the Icahn School of Medicine at Mount Sinai.

Vanderbilt University Medical Center's BioVU (BIOVU) projects are supported by numerous sources: institutional funding, private agencies, and federal grants. These include NIH funded Shared Instrumentation Grant S10OD017985, S10RR025141, and S10OD025092; CTSA grants UL1TR002243, UL1TR000445, and UL1RR024975. Genomic data are also supported by investigator-led projects that include U01HG004798, R01NS032830, RC2GM092618, P50GM115305, U01HG006378, U19HL065962, and R01HD074711. This work was conducted in part using the resources of the Advanced Computing Center for Research and Education at Vanderbilt University, Nashville, TN, supported in part by an S10 instrumentation award (1S10OD023680-01).

Bangladesh Population Cohort (BPC) was supported by US National Institute of Environmental Health Sciences Grants P42 ES10349 and P30 ES09089.

Cardiometabolic Genome Epidemiology (CAGE-AMAGASKI and CAGE-GWAS) was supported by grants for the Core Research for Evolutional Science and Technology (CREST) from the Japan Science Technology Agency; KAKENHI (Grant-in-Aid for Scientific Research) from the Ministry of Education, Culture, Sports, Science and Technology of Japan; and the Grant and research budget of National Center for Global Health and Medicine (NCGM). CAGE-AMAGASKI thanks Drs. Toshio Ogihara, Yukio Yamori, Akihiro Fujioka, Chikanori Makibayashi, Sekiharu Katsuya, Ken Sugimoto, Kei Kamide, and Ryuichi Morishita and the many physicians of the participating hospitals and medical institutions in Amagasaki Medical Association for their assistance in collecting the DNA samples and accompanying clinical information.

Cardiometabolic Genome Epidemiology Kita-Nagoya Genomic Epidemiology (CAGE-KING) was supported in part by Grants-in-Aid from MEXT (nos. 24390169, 16H05250, 15K19242, 16H06277) as well as by a grant from the Funding Program for Next-Generation World-Leading Researchers (NEXT Program, no. LS056).

Coronary Artery Risk Development in Young Adults (CARDIA) was conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the University of Alabama at Birmingham (HHSN268201800005I & HHSN268201800007I), Northwestern University (HHSN268201800003I), University of Minnesota (HHSN268201800006I), and Kaiser Foundation Research Institute (HHSN268201800004I). CARDIA was also partially supported by the Intramural Research Program of the National Institute on Aging (NIA) and an intra-agency agreement between NIA and NHLBI (AG0005). Genotyping was funded as part of the NHLBI Candidate-gene Association Resource (N01-HC-65226) and the NHGRI Gene Environment Association Studies (GENEVA) (U01-HG004729, U01-HG04424, and U01-HG004446).

Cleveland Family Study (CFS) is supported by grants to Case Western Reserve University (NIH HL 46380, M01RR00080) and Brigham and Women's Hospital (K01-HL135405-01, R01-HL113338-04, R35-HL135818-01, 5-R01-HL046380-15 and 5-KL2-RR024990-05).

China Health and Nutrition Survey (CHNS) was supported by: the National Institute for Nutrition and Health, the Chinese Center for Disease Control and Prevention; the National Institutes of Health (R01AG065357, R01HD30880, R01HL108427 and R01DK104371); the

Fogarty International Center of the National Institutes of Health (TW009077); the China-Japan Friendship Hospital, the Beijing Municipal Center for Disease Prevention and Control, the China National Health Commission (formerly the Chinese Ministry of Health); the Chinese National Human Genome Center at Shanghai; and the Carolina Population Center (P2CHD050924), The University of North Carolina at Chapel Hill.

Cardiovascular Health Study (CHS) was supported by NHLBI contracts HHSN268201200036C, HHSN268200800007C, HHSN268201800001C, N01HC55222, N01HC85079, N01HC85080, N01HC85081, N01HC85082, N01HC85083, N01HC85086, 75N92021D00006; and NHLBI grants U01HL080295, R01HL085251, R01HL087652, R01HL105756, R01HL103612, R01HL120393, and R01HL130114 with additional contribution from the National Institute of Neurological Disorders and Stroke (NINDS). Additional support was provided through R01AG023629 from the National Institute on Aging (NIA). A full list of principal CHS investigators and institutions can be found at CHS-NHLBI.org. The provision of genotyping data was supported in part by the National Center for Advancing Translational Sciences, CTSI grant UL1-TR-001881, and the National Institute of Diabetes and Digestive and Kidney Disease Diabetes Research Center (DRC) grant DK063491 to the Southern California Diabetes Endocrinology Research Center. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

China Kadoorie Biobank (CKB) chiefly acknowledges the participants, project staff, and the China National Centre for Disease Control and Prevention (CDC) and its regional offices. China's National Health Insurance provides electronic linkage to all hospital treatment. Funding sources: Baseline survey and first re-survey - Kadoorie Charitable Foundation, Hong Kong; long-term follow-up - UK Wellcome Trust (212946/Z/18/Z, 202922/Z/16/Z, 104085/Z/14/Z, 088158/Z/09/Z), National Natural Science Foundation of China (82192901, 82192904, 82192900), and National Key Research and Development Program of China (2016YFC 0900500, 0900501, 0900504, 1303904); DNA extraction and genotyping – GlaxoSmithKline, and the UK Medical Research Council (MC-PC-13049, MC-PC-14135); core funding for the project to the Clinical Trial Service Unit and Epidemiological Studies Unit at Oxford University - British Heart Foundation (CH/1996001/9454), UK MRC (MC-UU-00017/1, MC-UU-12026/2, MC_U137686851), and Cancer Research UK (C16077/A29186, C500/A16896).

Cebu Longitudinal Health and Nutrition Survey (CLHNS) was supported by: US National Institutes of Health grants DK078150, TW005596 and HL085144; pilot funds from RR020649, ES010126, and DK056350; and the Office of Population Studies Foundation.

Diabetic Cohort and Singapore Prospective Study Program (DC/SP2) was supported by the individual research grant and clinician scientist award schemes from the National Medical Research Council (NMRC) and the Biomedical Research Council (BMRC) of Singapore, Ministry of Health, Singapore, National University of Singapore and National University Health System, Singapore.

Durban Diabetes Study and Durban Diabetes Case Control (DDS/DCC) was supported by: the Wellcome Trust (grant number 098051); the African Partnership for Chronic Disease

Research (Medical Research Council UK partnership grant number MR/K013491/1); the National Institute for Health Research Cambridge Biomedical Research Centre (UK); Novo-Nordisk (South Africa); Sanofi-Aventis (South Africa); MSD Pharmaceuticals (Pty) Ltd (Southern Africa); Servier Laboratories (South Africa); South African Sugar Association; and the Victor Daitz Foundation.

deCODE genetics (DECODE) thank the participants in the deCODE study, the staff at deCODE genetics core facilities and the staff at the Research Service Center for their contribution to this work.

Diabetes Gene Discovery Group (DGDG) was supported by Genome Canada, Génome Québec, the Canada Foundation for Innovation, the French Government (“Agence Nationale de la Recherche”), the French Region of "Nord Pas De Calais" ("Contrat de Projets État-Région"), and the charities: “Association Française des Diabétiques”, “Programme National de Recherche sur le Diabète” and “Association de Langue Française pour l'Etude du Diabète et des Maladies Métaboliques”. This study was also supported in part by a grant from the European Union (Integrated Project EuroDia LSHM-CT-2006-518153in the Framework Programme 6 [FP6] of the European Community). This work was supported by grants from the French National Research Agency (ANR-10-LABX-46 [European Genomics Institute for Diabetes] and ANR-10-EQPX-07-01 [LIGAN-PM]). Case and control recruitment was supported by the Fédération Française des Diabetiques, INSERM, CNAMTS, Centre Hospitalier Universitaire Poitiers, La Fondation de France, and the Endocrinology-Diabetology department of the Corbeil-Essonnes Hospital. C. Petit, J.-P. Riveline, and S. Franc were instrumental in recruitment and S. Brunet, F. Bacot, R. Frechette, V. Catudal, M. Deweirder, F. Allegaert, P. Laflamme, P. Lepage, W. Astle, M. Leboeuf, and S. Leroux provided technical assistance. K. Shazand and N. Foisset provided organizational guidance. The D.E.S.I.R. study, which mostly contributed controls, was supported by CNAMTS, Lilly, Novartis Pharma and Sanofi-Aventis, by INSERM (“Réseaux en Santé Publique, Interactions entre les déterminants de la santé”), by “Association Diabète Risque Vasculaire”, “Fédération Française de Cardiologie”, “Fondation de France”, ALFEDIAM, ONIVINS, Ardix Medical, Bayer Diagnostics, Becton Dickinson, Cardionics, Merck Santé, Novo Nordisk, Pierre Fabre, Roche, Topcon. The D.E.S.I.R. Study Group: INSERM U780: B. Balkau, P. Ducimetière, E. Eschwège; INSERM U367: F. Alhenc-Gelas; CHU D'Angers: Y. Gallois, A. Girault; Bichat Hospital: F. Fumeron, M. Marre; Medical Examination Services: Alençon, Angers, Caen, Chateauroux, Cholet, Le Mans, and Tours; Research Institute for General Medicine: J. Cogneau; General practitioners of the region; Cross-Regional Institute for Health: C. Born, E. Caces, M. Cailleau, J. G. Moreau, F. Rakotozafy, J. Tichet, S. Vol. DGDG thank M. Deweider and F. Allegaert for the DNA bank management and are sincerely indebted to all study participants.

Diabetes Genetics Initiative (DGI) was supported by the Novartis Institute for BioMedical Research with additional support from The Richard and Susan Smith Family Foundation and American Diabetes Association Pinnacle Program Project Award. The Botnia Study (study subject cohort) was financially supported by the Folkhalsan Research Foundation, the Sigrid Juselius Foundation, Nordic Center of Excellence in Disease Genetics, EU (EXGENESIS), The Academy of Finland, University of Helsinki, Finnish Diabetes Research Foundation, Foundation for Life and Health in Finland, Finnish Medical Society, Helsinki University Central Hospital Research Foundation, Perklén Foundation, Ollqvist Foundation, Närpes Health Care

Foundation, Municipal Heath Care Center and Hospital in Jakobstad and Health Care Centers in Vasa, Närpes and Korsholm. The work in Malmö, Sweden, was also funded by a Linné grant from the Swedish Research Council (349-2006-237). The contribution of the Botnia and Skara research teams is gratefully acknowledged.

Electronic Medical Records and Genomics Network (EMERGE) was initiated and funded by NHGRI through the following grants: U01HG006828 (Cincinnati Children's Hospital Medical Center/Boston Children's Hospital); U01HG006830 (Children's Hospital of Philadelphia); U01HG006389 (Essentia Institute of Rural Health, Marshfield Clinic Research Foundation and Pennsylvania State University); U01HG006382 (Geisinger Clinic); U01HG006375 (Group Health Cooperative/University of Washington); U01HG006379 (Mayo Clinic); U01HG006380 (Icahn School of Medicine at Mount Sinai); U01HG006388 (Northwestern University); U01HG006378 (Vanderbilt University Medical Center); and U01HG006385 (Vanderbilt University Medical Center serving as the Coordinating Center). The Northwestern University Enterprise Data Warehouse was funded in part by a grant from the National Center for Research Resources, UL1RR025741. Part of the dataset(s) used for the analyses described were obtained from Vanderbilt University Medical Center's BioVU which is supported by institutional funding and by the Vanderbilt CTSA grant UL1 TR000445 from NCATS/NIH. The eMERGE imputed merged Phase I and Phase II dataset was generated by genotyping centers CIDR (U01HG004438) and the Broad Institute (U01HG004424).

European Prospective Investigation into Cancer and Nutrition (EPIC-INTERACT) project (LSHM-CT-2006-037197) is a European-Community funded project under Framework Programme 6. EPIC-INTERACT thank all EPIC participants and staff for their contribution to the study. EPIC-INTERACT thank Nicola Kerrison (MRC Epidemiology Unit, Cambridge) for managing the data for the InterAct Project and staff from the Laboratory Team, Field Epidemiology Team, and Data Functional Group of the MRC Epidemiology Unit in Cambridge, UK, for carrying out sample preparation, DNA provision and quality control, genotyping, and data-handling work. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. GWAS summary statistics from the EPIC-InterAct study are available to download from the Dryad Digital Repository (https://doi.org/10.5061/dryad.qnk98sfcg).

Epidemiologic Study of the Screenees for Diabetes Reduction Assessment with Ramipril and Rosiglitazone Medication (EPIDREAM) was funded by a grant from the Canadian Institutes of Health Research University Industry competition with partner funding from the GlaxoSmithKline and Sanofi Aventis Global, Sanofi Aventis Canada, Genome Quebec Innovation Centre, Heart and Stroke Foundation of Canada.

Estonian Biobank (ESTBB) was funded by the Estonian Research Council Grant IUT20-60, IUT24-6, PRG687, and the European Union through the European Regional Development Fund Project No. 2014-2020.4.01.15-0012 GENTRANSMED.

Family Heart Study (FAMHS) was supported by NIH grants R01-HL-087700 and R01-HL-088215 from NHLBI, and R01-DK-089256 and R01-DK-075681 from NIDDK.

Framingham Heart Study (FHS) was conducted and supported by the National Heart, Lung and Blood Institute (NHLBI) in collaboration with Boston University (contracts 75N92019D00031, HHSN268201500001I and N01-HC-25195), and its contract with Affymetrix, Inc for genotyping services (contract number N02-HL-6-4278). The analyses reflect intellectual input and resource development from the Framingham Heart Study investigators participating in the SNP Health Association Resource (SHARe) project. FHS was also supported by: NHLBI R01 HL105756, National Institute for Diabetes and Digestive and Kidney Diseases (NIDDK) R01 DK078616, U01 DK078616, NIDDK K24 DK080140 and American Diabetes Association Mentor-Based Postdoctoral Fellowship Award #7-09-MN-32 (to J.B.M.); and NIDDK K24 DK110550 (to J.C.F.).

Finland-United States Investigation of NIDDM Genetics (FUSION) was supported by DK093757, DK072193, DK062370, and ZIA-HG000024.

Genes & Health (G&H) is/has recently been core-funded by Wellcome (WT102627, WT210561), the Medical Research Council (UK) (M009017, MR/X009777/1, MR/X009920/1), Higher Education Funding Council for England Catalyst, Barts Charity (845/1796), Health Data Research UK (for London substantive site), and research delivery support from the NHS National Institute for Health Research Clinical Research Network (North Thames). Genes & Health is/has recently been funded by Alnylam Pharmaceuticals, Genomics PLC; and a Life Sciences Industry Consortium of Astra Zeneca PLC, Bristol-Myers Squibb Company, GlaxoSmithKline Research and Development Limited, Maze Therapeutics Inc, Merck Sharp & Dohme LLC, Novo Nordisk A/S, Pfizer Inc, Takeda Development Centre Americas Inc. We thank Social Action for Health, Centre of The Cell, members of our Community Advisory Group, and staff who have recruited and collected data from volunteers. We thank the NIHR National Biosample Centre (UK Biocentre), the Social Genetic & Developmental Psychiatry Centre (King's College London), Wellcome Sanger Institute, and Broad Institute for sample processing, genotyping, sequencing and variant annotation. We thank: Barts Health NHS Trust, NHS Clinical Commissioning Groups (City and Hackney, Waltham Forest, Tower Hamlets, Newham, Redbridge, Havering, Barking and Dagenham), East London NHS Foundation Trust, Bradford Teaching Hospitals NHS Foundation Trust, Public Health England (especially David Wyllie), Discovery Data Service/Endeavour Health Charitable Trust (especially David Stables), Voror Health Technologies Ltd (especially Sophie Don), NHS England (for what was NHS Digital) - for GDPR-compliant data sharing backed by individual written informed consent. Most of all we thank all of the volunteers participating in Genes & Health.

