## Supplementary material

**Supplementary methods**

Type 2 diabetes GWAS meta-analyses used for the construction of the PRSs

We leveraged the T2D GWAS summary statistics from a subset of cohorts participating in three large Consortia: Diabetes Meta-analysis of Trans-ethnic Association Studies (DIAMANTE)[1], the Million Veteran Program (MVP)[2], and the FinnGen Study.[3] An independent subset of cohorts was used for the development and validation of the PRSs. If a cohort was multi-ancestry, each individual was categorized by genetic similarity to one or more of the five ancestries available in the 1000 Genomes (1KG) Project[4] and/or the Human Genome Diversity Project[5] as reference panels: African/African American (AFR), Admixed American (AMR), East Asian (EAS), European (EUR), and South Asian (SAS). For single-ancestry cohorts, the grouping was based on the country of recruitment. We included 2,185,548 individuals (359,819 T2D cases and 1,825,729 controls) across 125 T2D GWAS to conduct ancestry-specific meta-analyses (Fig.1a, Supplementary Tables 1,2).

Each GWAS tested the association of the genetic variants with T2D adjusted for age, sex, the top genetic principal components (PCs), and cohort-specific covariates. We performed an inverse variance weighted (IVW) fixed-effect meta-analysis for each ancestry group with the METAL software.[6] We then applied quality control to keep biallelic, nonpalindromic SNPs in at least half of the effective sample size with a minor allele frequency (MAF) ≥0·01. For each ancestry-specific T2D GWAS meta-analysis, we intended to include most cohorts to maximize the sample size of the summary statistics for constructing PRSs while leaving out sufficient cohorts for each ancestry to be used for training and validation.

Cohorts for the training and validation of the PRSs

We trained the PRSs in one cohort per ancestry group and validated them in at least four validation cohorts per ancestry. All training and validation cohorts included unrelated individuals and were independent of those included in the GWAS summary statistics to avoid overfitting (Fig.1b-c, Supplementary Tables 3-6). Except for the AoU cohort, for which whole genome sequencing is available, the genotyping of the other cohorts was chip array-based. The genotypes were imputed to the 1KG[4] or the TOPMed r2[7,8] reference panels using the Michigan Imputation server.[9] We applied a separate post-imputation quality control in each cohort and ancestry to keep biallelic nonpalindromic SNPs with an imputation quality of $r^2$≥0·8 and MAF≥0·005. We excluded the variants not included in the LD reference panels, as explained below, or variants that showed an allelic frequency discordance≥0·2 compared to the 1KG ancestry-specific allelic frequency.

LD reference panels for the construction of the PRSs

To account for the correlation between variants, we constructed customized ancestry-specific LD reference panels using the same scripts used in PRS-CS[10] and PRS-CSx[11] tools. We built four new sets of ancestry-specific LD reference panels using the HapMap3 (HM3) set of variants, similar to the official panel (https://github.com/getian107/PRScsx), or an expanded 1KG set of variants, along with pairwise LD from the 1KG or in-house samples.

First, we identified ancestry-specific LD blocks using LDetect.[12] For each of the five ancestry groups in the 1KG dataset (https://mathgen.stats.ox.ac.uk/impute/impute_v2.html#reference), we selected common SNPs with MAF≥0·01 to generate a covariance matrix of variants based on the LD $r^2$ calculated in PLINK v1·9[13] and derived the boundaries of LD blocks. Second, we generated two sets of reference SNPs. One was based on the HM3 set of variants (https://www.sanger.ac.uk/data/hapmap-3/), similar to the official PRS-CS[10]/PRS-CSx[11] HM3 version. Another expanded set of variants based on 1KG (https://mathgen.stats.ox.ac.uk/impute/impute_v2.html#reference) was selected using the Tag(ging) It(erative) of SNPs in multiple populations (TagIt) program.[14] TagIt allows the selection of tag SNPs by leveraging genetic information from multiple diverse ancestries to maximize cross-population coverage. We only included non-palindromic SNPs with MAF≥0·01 in at least one ancestry group for both the HM3 and TagIt SNP lists. Third, the two sets of SNPs were extracted in each ancestry for individuals from the 1KG dataset (347 to 661 individuals per ancestry) and from the larger imputed in-house datasets (including around 10,000 individuals per ancestry). SNPs with low imputation quality ($r^2$<0·8) were further excluded in the in-house datasets. Last, we calculated the variants' pairwise LD ($r^2$) using PLINK v1·9 to generate ancestry-specific LD reference panels. In total, for each ancestry, we built four different LD reference panels and used them to construct the PRSs, combining two sets of variants (i.e., HM3 and TagIt) and two sources of LD information (i.e., 1KG and in-house samples) (Supplementary Table 7).