German Chronic Kidney Disease (GCKD) was funded by the German Ministry of Research and Education (Bundesminsterium für Bildung und Forschung, BMBF) and by the Foundation KfH Stiftung Präventivmedizin. Unregistered grants to support the study were provided by Bayer, Fresenius Medical Care and Amgen. Genotyping was supported by Bayer AG.

Genetic Study of Atherosclerosis Risk (GENESTAR) was supported by NIH grants through the National Heart, Lung, and Blood Institute (HL49762, HL58625, HL59684, HL071025, U01HL72518, and HL087698) and the National Institute of Nursing Research (NR0224103) and by M01-RR000052 to the Johns Hopkins General Clinical Research Center.

Genetic Epidemiology Network of Arteriosclerosis (GENOA) was supported by the National Institutes of Health grant numbers HL054457, HL054464, HL054481, HL087660 and HL119443 from the National Heart, Lung, and Blood Institute. Genotyping was performed at the Mayo Clinic by Stephen Turner, Mariza de Andrade, and Julie Cunningham. GENOA thanks Eric Boerwinkle and Megan Grove from the Human Genetics Center and Institute of Molecular Medicine and Division of Epidemiology, University of Texas Health Science Center, Houston, Texas, USA for their help with genotyping. GENOA also thanks the families that participated in the study.

Resource for Genetic Epidemiology on Adult Heath and Aging (GERA) was supported by a grant (RC2 AG033067; PIs Schaefer and Risch) awarded to the Kaiser Permanente Research Program on Genes, Environment, and Health (RPGEH) and the UCSF Institute for Human Genetics. The RPGEH was supported by grants from the Robert Wood Johnson Foundation, the Wayne and Gladys Valley Foundation, the Ellison Medical Foundation, Kaiser Permanente Northern California, and the Kaiser Permanente National and Northern California Community Benefit Programs.

Genetics of Diabetes and Audit Research in Tayside Scotland (GODARTS) was funded by The Wellcome Trust Study Cohort Functional Genomics Grant (2004-2008, 072960/Z/03/Z) and The Wellcome Trust Scottish Health Informatics Programme (SHIP, 2009-2012, 086113/Z/08/Z).

Genetics of Latinos Diabetic Retinopathy (GOLDR) was supported by grants EY14684 and UL1TR000124.

Genetic Overlap Between Metabolic and Psychiatric Traits and Teens of Attica: Genes and Environment (GOMAP-TEENAGE) was funded by the Wellcome Trust (098051) and was also co-financed by the European Union (European Social Fund - ESF) and Greek national funds through the Operational Program “Education and Lifelong Learning” of the National Strategic Reference Framework (NSRF) - Research Funding Program: Heracleitus II. GOMAP-TEENAGE thanks all study participants and their families, as well as all volunteers for their contribution in this study. GOMAP-TEENAGE is grateful to: Georgia Markou, Laiko General Hospital Diabetes Centre; Maria Emetsidou and Panagiota Fotinopoulou, Hippokratio General Hospital Diabetes Centre; Athina Karabela, Dafni Psychiatric Hospital; Eirini Glezou and Marios Mangioros, Dromokaiteio Psychiatric Hospital; Angela Rentari, Harokopio University of Athens; and Danielle Walker, Wellcome Trust Sanger Institute. GOMAP-TEENAGE thanks the Sample Management and Genotyping Facilities staff at the Wellcome Trust Sanger Institute for sample preparation, quality control and genotyping.

Genomic Research Cohort for CCMB Diabetes Study (GRCCDS) comprises of various cohorts that are supported by: Council of Scientific Industrial Research (CSIR); Ministry of Science and Technology, Govt. of India, India; and Wellcome Trust, London, UK. GRCCDS is grateful to the patients and subjects who voluntarily participated in the study, and thankfully acknowledge other researchers who have supported the study.

Health, Aging and Body Composition Study (HABC) was supported by NIA contracts N01AG62101, N01AG62103, and N01AG62106. The genome-wide association study was

funded by NIA grant 1R01AG032098-01A1 to Wake Forest University Health Sciences and genotyping services were provided by the Center for Inherited Disease Research (CIDR). CIDR is fully funded through a federal contract from the National Institutes of Health to The Johns Hopkins University, contract number HHSN268200782096C. This research was supported in part by the Intramural Research Program of the NIH, National Institute on Aging.

Healthy Aging in Neighborhoods of Diversity Across the Life Span Study (HANDLS) was supported by the Intramural Research Program of the NIH, National Institute on Aging (project Z01-AG000513 and human subjects' protocol 09 AGN248). Data analyses for HANDLS utilized the high-performance computational resources of the Biowulf Linux cluster at the National Institutes of Health, Bethesda, MD (http://hpc.nih.gov).

Hispanic Community Health Study/Study of Latinos (HCHS/SOL) is a collaborative study supported by contracts from the National Heart, Lung, and Blood Institute (NHLBI) to the University of North Carolina (HHSN268201300001I / N01-HC-65233), University of Miami (HHSN268201300004I / N01-HC-65234), Albert Einstein College of Medicine (HHSN268201300002I / N01-HC-65235), University of Illinois at Chicago (HHSN268201300003I / N01-HC-65236 Northwestern Univ), and San Diego State University (HHSN268201300005I / N01-HC-65237). The following Institutes/Centers/Offices have contributed to the HCHS/SOL through a transfer of funds to the NHLBI: National Institute on Minority Health and Health Disparities, National Institute on Deafness and Other Communication Disorders, National Institute of Dental and Craniofacial Research, National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Neurological Disorders and Stroke, NIH Institution-Office of Dietary Supplements. The Genetic Analysis Center at the University of Washington was supported by NHLBI and NIDCR contracts (HHSN268201300005C AM03 and MOD03).

Hong Kong Diabetes Registry (HKDR) acknowledge support from the Theme-based Research Scheme from the Research Grants Council of the Hong Kong Special Administrative Region, China (Project no: T12-402/13-N), the Research Grants Council Research Impact Fund (R4012-18), the Hong Kong Foundation for Research and Development in Diabetes, the Vice-Chancellor One-off Discretionary Fund, the Focused Innovations Scheme, the Postdoctoral Fellowship Scheme of the Chinese University of Hong Kong, and the Croucher Foundation Senior Medical Research Fellowship.

Health Professionals' Follow-Up Study (HPFS) and Nurses Health Study (NHS) acknowledge assistance with data cleaning that 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). Funding support for genotyping, which was performed at the Johns Hopkins University Center for Inherited Disease Research, 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). The datasets used for the analyses described in this manuscript

were obtained from dbGaP at http://www.ncbi.nlm.nih.gov/projects/gap/cgi-bin/study.cgi?study_id=phs000091.v1.p1 through dbGaP accession number phs000091.v1.p.

Mexican American Hypertension and Insulin Resistance (HTNIR) was supported by grant HL0597974.

Howard University Family Study (HUFS) was supported by National Institutes of Health grants S06GM008016-320107 to CNR and S06GM008016-380111 to AA. Participant enrollment was carried out at the Howard University General Clinical Research Center, supported by National Institutes of Health grant 2M01RR010284. Genotyping support was provided by the Coriell Institute for Medical Research. This research was supported by the Intramural Research Program of the Center for Research on Genomics and Global Health (CRGGH). The CRGGH is supported by the National Human Genome Research Institute, the National Institute of Diabetes and Digestive and Kidney Diseases, the Center for Information Technology, and the Office of the Director at the National Institutes of Health (Z01HG200362).

Indian Diabetes Consortium (INDICO) was majorly supported by Council of Scientific and Industrial Research (CSIR), Government of India through CARDIOMED project Grant Number: BSC0122 provided to CSIR-Institute of Genomics and Integrative Biology. INDICO was also partially funded by Department of Science and Technology-PURSE-II (DST/SR/PURSE II/11) given to Jawaharlal Nehru University. INDICO are very much thankful to all the volunteers who have participated in the study.

INTERHEART (INTERHEART) was funded by: the Canadian Institutes of Health Research, the Heart and Stroke Foundation of Ontario, and the International Clinical Epidemiology Network (INCLEN); unrestricted grants from several pharmaceutical companies (with major contributions from AstraZeneca, Novartis, Hoechst Marion Roussel [now Aventis], Knoll Pharmaceuticals [now Abbott], Bristol-Myers Squibb, King Pharma, and Sanofi-Synthelabo); and various national bodies in different countries (see Online Appendix at http://image.thelancet.com/extras/04art8001webappendix2.pdf). Funding sources had no involvement in the study design; in the collection, analysis, and interpretation of data; or the writing of the manuscript.

Jackson Heart Study (JHS) is supported and conducted in collaboration with Jackson State University (HHSN268201800013I), Tougaloo College (HHSN268201800014I), the Mississippi State Department of Health (HHSN268201800015I) and the University of Mississippi Medical Center (HHSN268201800010I, HHSN268201800011I and HHSN268201800012I) contracts from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institute on Minority Health and Health Disparities (NIMHD). The authors also wish to thank the staff and participants of the JHS.

Korean Association Resource (KARE) was supported by grants from Korea Centers for Disease Control and Prevention (4845–301, 4851–302, 4851–307) and intramural grants from the Korea National Institute of Health (2016-NI73001-00, 2019-NG-053-00). KARE was performed with bioresources from National Biobank of Korea, the Centers for Disease Control and Prevention, Republic of Korea.

Korean Biobank Array from the Korean Genome and Epidemiology (KoGES) Consortium (KBA) was supported by grants from Korea Centers for Disease Control and Prevention (4845–301, 4851–302, 4851–307) and intramural grants from the Korea National Institute of Health (2016-NI73001-00, 2019-NG-053-00). KBA was performed with bioresources from National Biobank of Korea, the Centers for Disease Control and Prevention, Republic of Korea. Genotype data were provided by the Collaborative Genome Program for Fostering New Post-Genome Industry (3000-3031b).

Collaborative Health Research in the Region of Augsburg (KORA) research platform was initiated and financed by the Helmholtz Zentrum München – German Research Center for Environmental Health, which is funded by the German Federal Ministry of Education and Research and by the State of Bavaria. Furthermore, KORA research was supported within the Munich Center of Health Sciences (MC Health), Ludwig-Maximilians-Universität, as part of LMUinnovativ and by the German Center for Diabetes Research (DZD).

Los Angeles Latino Eye Study (LALES) acknowledges funding from NEI grant U10EY011753.

London Life Sciences Prospective Population (LOLIPOP) is supported by the National Institute for Health Research (NIHR) Comprehensive Biomedical Research Centre Imperial College Healthcare NHS Trust, the British Heart Foundation (SP/04/002), the Medical Research Council (G0601966, G0700931), the Wellcome Trust (084723/Z/08/Z, 090532 & 098381) the NIHR (RP-PG-0407-10371), the NIHR Official Development Assistance (ODA, award 16/136/68), the European Union FP7 (EpiMigrant, 279143) and H2020 programs (iHealth-T2D, 643774). LOLIPOP acknowledges support of the MRC-PHE Centre for Environment and Health, and the NIHR Health Protection Research Unit on Health Impact of Environmental Hazards. The work was carried out in part at the NIHR/Wellcome Trust Imperial Clinical Research Facility. The views expressed are those of the author(s) and not necessarily those of the Imperial College Healthcare NHS Trust, the NHS, the NIHR or the Department of Health. LOLIPOP thanks the participants and research staff who made the study possible.

Mexican American Study of Coronary Artery Disease (MACAD) was supported by grant HL088457.

Mexico City (MC) was supported, in Mexico, by the Fondo Sectorial de Investigación en Salud y Seguridad Social (SSA/IMSS/ISSSTECONACYT, project 150352), Temas Prioritarios de Salud Instituto Mexicano del Seguro Social (2014-FIS/IMSS/PROT/PRIO/14/34), and the Fundación IMSS. MC thanks Jaime Gómez Zamudio and Araceli Méndez Padrón for technical support. In Canada, computations were performed on the GPC supercomputer at the SciNet HPC Consortium. SciNet is funded by: the Canada Foundation for Innovation under the auspices of Compute Canada; the Government of Ontario; Ontario Research Fund - Research Excellence; and the University of Toronto.

Multi-Ethnic Study of Atherosclerosis (MESA). MESA and the MESA SHARe projects are conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with MESA investigators. Support for MESA is provided by contracts

75N92020D00001, HHSN268201500003I, N01-HC-95159, 75N92020D00005, N01-HC-95160, 75N92020D00002, N01-HC-95161, 75N92020D00003, N01-HC-95162, 75N92020D00006, N01-HC-95163, 75N92020D00004, N01-HC-95164, 75N92020D00007, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168, N01-HC-95169, UL1-TR-000040, UL1-TR-001079, and UL1-TR-001420, UL1TR001881, DK063491, and R01HL105756. Funding for SHARe genotyping was provided by NHLBI Contract N02-HL-64278. Genotyping was performed at Affymetrix (Santa Clara, California, USA) and the Broad Institute of Harvard and MIT (Boston, Massachusetts, USA) using the Affymetrix Genome-Wide Human SNP Array 6.0. The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutes can be found at http://www.mesa-nhlbi.org.

Metabolic Syndrome in Men (METSIM) was supported by the Academy of Finland (contract 124243), the Finnish Heart Foundation, the Finnish Diabetes Foundation, Tekes (contract 1510/31/06), and the Commission of the European Community (HEALTH-F2-2007 201681), and the US National Institutes of Health grants DK093757, DK072193, DK062370, and ZIA-HG000024.

Mass General Brigham Biobank (MGB) acknowledges the Partners HealthCare System for support of the MGB biobank and MGB patients for providing samples, genomic data, and health information data, as well as research support by NIDDK K24 DK110550 (to J.C.F.), K24 DK080140 (to J.B.M.) and NIDDK K23DK114551 (to M.S.U).

Michigan Genomics Initiative (MGI) was supported by NIH research grants HL117626 and HG007022. MGI was supported by internal research funds from the University of Michigan School of Public Health, the University of Michigan Medical School, and the University of Michigan President's Office. MGI are especially grateful to the generosity of all research participants.

VA Million Veteran Program (MVP). This research is based on data from the MVP, Office of Research and Development, Veterans Health Administration and was supported by award MVP000. This publication does not represent the views of the Department of Veterans Affairs, the US Food and Drug Administration, or the US Government. This research was also supported by funding from the Department of Veterans Affairs awards I01- BX003362 (P.S.T. and K.-M.C.). K.-M.C. and P.S.T. are supported by the VA Cooperative Studies Program. Research support for this study was generously provided by the Department of Veterans Affairs (VA) Informatics and Computing Infrastructure (VINCI) (VA HSR RES 13-457).

Nagahama Study (NAGAHAMA) was supported by a university grant, The Center of Innovation Program, The Global University Project, and a Grant-in-Aid for Scientific Research (25293141, 26670313, 26293198, 17H04182, 17H04126, 17H04123, 18K18450) from the Ministry of Education, Culture, Sports, Science and Technology of Japan, the Practical Research Project for Rare/Intractable Diseases (ek0109070, ek0109070, ek0109196, ek0109348), the Comprehensive Research on Aging and Health Science Research Grants for Dementia R&D (dk0207006, dk0207027), the Program for an Integrated Database of Clinical and Genomic Information (kk0205008), the Practical Research Project for Life-style-related Diseases including Cardiovascular Diseases and Diabetes Mellitus (ek0210066, ek0210096,

ek0210116), and the Research Program for Health Behavior Modification by Utilizing IoT (le0110005) from Japan Agency for Medical Research and Development (AMED); Takeda Medical Research Foundation, and Mitsubishi Foundation, Daiwa Securities Health Foundation, and Sumitomo Foundation.

Netherlands Epidemiology of Obesity (NEO) thanks all individuals who participated in the study, all participating general practitioners for inviting eligible participants and all research nurses for collection of the data. NEO thank the study group, Pat van Beelen, Petra Noordijk and Ingeborg de Jonge for the coordination, lab and data management of the study. Genotyping was supported by the Centre National de Génotypage (Paris, France), headed by Jean-Francois Deleuze. NEO is supported by the participating Departments, the Division and the Board of Directors of the Leiden University Medical Center, and by the Leiden University, Research Profile Area Vascular and Regenerative Medicine.

NIDDM-Atherosclerosis Study Hispanic Cohorts (NIDDM) was supported by grant HL055798.

Northewestern University Genetics (NUGENE) was funded by the Northwestern University's Center for Genetic Medicine, Northwestern University, and Northwestern Memorial Hospital. Samples and data used in this study were provided by the NUgene Project (www.nugene.org). Assistance with phenotype harmonization was provided by the eMERGE Coordinating Center (Grant number U01HG04603). This study was funded through the NIH, NHGRI eMERGE Network (U01HG004609). Funding support for genotyping, which was performed at The Broad Institute, was provided by the NIH (U01HG004424). Assistance with phenotype harmonization and genotype data cleaning was provided by the eMERGE Administrative Coordinating Center (U01HG004603) and the National Center for Biotechnology Information (NCBI). The datasets used for the analyses described in this manuscript were obtained from dbGaP at http://www.ncbi.nlm.nih.gov/gap through dbGaP accession number phs000237.v1.p1.

Prospective Investigation of the Vasculature in Uppsala Seniors (PIVUS) was supported by Wellcome Trust Grants (WT098017, WT064890, WT090532), Uppsala University, Uppsala University Hospital, the Swedish Research Council, and the Swedish Heart-Lung Foundation.

Penn Medicine BioBank (PMBB). We acknowledge the PMBB for providing data and thank the patient-participants of Penn Medicine who consented to participate in this research program. We would also like to thank the Penn Medicine BioBank team and Regeneron Genetics Center for providing genetic variant data for analysis. The PMBB is approved under IRB protocol# 813913 and supported by Perelman School of Medicine at University of Pennsylvania, a gift from the Smilow family, and the National Center for Advancing Translational Sciences of the National Institutes of Health under CTSA award number UL1TR001878.

Pakistan Risk of Myocardial Infarction Study (PROMIS) was funded by the Wellcome Trust, UK, and Pfizer (genotyping) and was supported through funds available to investigators at the Center for Non-Communicable Diseases, Pakistan, and the University of Cambridge, UK (fieldwork). Biomarker assays in PROMIS have been funded through grants awarded by the

National Institutes of Health (RC2HL101834 and RC1TW008485) and the Fogarty International (RC1TW008485).

Prospective Study of Pravastatin in the Elderly at Risk (PROSPER) was supported by an investigator-initiated grant obtained from Bristol-Myers Squibb. Prof. J.W.J. is an Established Clinical Investigator of the Netherlands Heart Foundation (grant 2001 D 032). Support for genotyping was provided by the seventh framework program of the European commission (grant 223004) and by the Netherlands Genomics Initiative (Netherlands Consortium for Healthy Aging grant 050-060-810).

Sea Islands Genetic Network Reasons for Geographic and Racial Differences in Stroke (REGARDS) is supported by cooperative agreement U01 NS041588 co-funded by the National Institute of Neurological Disorders and Stroke (NINDS) and the National Institute on Aging (NIA), National Institutes of Health, Department of Health and Human Service. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NINDS or the NIA. Additional funding was from R01 DK084350 from the National Institutes of Health.

Ragama Health Study (RHS) was supported by a grant from the National Center for Global Health and Medicine (NCGM).

Rotterdam Study (RS) are grateful to the participants and staff involved in the study, and the participating general practitioners and pharmacists. RS is funded by Erasmus Medical Center and Erasmus University, Rotterdam, Netherlands Organization for the Health Research and Development (ZonMw), the Research Institute for Diseases in the Elderly (RIDE), the Ministry of Education, Culture and Science, the Ministry for Health, Welfare and Sports, the European Commission (DG XII), and the Municipality of Rotterdam.

Shanghai Breast Cancer Study and Shanghai Women's Health Study (SBCS/SWHS) was supported in part by US National Institutes of Health grants R01CA64277 and R01CA124558, as well as Ingram Professorship and Research Reward funds from the Vanderbilt University School of Medicine. We want to thank participants and research staff of the study, Regina Courtney for plasma and DNA sample preparation, and Hui Cai, Ben Zhang and Jing He for data processing and analyses.

Singapore Chinese Eye Study (SCES) is supported by the National Medical Research Council (NMRC), Singapore (grants 0796/2003, 1176/2008, 1149/2008, STaR/0003/2008, 1249/2010, CG/SERI/2010, CIRG/1371/2013, and CIRG/1417/2015), and Biomedical Research Council (BMRC), Singapore (08/1/35/19/550 and 09/1/35/19/616).

Starr County Health (SCH) was supported by grants from the National Institutes of Health (DK073541, DK085501, HL102830 and DK116378) and funds from the State of Texas. SCH thank the field staff in Starr County for their careful collection of these data and are especially grateful to the participants who so graciously cooperated and gave of their time. Starr County Health

Singapore Chinese Health Study (SCHS) was supported by the US National Institutes of Health grants R01DK08072, R01CA144034 and UM1CA182876.

Slim Initiative for Genomic Medicine in the Americas (SIGMA). This work was conducted as part of the Slim Initiative for Genomic Medicine, a joint U.S.-Mexico project funded by the Carlos Slim Health Institute. The UNAM/INCMNSZ diabetes study was supported by Consejo Nacional de Ciencia y Tecnología grants 138826, 128877, CONACyT- SALUD 2009-01-115250, and a grant from Dirección General de Asuntos del Personal Académico, UNAM, IT 214711. The Diabetes in Mexico Study was supported by Consejo Nacional de Ciencia y Tecnología grant 86867 and by Instituto Carlos Slim de la Salud, A.C. The Mexico City Diabetes Study was supported by National Institutes of Health (NIH) grant R01HL24799 and by the Consejo Nacional de Ciencia y Tenologia grants: 2092, M9303, F677-M9407, 251M, and 2005-C01-14502, SALUD 2010-2-151165. The Multiethnic Cohort was supported by NIH grants CA164973, CA054281, and CA063464.

Singapore Malay Eye Study (SIMES) is supported by the National Medical Research Council (NMRC), Singapore (grants 0796/2003, 1176/2008, 1149/2008, STaR/0003/2008, 1249/2010, CG/SERI/2010, CIRG/1371/2013, and CIRG/1417/2015), and Biomedical Research Council (BMRC), Singapore (08/1/35/19/550 and 09/1/35/19/616).

Singapore Indian Eye Study (SINDI) is supported by the National Medical Research Council (NMRC), Singapore (grants 0796/2003, 1176/2008, 1149/2008, STaR/0003/2008, 1249/2010, CG/SERI/2010, CIRG/1371/2013, and CIRG/1417/2015), and Biomedical Research Council (BMRC), Singapore (08/1/35/19/550 and 09/1/35/19/616).

Samsung Medical Center (SMC) was supported by a grant from Samsung Biomedical Research Institute. Genotyping of the patients and control subjects from SMC was conducted by Duk-Hwan Kim in the Dept. of Molecular Cell Biology, Sungkyunkwan University School of Medicine, and was supported by a grant from Samsung Biomedical Research Institute.

Seoul National University Hospital (SNUH) was supported by a grant from the Korea Health Technology R&D Project through the Korea Health Industry Development Institute, funded by the Ministry of Health & Welfare (grant numbers HI15C1595, HI14C0060, HI15C3131).

Taiwan Metabochip Consortium Zhonghua (TAICHI-G) was supported by grants from: the National Health Research Institutes, Taiwan (PH-099-PP-03, PH-100-PP-03, and PH-101-PP-03); the National Science Council, Taiwan (NSC 101-2314-B-075A-006-MY3, MOST 104-2314-B-075A-006-MY3, MOST 104-2314-B-075A-007, and MOST 105-2314-B-075A-003); and the Taichung Veterans General Hospital, Taiwan (TCVGH-1020101C, TCVGH-1020102D, TCVGH-1023102B, TCVGH-1023107D, TCVGH-1030101C, TCVGH- 1030105D, TCVGH-1033503C, TCVGH-1033102B, TCVGH-1033108D, TCVGH-1040101C, TCVGH-1040102D, TCVGH-1043504C, and TCVGH-1043104B). TAICHI-G was also supported in part by the National Center for Advancing Translational Sciences (CTSI grant UL1TR001881).

Thrombolysis in Myocardial Infarction Study Group (TIMI) acknowledge Marc S. Sabatine, Robert P. Giugliano, Steve D. Wiviott, Ben M. Scirica, Michelle L. O'Donoghue, and Elliott Antman.

Taiwan Type 2 Diabetes (TWT2D) was supported by the GMM Study, Academia Sinica, Taiwan.

Danish T2D Case-Control Study (UCPH) was undertaken by the Novo Nordisk Foundation Center for Basic Metabolic Research, which is an independent Research Center, based at the University of Copenhagen, Denmark and partially funded by an unconditional donation from the Novo Nordisk Foundation (www.cbmr.ku.dk, Grant number NNF18CC0034900). Included study samples were supported by the Danish Research Fund and the National Danish Research Fund (The Vejle Diabetes Biobank), the Velux Foundation, The Danish Medical Research Council and Danish Agency for Science, Technology and Innovation (Health 2006); the Danish Research Council, the Danish Centre for Health Technology Assessment and Novo Nordisk Inc. (Inter99), the Timber Merchant Vilhelm Bang's Foundation and the Danish Heart Foundation (Health 2008), TrygFonden, the Lundbeck Foundation and the Novo Nordisk Foundation (NNF15OC0015896, DanFunD).

UK Biobank (UKBB) analyses were conducted using the UK Biobank resource under applications 236, 9161, and 10035. This research was supported by the British Heart Foundation (grant SP/13/2/30111). Large-scale comprehensive genotyping of UK Biobank for cardiometabolic traits and diseases: UK CardioMetabolic Consortium (UKCMC).

Uppsala Longitudinal Study of Adult Men (ULSAM) was supported by Wellcome Trust Grants (WT098017, WT064890, WT090532), Uppsala University, Uppsala University Hospital, the Swedish Research Council, and the Swedish Heart-Lung Foundation.

Wake Forest School of Medicine (WFSM) was supported by NIH grants K99 DK081350, R01 DK066358, R01 DK053591, R01 DK087914, U01 DK105556, R01 HL56266, R01 DK070941 and in part by the General Clinical Research Center of the Wake Forest School of Medicine grant M01 RR07122. Genotyping services were provided by the Center for Inherited Disease Research (CIDR), which is fully funded through a federal contract from the National Institutes of Health to The Johns Hopkins University, contract number HHSC268200782096C.

Women's Health Initiative (WHI). The WHI program is funded by the National Heart, Lung, and Blood Institute, National Institutes of Health, U.S. Department of Health and Human Services through 75N92021D00001, 75N92021D00002, 75N92021D00003, 75N92021D00004, 75N92021D00005. Funding for WHI SHARe genotyping was provided by NHLBI Contract N02-HL-64278. The Molecular Epidemiology of Diabetes in the WHI is supported by R01DK125403 (to S.Liu). The contents of this publication are solely the responsibility of the authors and do not necessarily represent the official view of the National Institutes of Health. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. A list of WHI investigators is available at: https://www-whi-org.s3.us-west-2.amazonaws.com/wp-content/uploads/WHI-Investigator-Short-List.pdf.

Wellcome Trust Case Control Consortium (WTCCC) analysis and genotyping was supported by: Wellcome Trust funding 090367, 098381, 090532, 083948, 085475, 101630 and 203141; MRC (G0601261); EU (Framework 7) HEALTH-F4-2007-201413; and NIDDK DK098032 and U01-DK105535.

GWAS analyses of GLP-1 concentrations. The PPP-Botnia Study has been financially supported by grants from Folkhälsan Research Foundation, the Sigrid Juselius Foundation, The Academy of Finland (grants no. 263401, 267882, 312063, 336822, 312072 and 336826), University of Helsinki, Nordic Center of Excellence in Disease Genetics, EU (EXGENESIS, MOSAIC FP7-600914), Ollqvist Foundation, Swedish Cultural Foundation in Finland, Finnish Diabetes Research Foundation, Foundation for Life and Health in Finland, Finnish Medical Society, State Research Funding via the Helsinki University Hospital, Perklén Foundation, Närpes Health Care Foundation and Ahokas Foundation. The study has also been supported by the Ministry of Education in Finland, the Municipal Heath Care Center and Hospital in Jakobstad and Health Care Centers in Vasa, Närpes and Korsholm. The research leading to these results has received funding from the European Research Council under the European Union's Seventh Framework Programme (FP7/2007-2013) / ERC grant agreement no. 269045. The role of the founding PI, Professor Leif Groop, and the skilful assistance of the Botnia Study Group is gratefully acknowledged.

Personal acknowledgements. K.Suzuki was supported by Japan Agency for Medical Research and Development (JP21km0405213, JP20km0405202, JP21tm0424218). R.Mandla was supported by NHGRI U01HG011723, 1-19-ICTS-068. A.H.-C. was supported by NHGRI U01HG011723, 1-19-ICTS-068. O.B. was supported by the European Union's Horizon 2020 research and innovation programme under Grant Agreement No 101017802 (OPTOMICS). L.E.P. was supported by R01GM133169, R01HL142302, R01DK127084. P.S. was supported by NHGRI U01HG011723, 1-19-ICTS-068. F.B. was supported by BHF Centre of Research Excellence, Oxford (RE/13/1/30181). W.Zhang acknowledges support from iHealth-T2D, 643774 and the National Institute for Health Research/Wellcome Trust Imperial Clinical Research Facility. R.A.S. acknowledges support from the Medical Research Council Epidemiology Unit (MC_UU_12015/1). D.T. acknowledges funding from US National Institutes of Health grant DK062370. E.J.P. was supported by the Canadian Institutes of Health Research (CIHR) and the Banting and Best Diabetes Center, University of Toronto. M.W. was supported by the Deutsche Forschungsgemeinschaft (DFG, German Research Foundation) – CRC 1453 Project-ID 431984000. C.Sarnowski acknowledges support from NIA R00 AG066849. D.N. acknowledges support from NIEHS grant T32ES013678. S.-H.K. acknowledges funding from Korea Health Technology R&D Project through the Korea Health Industry Development Institute (grant number HI15C3131). A.W. is supported by a PhD studentship funded by the Wellcome Trust. L.S.A. acknowledges support from the National Institute for Health (NIH), the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) for R01 HD30880, National Institute on Aging (NIA) for R01 AG065357, National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) for R01 DK104371 and R01 HL108427. C.F.B. acknowledges funding from the Dr. Robert C. and Veronica Atkins Foundation. J.Chen acknowledges Inês Barroso for supervision and support (Wellcome WT098051 and WT206194). J.Danesh holds a British Heart Foundation Professorship and a NIHR Senior Investigator Award, and this work was supported by core funding from the: British Heart Foundation (RG/13/13/30194; RG/18/13/33946) and NIHR

Cambridge Biomedical Research Centre (BRC-1215-20014).S.K.D. acknowledges support from the NIH/NIDDK grant R01 DK090111. S.D. acknowledges funding from the US National Institutes of Health Fogarty grant D43 TW009077, the National Institute for Health (NIH), the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) for R01 HD30880, National Institute on Aging (NIA) for R01 AG065357, National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) for R01DK104371 and R01HL108427. D.S.E. acknowledges support from the US National Institutes of Health U24AG051129. P.G.-L. acknowledges support from the National Institute for Health (NIH), the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) for R01 HD30880, National Institute on Aging (NIA) for R01 AG065357, National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) for R01DK104371 and R01HL108427. A.T.H. acknowledges support from a Wellcome Trust Senior Investigator award (grant number 098395/Z/12/Z). K.Läll acknowledges funding from the Estonian Research Council grant 1911. N.R.L. acknowledges funding from the US National Institutes of Health TW008288. C.M.L. is supported by the Li Ka Shing Foundation, WT-SSI/John Fell funds Oxford, NIHR Oxford Biomedical Research Centre, Widenlife, and NIH (5P50HD028138-27). A.E.L. acknowledges funding from US National Institutes of Health grant DK062370. J.Luan acknowledges support from the Medical Research Council Epidemiology Unit (MC_UU_12015/1). S.Maeda is supported by the grant for Okinawa innovation/eco-system promotion project from the Okinawa prefecture. M.A.N. was supported in part by the Intramural Research Program of the NIH, National Institute on Aging (NIA), National Institutes of Health, Department of Health and Human Services (project number ZO1 AG000535), as well as the National Institute of Neurological Disorders and Stroke (NINDS); participation in this project was part of a competitive contract awarded to Data Tecnica International LLC by the National Institutes of Health to support open science research. Y.O. was supported by JSPS KAKENHI (22H00476), and AMED (JP21gm4010006, JP22km0405211, JP22ek0410075, JP22km0405217, JP22ek0109594, JP223fa627002, JP223fa627010, JP233fa627011), JST Moonshot R&D (JPMJMS2021, JPMJMS2024), Takeda Science Foundation, Bioinformatics Initiative of Osaka University Graduate School of Medicine, Institute for Open and Transdisciplinary Research Initiatives and Center for Infectious Disease Education and Research (CiDER), Osaka University. H.G.P. was supported by R01GM133169, R01HL142302, R01DK127084. N.Sattar is supported by British Heart Foundation Centre of Excellence Grant RE/18/6/34217. N.Shojima was supported by Japan Agency for Medical Research and Development (JP20km0405202, JP21tm0424218). E.W. acknowledges Inês Barroso for supervision and support (Wellcome WT098051 and WT206194), and acknowledges support from the Medical Research Council Epidemiology Unit (MC_UU_12015/1). Y.S.C. acknowledges support from the National Research Foundation of Korea (NRF) Grant funded by the Ministry of Education (NRF-2020R1I1A2075302). E.Ingelsson was supported by NIH/NIDDK 1R01DK106236-01A1. J.-Y.W. was supported by Academia Sinica GMM Study. R.C.W.M. acknowledges funding from the Research Grants Council Theme-based Research Scheme (T12-402/13-N), the RGC Research Impact Fund (R4012-18), and a Croucher Foundation Senior Medical Research Fellowship. F.S.C. acknowledges support from United States' National Institutes of Health (NIH) grant ZIA-HG000024. K.-S.P. acknowledges funding from Korea Health Technology R&D Project through the Korea Health Industry Development Institute (grant numbers HI15C1595, HI14C0060). R.M.-C. acknowledges support from grants NIH U10 EY 11753 and NIH U10 EY 11753. C.-Y.C. acknowledges funding from the National Medical Research Council (NMRC), Singapore (CSA-

SI/0012/2017). J.Dupuis is supported by R01 DK078616 and U01 DK078616. A.Köttgen was supported by the by the Deutsche Forschungsgemeinschaft (DFG, German Research Foundation) KO 3598/5-1 and CRC 1453 Project-ID 431984000. D.W.B. acknowledges support from the US National Institutes of Health U01DK105556 and R01DK66358. K.E.N. acknowledges support by R01HD057194, R01DK122503, R01HG010297, R01HL142302, R01HL143885, R01HG009974, and R01DK101855. D.S. has received funding from NHLBI, NINDS, the British Heart Foundation, Pfizer, Regeneron, Genentech, and Eli Lilly pharmaceuticals. N.J.W. acknowledges support from the Medical Research Council Epidemiology Unit (MC_UU_12015/1). E.A. was funded by grants from the Swedish Research Council (2020-02191) and the Novo Nordisk Foundation (NNF21OC0070457). M.O.G. acknowledges support from the US National Institutes of Health grants P30DK063491 and UL1TR001881, as well as the Eris M. Field Chair in Diabetes Research. K.L.M. acknowledges funding from the US National Institutes of Health R01DK072193, R01DK093757, U01DK105561. C.L. acknowledges support from the Medical Research Council Epidemiology Unit (MC_UU_12015/1). R.J.F.L. acknowledges support from R01DK110113, R01DK107786, R01HL142302, and R56HG010297. J.C.F. is a Massachusetts General Hospital Research Scholar and was supported by NIDDK U01 DK105554 and NIDDK K24 DK110550. J.C.D. acknowledges support from United States' National Institutes of Health (NIH) grant ZIA-HG200417. T.Y. was supported by Japan Agency for Medical Research and Development (JP20km0405202, JP21tm0424218). T.Kadowaki was supported by Japan Agency for Medical Research and Development (JP20km0405202, JP21tm0424218). J.C.C. acknowledges support from the Singapore Ministry of Health's National Medical Research Council under its Singapore Translational Research Investigator (STaR) Award (NMRC/STaR/0028/2017), iHealth-T2D 643774, and the National Institute for Health Research/Wellcome Trust Imperial Clinical Research Facility. M.C.Y.N. acknowledges support from the US National Institutes of Health U01DK105556, R01DK66358, and a supplement to R01DK78616-06S1. J.E.B. acknowledges support from R01GM133169, R01HL142302, and R01DK127084. M.I.M. acknowledges funding from: The European Commission (ENGAGE: HEALTH-F4-2007-201413); MRC (G0601261, L020149); National Institutes of Health (RC2-DK088389, DK085545, R01-DK098032, U01-DK105535); Wellcome (083948, 085475, 090367, 090532, 098381, 101630, 203141, 212259). J.B.M. acknowledges funding through NIH grants R01DK078616, U01DK078616 and K24DK080140. C.N.S. was supported by American Heart Association Postdoctoral Fellowship 15POST24470131 and 17POST33650016, and American Diabetes Association 11-22-JDFPM-06. J.M.M. is funded by American Diabetes Association Innovative and Clinical Translational Award 1-19-ICTS-068, and NHGRI U01HG011723. M.B. acknowledges funding from US National Institutes of Health grant DK062370. M.V. acknowledges support from the Corporal Michael J. Crescenz VA Medical Center Research Department. B.F.V. acknowledges support from the NIH/NIDDK (DK126194). A.P.M. acknowledges support from US National Institutes of Health U01DK105535, Versus Arthritis (grant reference 21754), NIHR Manchester Biomedical Research Centre (NIHR203308), and MRC (MR/W029626/1).

The views expressed in this article are those of the authors and do not necessarily represent those of: the UK National Health Service, the UK National Institute for Health Research, or the UK Department of Health and Social Care; the US National Heart, Lung, and Blood Institute, the US National Institute of Neurological Disorders and Stroke, the US National Institute on Aging, the US National Institutes of Health, the US Department of

Health and Human Services, the US Department of Veterans Affairs, the US Food and Drug Administration, or the US Government.

Ethics statements

Anti-aging study cohort (AASC). The ethics committees of Ehime University Graduate School of Medicine approved all study procedures. Written informed consent was obtained from all participants.

All Of Us Research Program (AOURP). All research was conducted under the guidelines defined by the All of Us Ethical Conduct of Research Policy.

Atherosclerosis Risk in Communities (ARIC). Institutional Review Board approvals were obtained at all study sites: National Heart, Lung, and Blood Institute, University of North Carolina at Chapel Hill, Wake Forest Baptist Medical Center, University of Mississippi Medical Center, University of Minnesota, and Johns Hopkins University. All participants provided written informed consent.

Biobank Japan (BBJ). All participants provided written informed consent as approved by the ethical committees of the RIKEN Yokohama Institute and the Institute of Medical Science, the University of Tokyo. Ethical approvals of AMED GRIFIN Diabetes Initiative Japan were gained from the Ethics Committees of Osaka University and the University of Tokyo.

Beijing Eye Study (BES). Approval was obtained from the Medical Ethics Committee of the Beijing Tongren Hospital. All participants gave written informed consent.

BioMe Biobank (BIOME). Approval was obtained from the Institutional Review Board at the Icahn School of Medicine at Mount Sinai. All participants provided written informed consent for genomic data sharing.

Vanderbilt University Medical Center's BioVU (BIOVU). Analyses of DIAMANTE data at Vanderbilt University Medical Center are approved under IRB #190891 and analysis of BioVU data are approved under IRBs #210163 and #171279. In all three cases, the data analyzed received non-human subject determinations.

Bangladesh Population Cohort (BPC). The conduct of the BPC was reviewed and approved by Ethical Committees of the Bangladesh Medical Research Council and Institutional Review Boards of the University of Chicago.

Cardiometabolic Genome Epidemiology (CAGE-AMAGASKI and CAKE-GWAS). Approval was obtained from the Institutional Review Boards at the National Center for Global Health and Medicine. All participants provided written informed consent.

Cardiometabolic Genome Epidemiology (CAGE-KING). Approval was obtained from the ethics committees of Aichi Gakuin University, Jichi Medical University, Nagoya University and Kyushu University. All participants provided written informed consent.

Coronary Artery Risk Development in Young Adults (CARDIA). Participating centers (Northwestern University, University of Alabama Birmingham, University of Minnesota, and

Kaiser Foundation Research Institute) provided ethics approval for the CARDIA study, and all participants provided written informed consent to participate.

Cleveland Family Study (CFS). Approval was obtained from the Institutional Review Board of Mass General Brigham (formerly Partners HealthCare). Written informed consent was obtained from all participants.

China Health and Nutrition Survey (CHNS). Approval was obtained from the Institutional review Boards at the University of North Carolina at Chapel Hill, the Chinese National Human Genome Center at Shanghai, and the Institute of Nutrition and Food Safety at the China Centers for Disease Control. All participants provided written informed consent.

Cardiovascular Health Study (CHS). Approval was obtained from the Institutional Review Boards at Wake Forest University, University of California, Davis, Johns Hopkins, University of Pittsburgh, and the University of Washington, Seattle. All participants provided written informed consent.

China Kadoorie Biobank (CKB). All participants provided written informed consent. Ethical approval was obtained from Oxford Tropical Research Ethics Committee (OxTREC) and from the Ethical Review Committees of the Chinese Centre for Disease Control and Prevention and the Chinese Academy of Medical Sciences/Peking Union Medical College.

Cebu Longitudinal Health and Nutrition Survey (CLHNS). Written informed consent was obtained from all participants. Study protocols were approved by the University of North Carolina Institutional review Board for the Protection of Human Subjects.

Diabetic Cohort and Singapore Prospective Study Program (DC/SP2). Study protocols were approved by the Singapore General Hospital Ethics Committee, and National University of Singapore Institutional Review Board. All participants provided written informed consent.

Durban Diabetes Study and Durban Diabetes Case Control (DDS/DCC). Approvals were granted by the Biomedical Research Ethics Committee at the University of KwaZulu-Natal and the UK National Research Ethics Service. All participants provided written informed consent.

deCODE genetics (DECODE). The study was approved by the Icelandic National Bioethics Committee (approval no. VSN-16-112) after evaluation by the Icelandic Data Protection Authority. We obtained written informed consent for all participants in this study who donated samples. All data processing complies with the Icelandic Data Protection Authority (no. PV_2017060950ÞS).

Diabetes Gene Discovery Group (DGDG). All participants signed informed consent, and the protocol was approved by the French ethics committee.

Diabetes Genetics Initiative (DGI). The study was approved by the Ethics Committees of the Helsinki University Hospital, Helsinki, Finland, and Lund University, Sweden.

Estonian Genome Center of the University of Tartu (EGCUT). All analyses were approved by the Ethics Review Committee of the University of Tartu. All participants provided written informed consent.

Electronic Medical Records and Genomics Network (EMERGE). Approval was obtained from the Institutional Review Boards at Boston Children's Hospital, Children's Hospital of Philadelphia, Cincinnati Children's Hospital Medical Center, Essentia Institute of Rural Health, Geisinger Clinic, Group Health Cooperative, Marshfield Clinic Research Foundation, Mayo Clinic, Icahn School of Medicine at Mount Sinai, Northwestern University, Pennsylvania State University, Vanderbilt University Medical Center, and University of Washington. All participants provided written informed consent.

European Prospective Investigation into Cancer and Nutrition (EPIC-INTERACT). The EPIC-InterAct study was approved by the local ethics committee in the participating countries and the Internal Review Board of the International Agency for Research on Cancer. All participants gave written informed consent. The study was coordinated by the Medical Research Council Epidemiology Unit at the University of Cambridge.

Epidemiologic Study of the Screenees for Diabetes Reduction Assessment with Ramipril and Rosiglitazone Medication (EPIDREAM). All study participants consented to analysis of blood samples. Approval was granted by the Hamilton Integrated Research Ethics Board, at McMaster University, Hamilton, Canada.

Family Heart Study (FAMHS). Approval was obtained from the Institutional Review Board at Washington University, St. Louis. Written informed consent, including consent to participate in genetic studies, was obtained from all participants.

Framingham Heart Study (FHS). Approval was obtained from the Institutional review Board of Boston University Medical Campus. All study participants provided written informed consent.

Finland-United States Investigation of NIDDM Genetics (FUSION). Approval was obtained from the coordinating Ethics Committee of the Hospital District of Helsinki and Uusimaa. All participants provided written informed consent.

Genes & Health (G&H). Genes & Health has NHS Health Research Authority favourable ethical opinion from NRES Committee London – South East 14/LO/1240.

German Chronic Kidney Disease (GCKD). All participants provided written informed consent. The study was registered in the national registry for clinical studies (DRKS 00003971) and was approved by local ethics committees.

Genetic Study of Atherosclerosis Risk (GENESTAR). Approval was obtained from the Johns Hopkins Medicine Institutional Review Board. All participants gave written informed consent.

Genetic Epidemiology Network of Arteriosclerosis (GENOA). Approval was granted by Institutional Review Boards of the University of Michigan, University of Mississippi Medical Center and Mayo Clinic. Written informed consent was obtained from all participants.

Resource for Genetic Epidemiology on Adult Heath and Aging (GERA). The Institutional Review Boards for Human Subjects Research of both Kaiser Permanente Medical Care Plan (Northern California Region) and the University of California at San Francisco approved the project.

Genetics of Diabetes and Audit Research in Tayside Scotland (GODARTS). Approval was obtained from the Tayside Medical Ethics Committee. Informed consent was obtained for all participants.

Genetics of Latinos Diabetic Retinopathy (GOLDR). Approval was granted by the Institutional Review Board of the Lundquist Institute for Biomedical Innovation at Harbor-UCLA Medical Center.

Genetic Overlap Between Metabolic and Psychiatric Traits and Teens of Attica: Genes and Environment (GOMAP-TEENAGE). Ethical permission for TEENAGE was obtained from the Bioethics Committee of Harokopio University, Athens. Ethical permission for GOMAP was obtained from the Dromokaiteio Scientific Committee, Dromokaiteio Management Committee, Dafni Scientific Committee, Eginitio Scientific Committee and Harokopio Ethics Committee. All participants of GOMAP-TEENAGE gave written informed consent.

Genomic Research Cohort for CCMB Diabetes Study (GRCCDS). Ethics committees of CSIR-Centre for Cellular and Molecular Biology and KEM Hospital and Research Centre approved the project.

Health, Aging and Body Composition Study (HABC). The Institutional Review Boards at the University of Memphis and the University of Pittsburgh granted approval to conduct the Health ABC Study, and all participants provided written informed consent.

Healthy Aging in Neighborhoods of Diversity Across the Life Span Study (HANDLS). Approval was granted by the National Institutes of Health Institutional Review Board (study number 09AGN248). All participants provided written informed consent.

Hispanic Community Health Study/Study of Latinos (HCHS/SOL). Approval was obtained from Institutional Review Boards at the University of North Carolina at Chapel Hill, Albert Einstein College of Medicine, University of Illinois at Chicago, University of Miami, and San Diego State University. All participants provided written informed consent.

Hong Kong Diabetes Registry (HKDR). Approval was obtained from the Chinese University of Hong Kong Clinical Research Ethics Committee.

Health Professionals' Follow-Up Study (HPFS). Approval was obtained from the Human Research Committee at the Brigham and Women's Hospital. All participants provided written informed consent**.**

Mexican American Hypertension and Insulin Resistance (HTNIR). Approval was granted by Human Subjects Protection Institutional Review Boards at the University of California at Los Angeles, University of Southern California, Lundquist/LABioMed/Harbor-UCLA and Cedars-Sinai Medical Center.

Howard University Family Study (HUFS). All human participants from the HUFS included in the analyses of this manuscript provided written informed consent prior to enrollment. The HUFS study was approved by the Institutional Review Board at Howard University.

Indian Diabetes Consortium (INDICO). Approval was obtained by the Human Ethics Committees of All India Institute of Medical Sciences, New Delhi and CSIR-Institute of Genomics and Integrative Biology, New Delhi, India, and was conducted in accordance with the principles of Helsinki Declarations. Informed written consent was obtained from all of participants.

INTERHEART (INTERHEART). All study participants consented to analysis of blood samples. Approval was granted by the Hamilton Integrated Research Ethics Board, at McMaster University, Hamilton, Canada.

Jackson Heart Study (JHS). Approval was obtained from Institutional Review Boards at Jackson State University, Tougaloo College and the University of Mississippi Medical Center. All participants provided written informed consent.

Korean Association Resource (KARE). Approval was granted by the Institutional review Board at the Korean National Institute of Health. All participants provided written informed consent.

Korean Biobank Array from the Korean Genome and Epidemiology (KoGES) Consortium (KBA). Approval was granted by the Institutional Review Board of the Korean National Institute of Health. All participants provided written informed consent.

Collaborative Health Research in the Region of Augsburg (KORA). Approval was granted by the Ethics Committee of the Medical Association of Bavaria (number 06068). All participants provided informed consent.

Los Angeles Latino Eye Study (LALES). Approval was obtained from the Los Angeles County/University of Southern California Institutional Review Board, and Western Institutional Review Board at Southern California Eye Institute. All participants provided written informed consent.

London Life Sciences Prospective Population (LOLIPOP). Approval was obtained from the London-Fulham Research Ethics Committee (ref 07/H0712/150). All participants gave an written informed consent.

Mexican American Study of Coronary Artery Disease (MACAD). Approval was granted by Human Subjects Protection Institutional Review Boards at the University of California at Los

Angeles, University of Southern California, Lundquist/LABioMed/Harbor-UCLA and Cedars-Sinai Medical Center.

Mexico City (MC). Approval was obtained from Institutional Review Boards at the Ethics and Scientific Commission members and the AUTHORIZATION is issued with registration number R-2011-785-018 and the Conacyt SALUD-2010-02-150352. In Canada, approval was obtained from the Research Ethics Board from the University of Toronto (Protocol 15770).

Malmo Diet and Cancer Study (MDCS). The study protocol for MDC was sanctioned by the Ethics Review Committee of Lund University (approval numbers 532/2006, 51-90). All participants provided their written consent.

Multi-Ethnic Study of Atherosclerosis (MESA). Approval was obtained from Institutional Review Boards at the University of Washington, Wake Forest School of Medicine, Northwestern University, University of Minnesota, Columbia University, Johns Hopkins University, Cedars-Sinai Medical Center, and the University of California at Los Angeles.

Metabolic Syndrome in Men (METSIM). Approval was granted by the Ethics Committee of the University of Kuopio and the Kuopio University Hospital. All participants gave written informed consent.

Mass General Brigham Biobank (MGB). The MGB Biobank protocol and informed consent documents are reviewed annually by the Partners-MGB Institutional Review Board (#2009P002312). All patients who participate in the MGB Biobank are consented for their samples to be linked to their identified clinical information. They have also consented for their information to be used for a broad range of research and for their deidentified information to be shared outside of MGB.

Michigan Genomics Initiative (MGI). Approval was granted by the IRBMED Institutional Review Board of the University of Michigan. All participants gave written informed consent.

VA Million Veteran Program (MVP). All participating studies were conducted in compliance with the Declaration of Helsinki and comply with all relevant ethical and local regulatory requirements. Specifically, the contributing genetic association studies were approved by the Department of Veteran's Affairs central IRB.

Nagahama Study (NAGAHAMA). Approval was granted by the ethics committees of Kyoto University Graduate School of Medicine. Written informed consent was obtained from all participants.

Netherlands Epidemiology of Obesity (NEO). Approval was obtained from the Medical Ethics Committee of Leiden University Medical Center. All participants gave written informed consent.

Nurses Health Study (NHS). Approval was obtained from the Human Research Committee at the Brigham and Women's Hospital. All participants provided written informed consent**.**

NIDDM-Atherosclerosis Study Hispanic Cohorts (NIDDM). Approval was granted by Human Subjects Protection Institutional Review Boards at the University of California at Los Angeles, University of Southern California, City of Hope, Lundquist/LABioMed/Harbor-UCLA and Cedars-Sinai Medical Center.

Northewestern University Genetics (NUGENE). Approval was obtained from Institutional Review Boards at Northwestern University and Vanderbilt University.

Prospective Investigation of the Vasculature in Uppsala Seniors (PIVUS). Approval was granted by the Ethics Committee of Uppsala University. All participants provided written informed consent.

Penn Medicine BioBank (PMBB). All participating studies were conducted in compliance with the Declaration of Helsinki and comply with all relevant ethical and local regulatory requirements. Specifically, the contributing genetic association studies were approved by the IRB of Perelman School of Medicine at the University of Pennsylvania (IRB protocol #813913).

Prevalence, Prediction and Prevention of type 2 diabetes (PPP)-Botnia Study. The study protocol was sanctioned by the Ethics Committee of Helsinki University (approval number 608/2003). All participants provided their written consent.

Pakistan Risk of Myocardial Infarction Study (PROMIS). The study was approved by the Institutional Review Board of the Center for Non-Communicable Diseases Pakistan and by regional Ethical Review Committees in the different centres across Pakistan involved in the study. Institutional Review Boards at the National Institute of Cardiovascular Disorders, Karachi, Punjab Institute of Cardiology, Lahore, and Tabba Heart Institute, Karachi approved the study. All participants provided written informed consent.

Prospective Study of Pravastatin in the Elderly at Risk (PROSPER). Approval was obtained from the Institutional Ethics Review Boards of Cork University (Ireland), Glasgow University (UK) and Leiden University Medical Center (The Netherlands). All participants gave written informed consent.

Sea Islands Genetic Network Reasons for Geographic and Racial Differences in Stroke (REGARDS). The REGARDS study protocol was approved by the institutional review boards of each participating institution, and written informed consents were obtained from all participants.

Ragama Health Study (RHS). Approval was obtained from Institutional Review Boards at the National Center for Global Health and the University of Kelaniya (P38/09/2006). All participants provided written informed consent.

Rotterdam Study (RS). Approval was granted by the Institutional review Board at Erasmus University Medical Center. All participants provided written informed consent.

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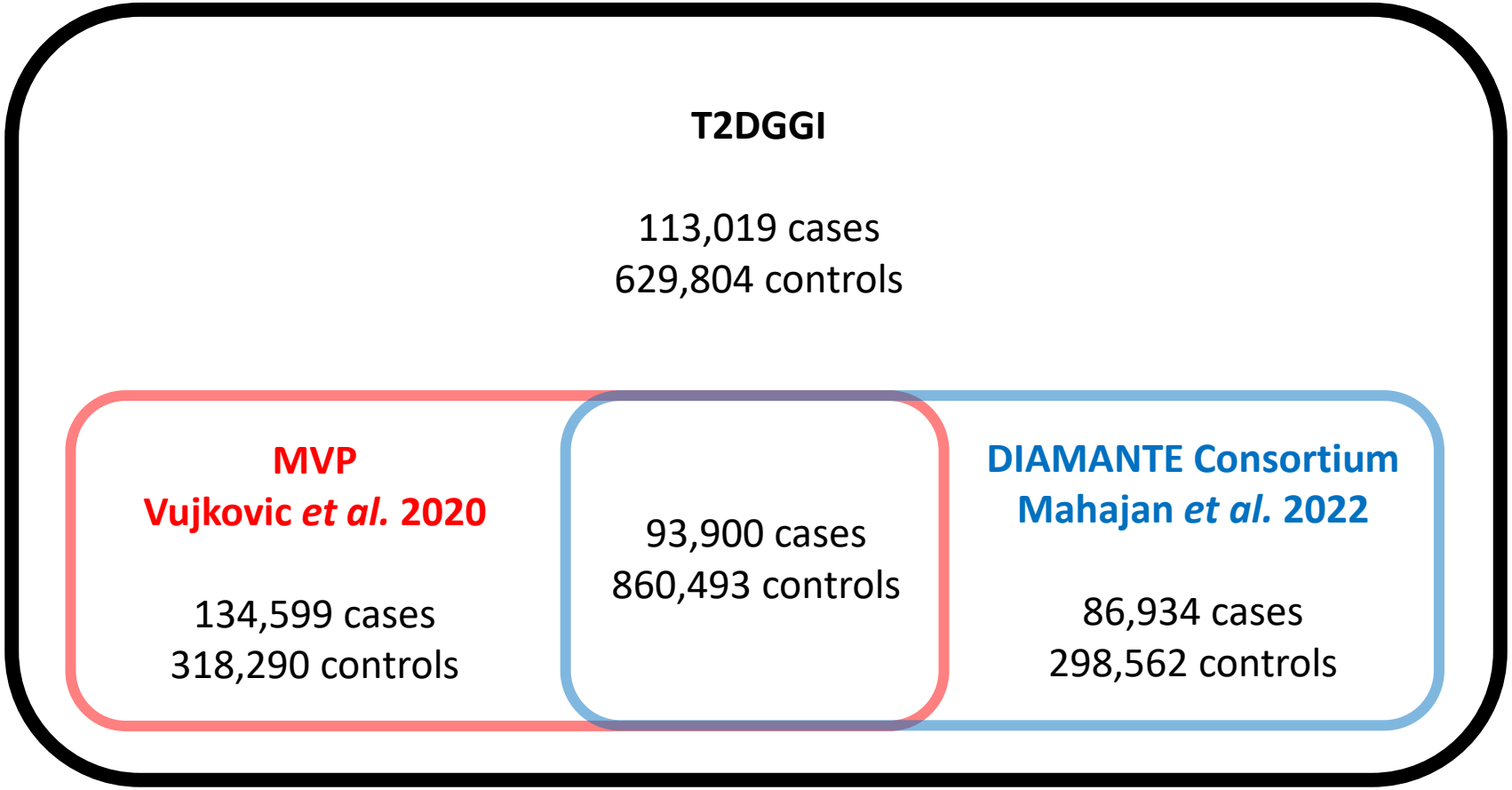
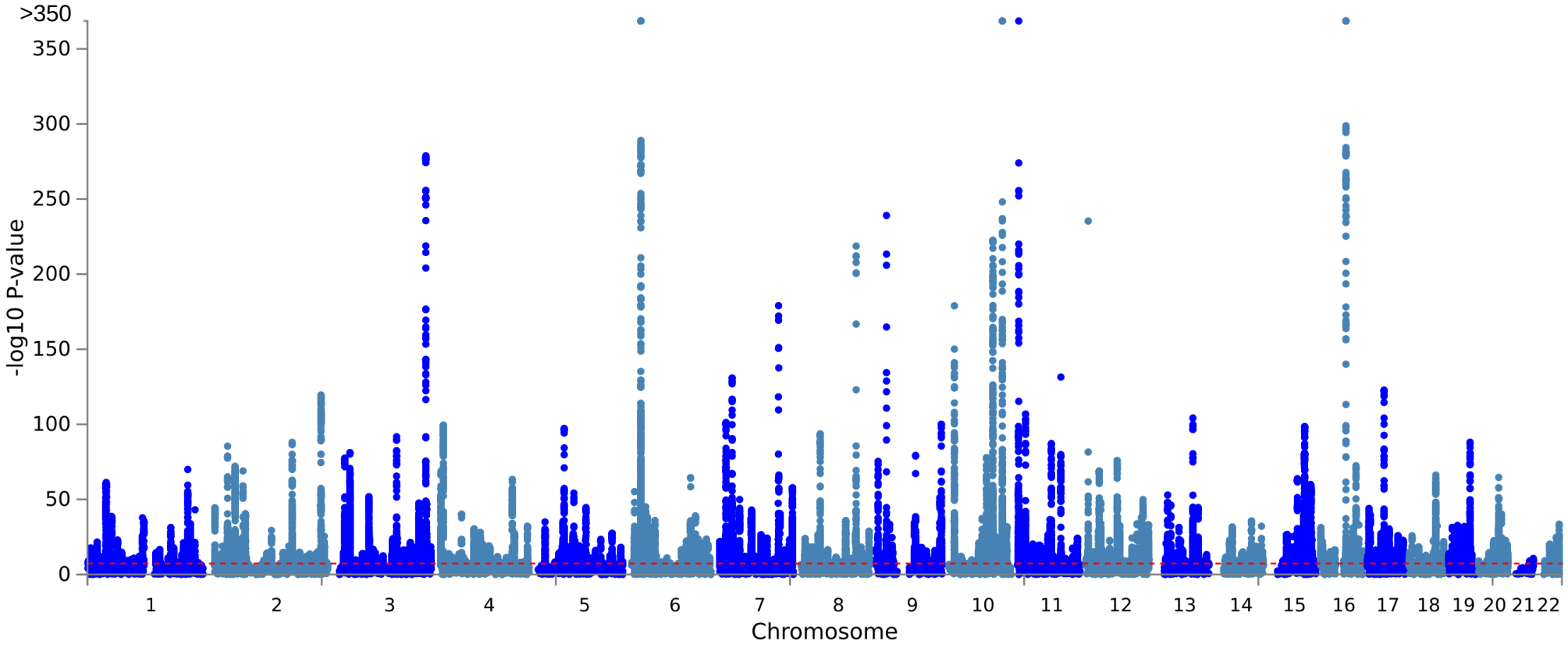
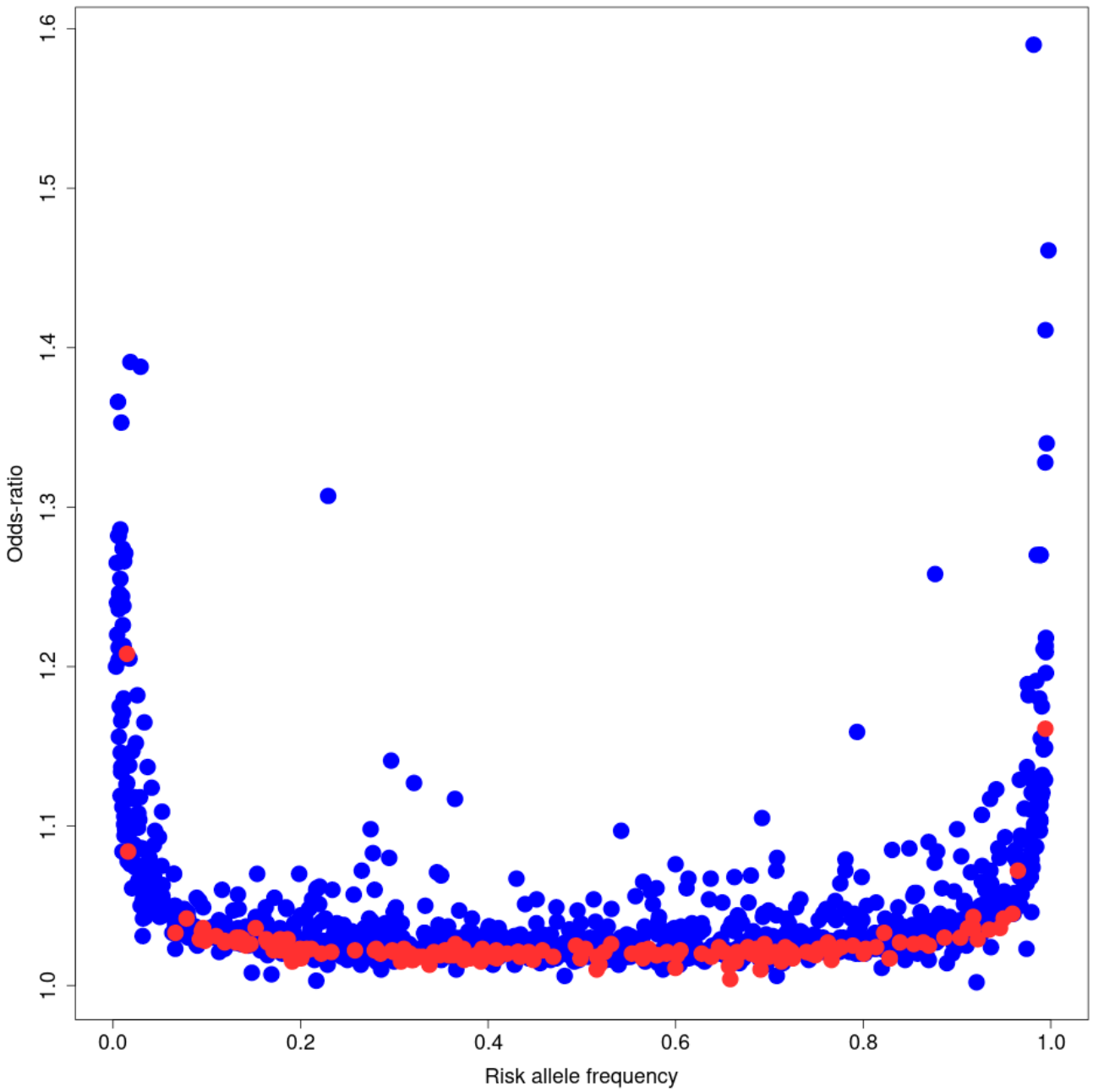
Supplementary Figure 1. Overlap of samples contributing to recent multi-ancestry T2D GWAS meta-analyses. The Type 2 Diabetes Global Genomics Initiative (T2DGGI) includes 428,452 cases and 2,107,149 controls, of which 315,433 cases and 1,477,345 controls have contributed to previous multi-ancestry investigations of the genetic contribution to T2D from the Million Veterans Program (MVP) and the DIAMANTE Consortium.

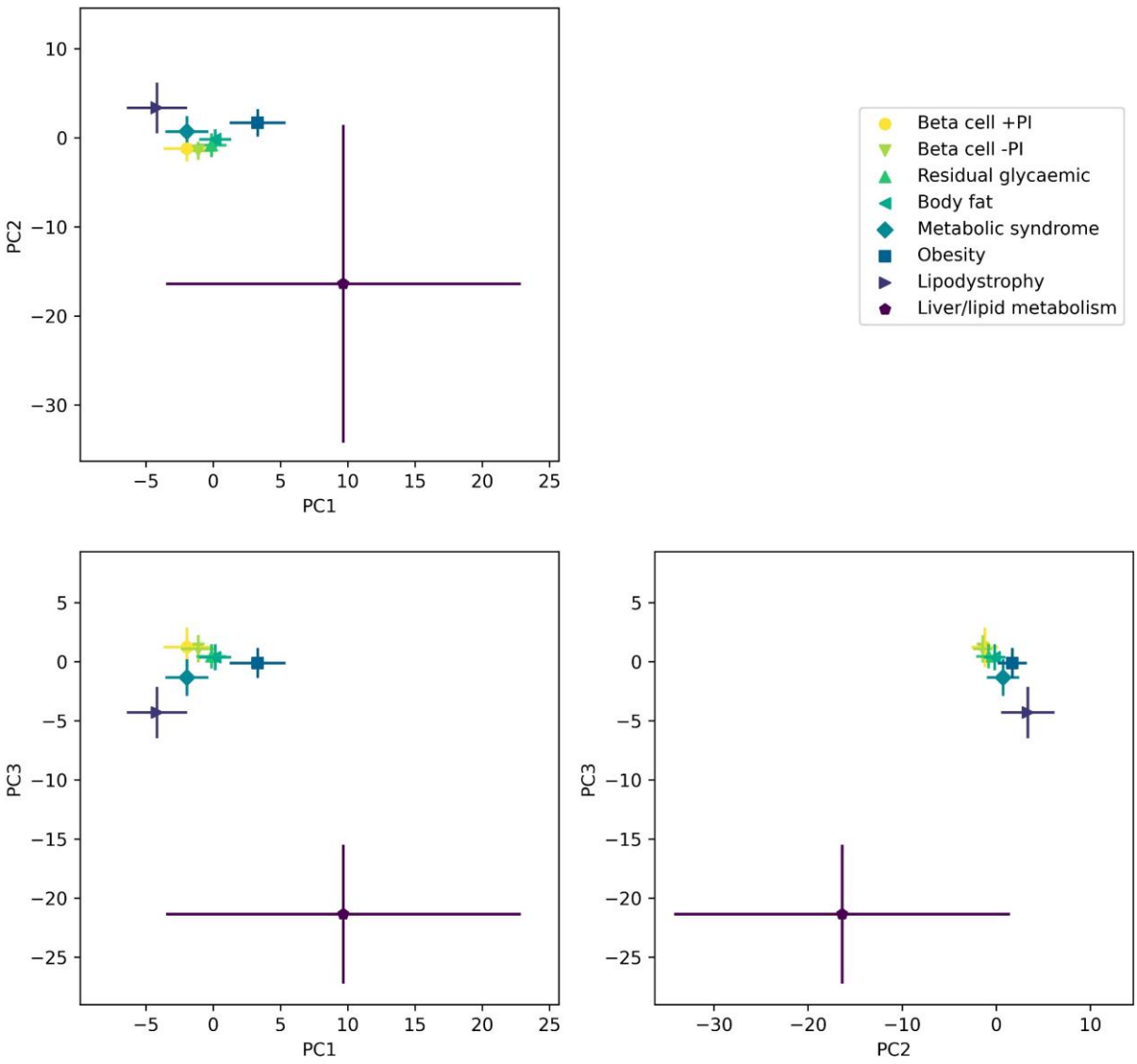




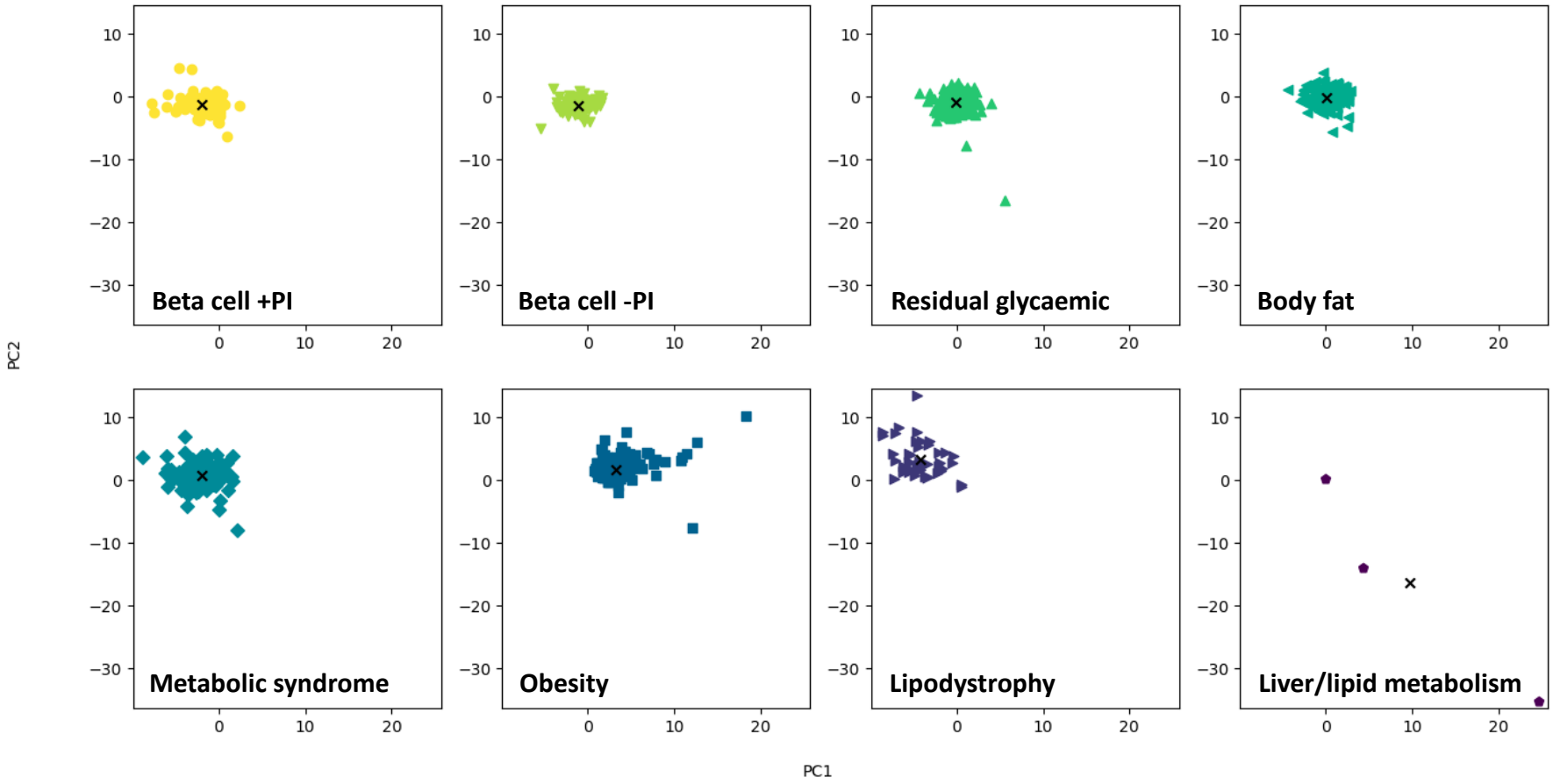
Supplementary Figure 2. Manhattan plot of genome-wide T2D association from multi-ancestry meta-regression (MR-MEGA) of up to 428,452 T2D cases and 2,107,149 controls across multiple ancestry groups. Each point represents a SNV passing quality control in the multi-ancestry meta-regression, plotted with their association p-value (on a -$\log_{10}$ scale, truncated at 300) as a function of genomic position (NCBI build 37). Genome-wide significance ($P<5x10^{-8}$) is highlighted by the dashed horizontal red line.



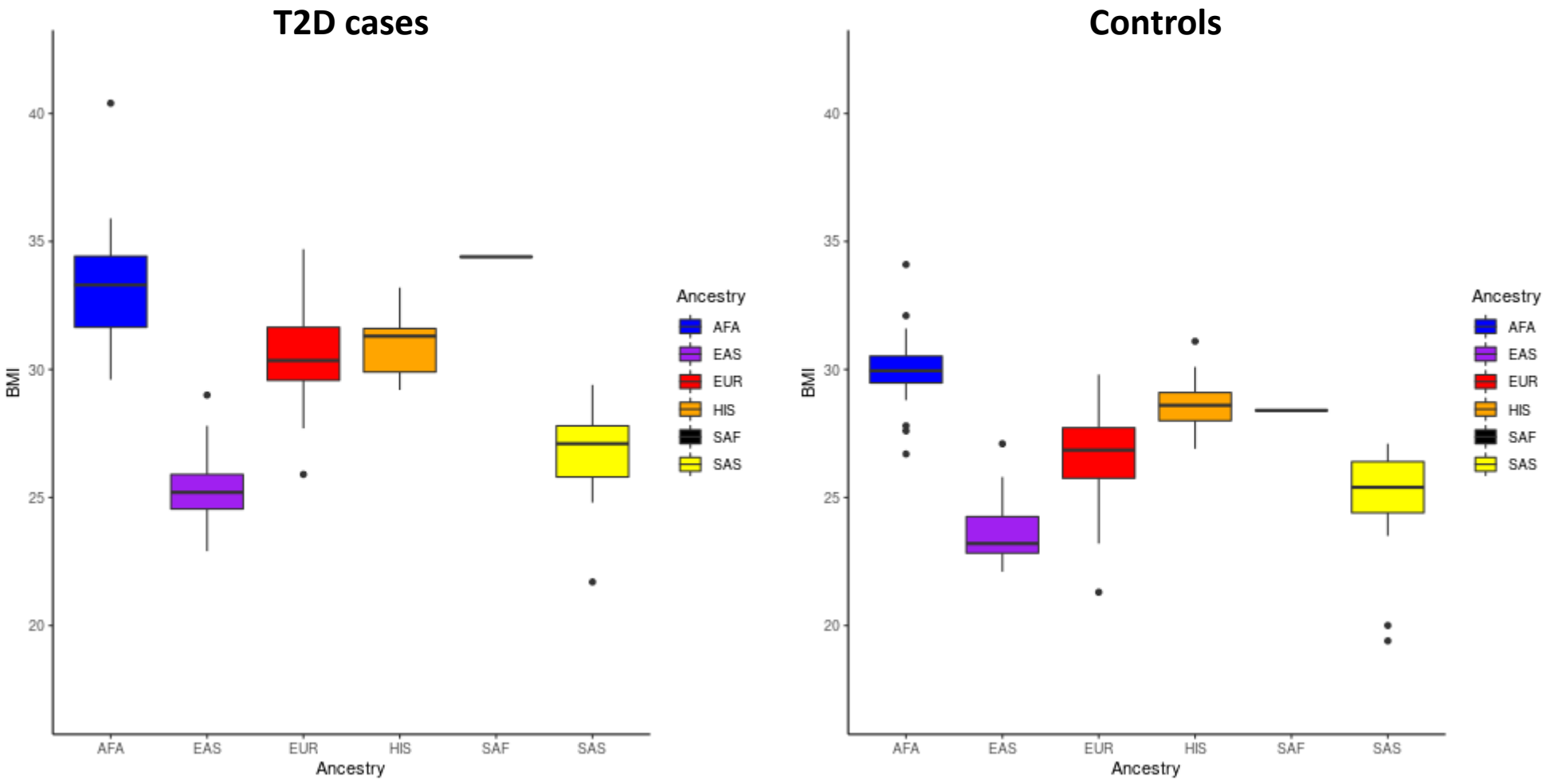
Supplementary Figure 3. Distribution of risk allele frequency and odds-ratio at index SNVs for distinct T2D association signals. Each point corresponds to an index SNV, plotted according to the mean risk allele frequency across GWAS (on the x-axis) and the odds-ratio from fixed-effects meta-analysis (on the y-axis). Index SNVs highlighted in blue map to previously reported loci for T2D susceptibility. Index SNVs highlighted in red do not map to previously reported loci for T2D susceptibility.



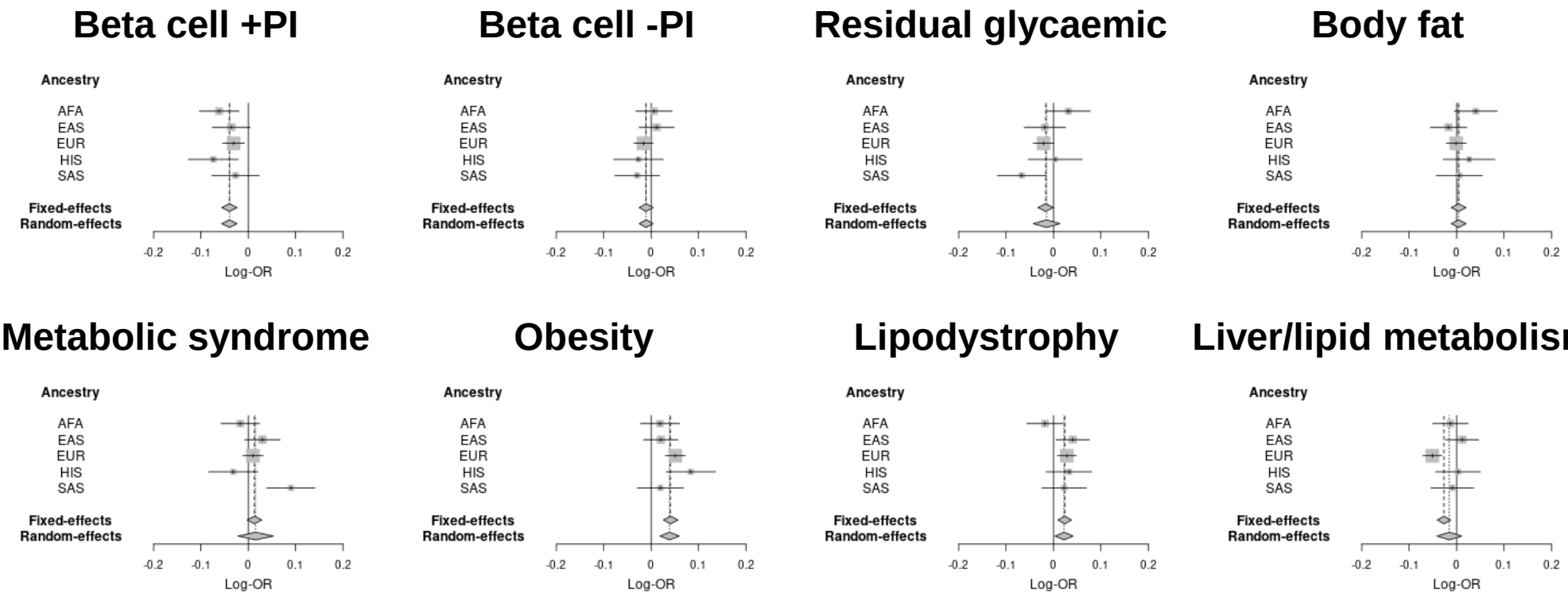
Supplementary Figure 4. Distribution of clusters of SNVs on the first three principal components derived from 37 cardiometabolic traits. The principal components analysis was conducted on the final imputed dataset obtained from K-means clustering with ClustImpute. Each point corresponds to the mean values of the first three principal components for SNVs assigned to the cluster. The bars correspond to +/- standard deviation. The percentage of variance explained by each principal component (PC) was: 16.7% by PC 1, 12.6% by PC 2, and 10.5% by PC 3.

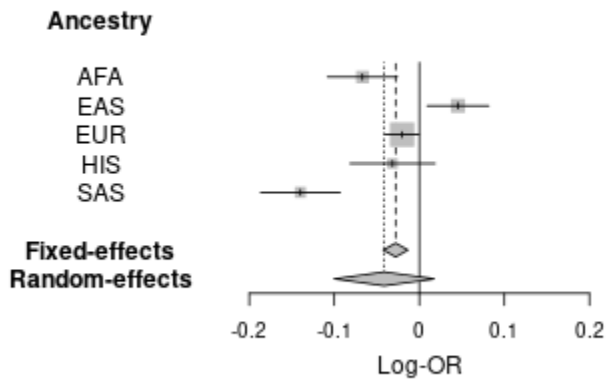


Supplementary Figure 5. Distribution of clusters of SNVs on the first two principal components derived from 37 cardiometabolic traits. The principal components analysis was conducted on the final imputed dataset obtained from K-means clustering with ClustImpute. The "X" corresponds to the cluster centroid. The percentage of variance explained by each principal component (PC) was: 16.7% by PC 1 and 12.6% by PC 2.

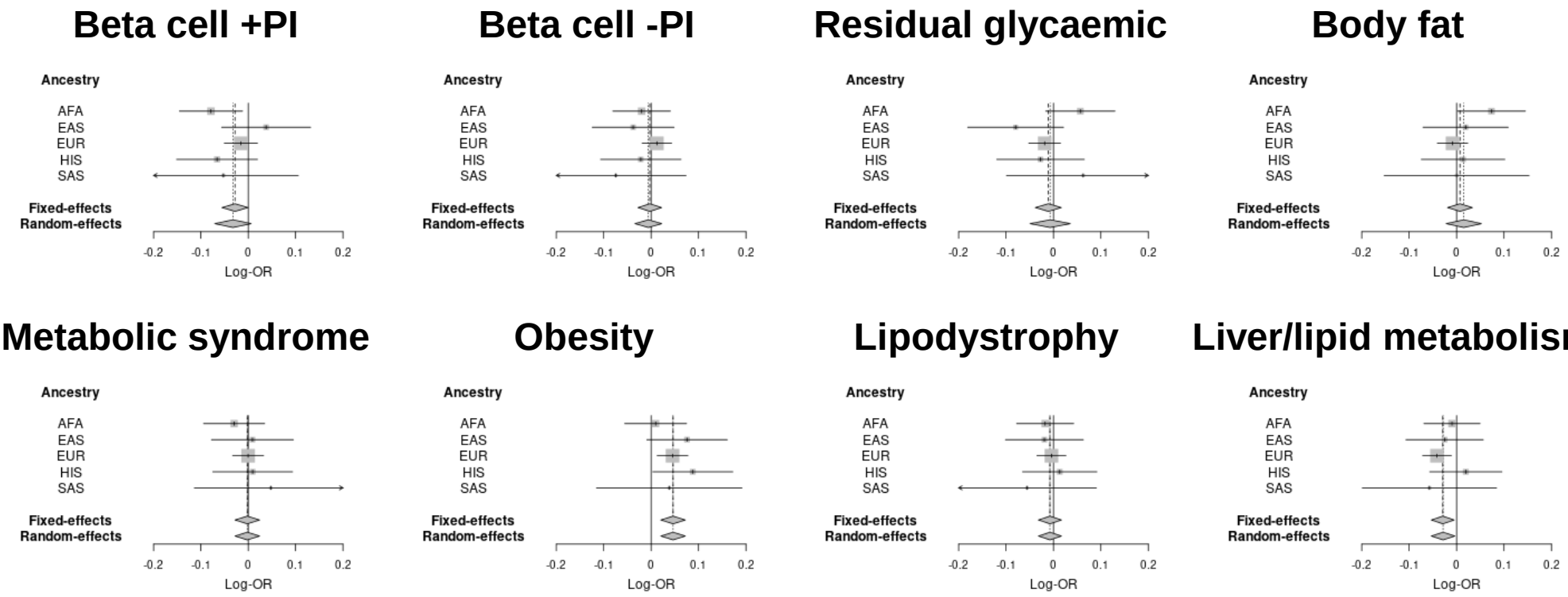


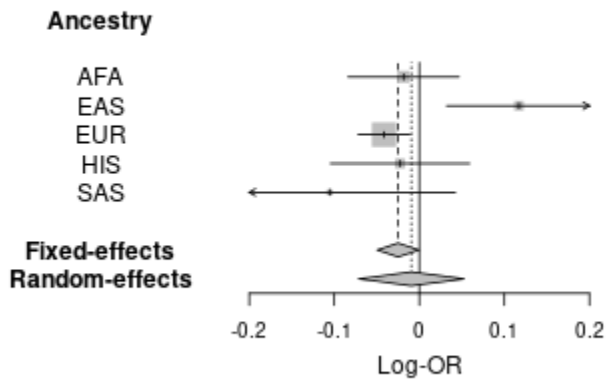
Supplementary Figure 6. Distribution of study-level mean BMI in T2D cases and controls across ancestry groups. Each box and whisker plot presents the median (back horizontal line), upper and lower quartiles (extremes of coloured boxes), minimum and maximum (excluding outliers, extremes of black vertical line), and outliers (more than 1.5x inter-quartile range, black dots). AFA: African American ancestry group (n=25 GWAS). EAS: East Asian ancestry group (n=40 GWAS). EUR: European ancestry group (n=36 GWAS). HIS: Hispanic ancestry group (n=17 GWAS). SAF: South African ancestry group (n=1 GWAS). SAS: South Asian ancestry group (n=17 GWAS).





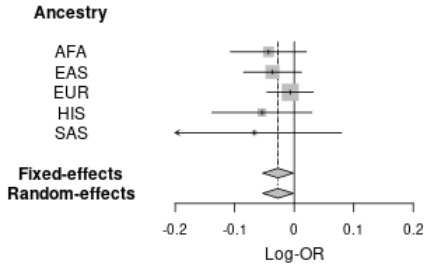
Supplementary Figure 7. Association of overall T2D PS and cluster-specific components of partitioned PS with CAD across multiple ancestry groups. In each forest plot, the log-odds ratio (log-OR) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the log-OR of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in all individuals with adjustment for T2D status. AFA: African American ancestry group (3,537 cases and 40,932 controls). EAS: East Asian ancestry group (4,078 cases and 58,904 controls). EUR: European ancestry group (13,602 cases and 96,793 controls). HIS: Hispanic ancestry group (2,171 cases and 31,612 controls). SAS: South Asian ancestry group (2,398 cases and 25,525 controls).





Supplementary Figure 8. Association of overall T2D PS and cluster-specific components of partitioned PS with peripheral artery disease across multiple ancestry groups. In each forest plot, the log-odds ratio (log-OR) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the log-OR of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in all individuals with adjustment for T2D status. AFA: African American ancestry group (1,241 cases and 43,228 controls). EAS: East Asian ancestry group (615 cases and 62,367 controls). EUR: European ancestry group (4,847 cases and 105,548 controls). HIS: Hispanic ancestry group (723 cases and 33,060 controls). SAS: South Asian ancestry group (199 cases and 27,724 controls).

Beta cell +PI



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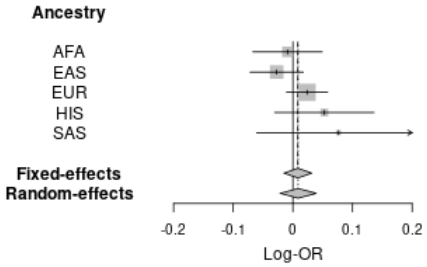
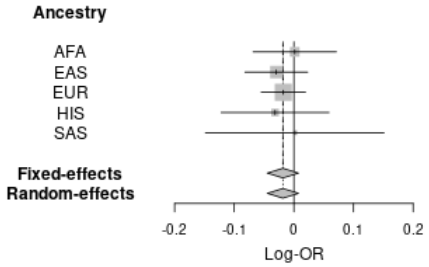
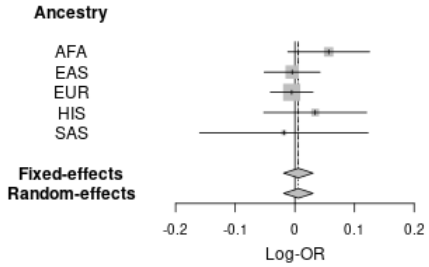


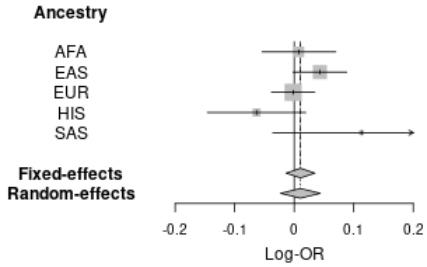
Residual glycaemic



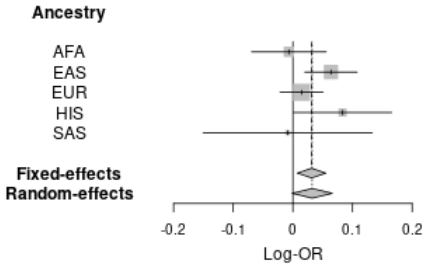
Body fat



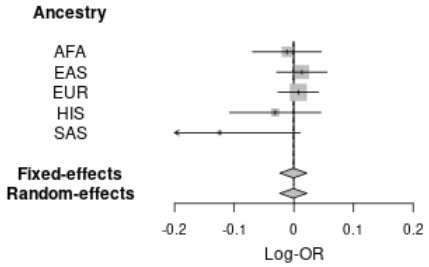
Metabolic syndrome



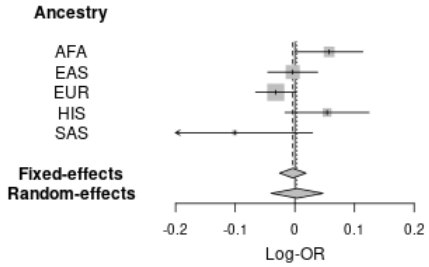
Obesity



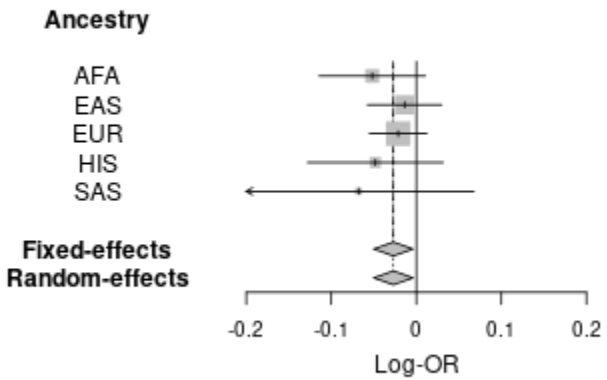
Lipodystrophy



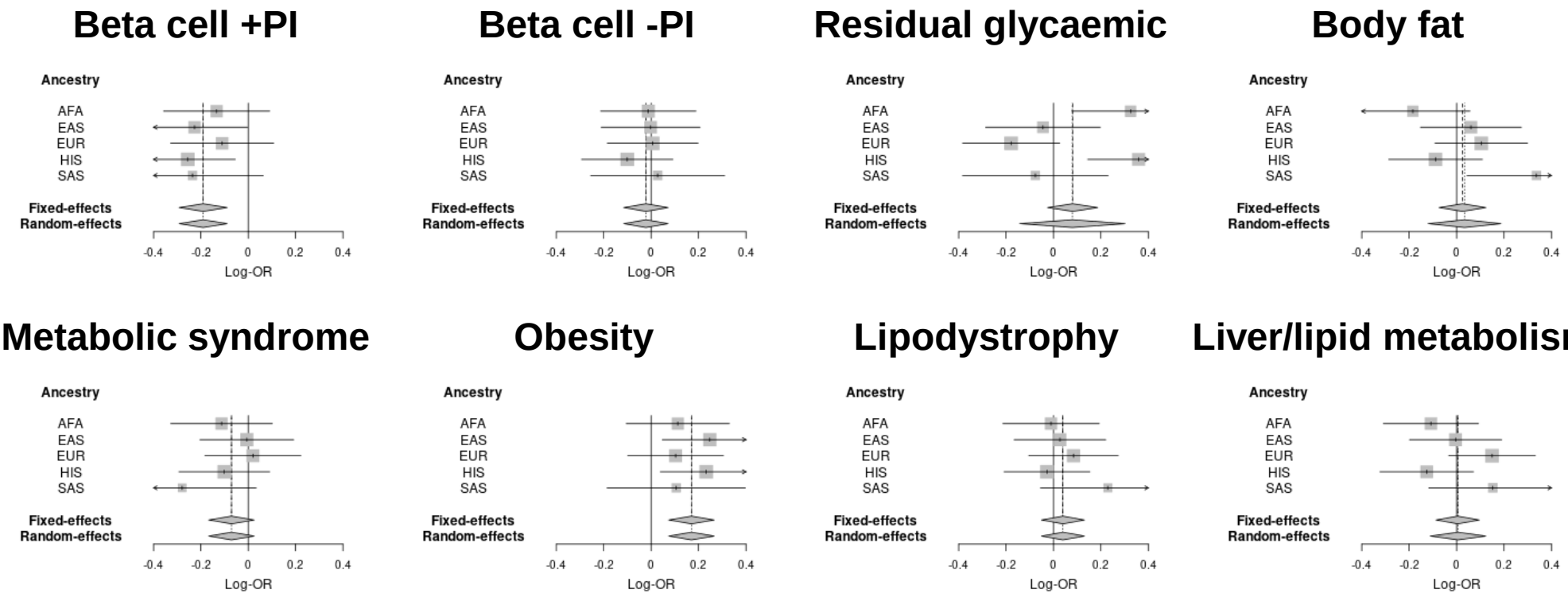
Liver/lipid metabolism

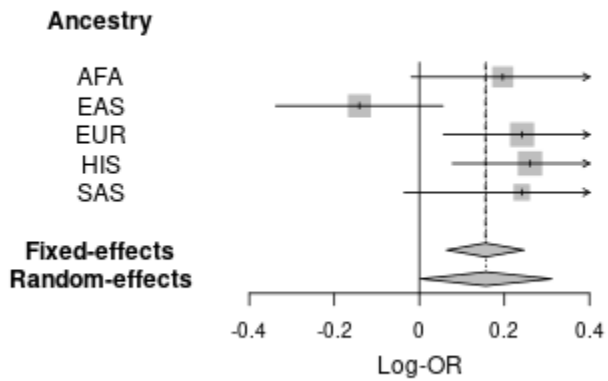


Overall

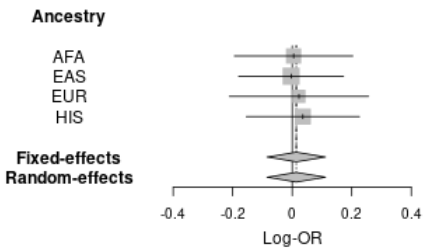


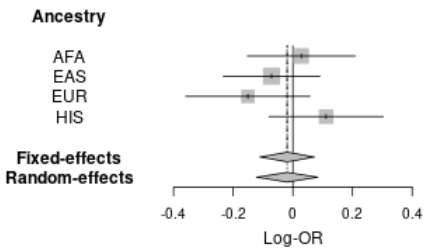
Supplementary Figure 9. Association of overall T2D PS and cluster-specific components of partitioned PS with ischemic stroke across multiple ancestry groups. In each forest plot, the log-odds ratio (log-OR) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the log-OR of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in all individuals with adjustment for T2D status. AFA: African American ancestry group (1,241 cases and 43,228 controls). EAS: East Asian ancestry group (2,396 cases and 60,586 controls). EUR: European ancestry group (3,782 cases and 106,613 controls). HIS: Hispanic ancestry group (722 cases and 33,061 controls). SAS: South Asian ancestry group (230 cases and 27,693 controls).

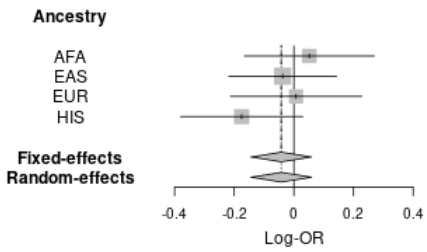


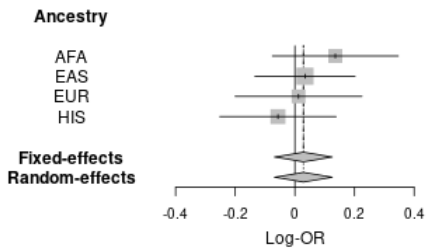


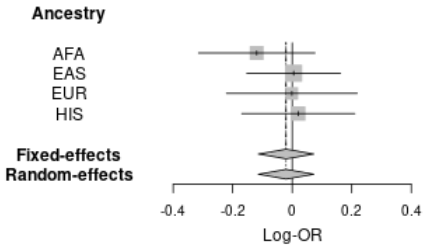
Supplementary Figure 10. Association of overall T2D PS and cluster-specific components of partitioned PS with ESDN across multiple ancestry groups. In each forest plot, the log-odds ratio (log-OR) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the log-OR of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in individuals with T2D only. AFA: African American ancestry group (105 cases and 5,330 controls). EAS: East Asian ancestry group (133 cases and 3,155 controls). EUR: European ancestry group (116 cases and 9,538 controls). HIS: Hispanic ancestry group (141 cases and 3,695 controls). SAS: South Asian ancestry group (56 cases and 8,019 controls).

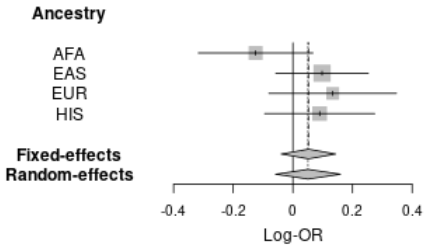


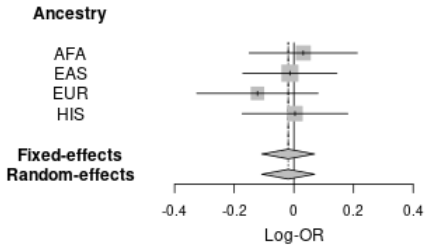


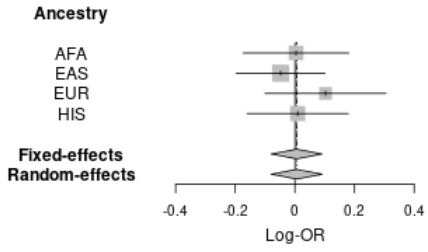


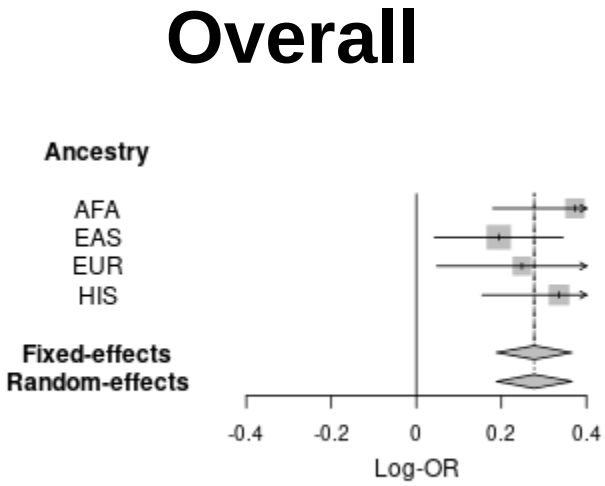




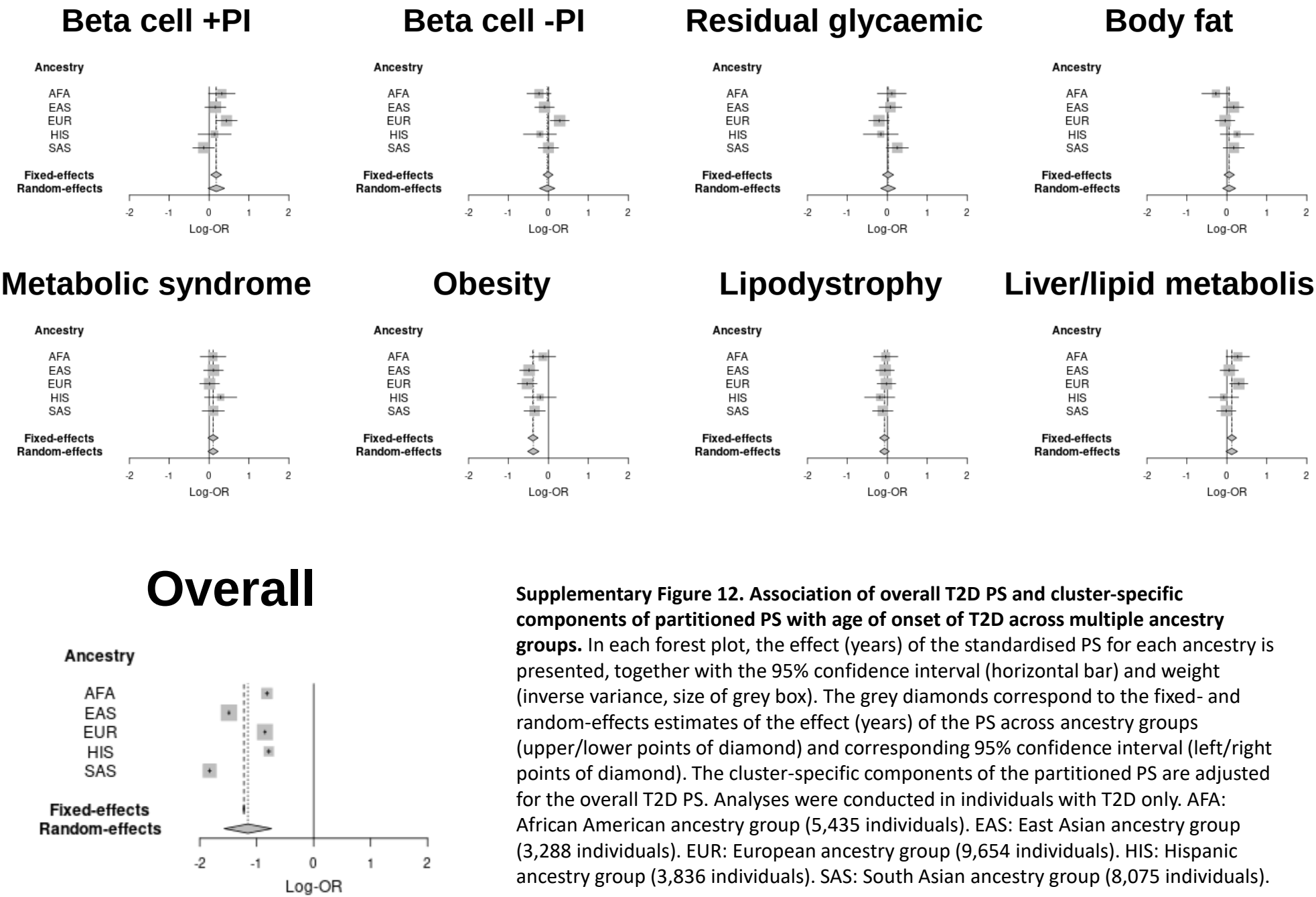




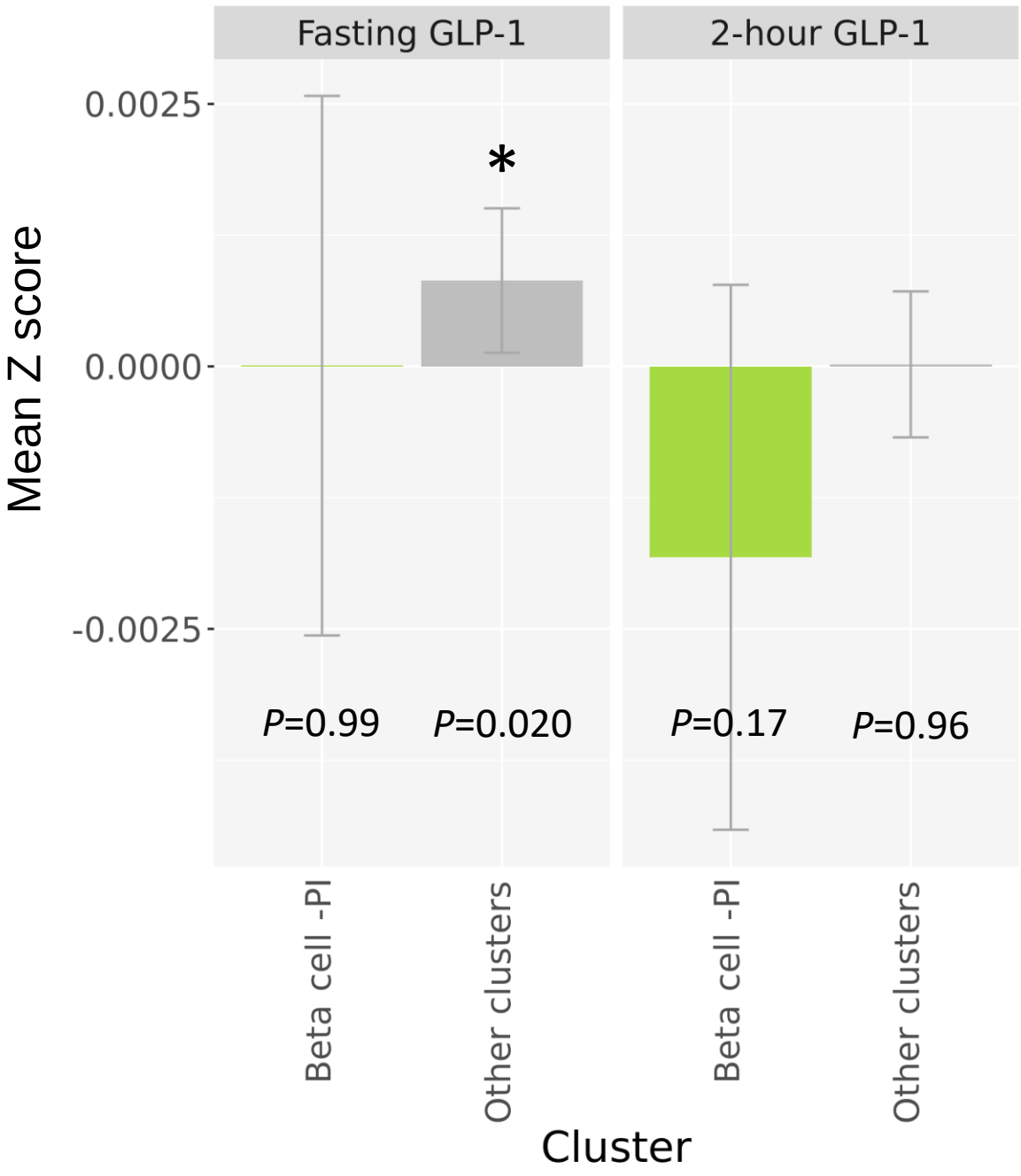




Supplementary Figure 11. Association of overall T2D PS and cluster-specific components of partitioned PS with end stage diabetic retinopathy across multiple ancestry groups. In each forest plot, the log-odds ratio (log-OR) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the log-OR of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in individuals with T2D only. AFA: African American ancestry group (132 cases and 5,072 controls). EAS: East Asian ancestry group (196 cases and 3,461 controls). EUR: European ancestry group (100 cases and 9,417 controls). HIS: Hispanic ancestry group (146 cases and 3,441 controls).



Supplementary Figure 12. Association of overall T2D PS and cluster-specific components of partitioned PS with age of onset of T2D across multiple ancestry groups. In each forest plot, the effect (years) of the standardised PS for each ancestry is presented, together with the 95% confidence interval (horizontal bar) and weight (inverse variance, size of grey box). The grey diamonds correspond to the fixed- and random-effects estimates of the effect (years) of the PS across ancestry groups (upper/lower points of diamond) and corresponding 95% confidence interval (left/right points of diamond). The cluster-specific components of the partitioned PS are adjusted for the overall T2D PS. Analyses were conducted in individuals with T2D only. AFA: African American ancestry group (5,435 individuals). EAS: East Asian ancestry group (3,288 individuals). EUR: European ancestry group (9,654 individuals). HIS: Hispanic ancestry group (3,836 individuals). SAS: South Asian ancestry group (8,075 individuals).



Supplementary Figure 13. Cluster-specific associations of T2D risk alleles at index SNVs with circulating GLP-1 concentrations. Association was assessed in 3,514 individuals of European ancestry from the Malmo Diet and Cancer Study and the PPP-Botnia Study. The height of each bar corresponds to the mean Z-score, and the grey line shows the 95% confidence interval. The liver/lipid metabolism cluster has been removed for ease of presentation. *P<0.05, nominal association.