Training of the PRSs

We used the PRS-CS[10] Bayesian polygenic method to construct single-ancestry PRSs. For each ancestry group, we leveraged the T2D GWAS summary statistics and LD reference panels matching the ancestry of the validation cohort (e.g., a PRS trained using the AMR GWAS and AMR LD reference panel to be validated in AMR cohorts). When applicable, we also modeled non-matched single-ancestry PRSs (e.g., a PRS trained using the EUR GWAS and EUR LD reference panel to be evaluated in AMR cohorts). The PRS-CS method returns a single-ancestry posterior variant effect size.

We then used PRS-CSx[11] to construct multi-ancestry PRSs. Instead of meta-analyzing the ancestry-specific GWAS summary statistics, PRS-CSx jointly models the GWAS summary statistics along with their matching LD reference panels, using a shared continuous shrinkage prior, to generate ancestry-specific variant posterior effect sizes in a coupled manner that leverages cross-population genetic architecture. We used these ancestry-specific effect sizes to compute standardized ancestry-specific z-scores, which were then combined in a linear regression model to derive the multi-ancestry posterior variant effect size as follows:

$$y = PRS_{\theta,AFR} + PRS_{\theta,AMR} + PRS_{\theta,EAS} + PRS_{\theta,EUR} + PRS_{\theta,SAS}$$

Where y is the T2D status, and $PRS_{\theta,\text{ ancestry group}}$ is the standardized PRS for a given shrinkage prior and ancestry.

For both PRS-CS and PRS-CSx, we used the training cohorts to select the optimal continuous shrinkage prior from five phi values (i.e., 0·01, 0·001, $1x10^{-4}$, $1x10^{-5}$, $1x10^{-6}$) based on predictive performance. In total, we constructed 80 PRSs for each of the AFR, AMR, and SAS ancestry groups (i.e., 1 matched-ancestry PRS, 2 non-matched ancestry PRSs, and 1 multi-ancestry PRS × 4 LD panels × 5 phi values = 80 models) and 60 PRSs for each of the EAS and EUR ancestry groups (i.e., 1 matched-ancestry PRS, 1 non-matched ancestry PRS, and 1 multi-ancestry PRS × 4 LD panels × 5 phi values = 60 models), resulting in 360 PRS models overall.

To test the predictive performance, we applied the posterior variant effect sizes for each PRS model to calculate the individual scores in each of the five training cohorts using the --score function in PLINK v1·9.[9] We standardized them to have a mean of zero and unit variance. Then, we fitted two logistic regression models and calculated the area under the receiver operator characteristic curve (AUC) using the "pROC" package[15] in R. One model included the explanatory variables sex, age, and genetic PCs, and a second full model also included the standardized PRS. We also fitted logistic regression models adjusted for BMI.

We calculated the incremental AUC (iAUC) by subtracting the AUC of the model without the PRS from the AUC of the full model. We defined the best-trained PRS models as those with the continuous shrinkage prior and LD panel that maximized the iAUC. After the training step, we ended up with four best-trained PRS models for the AFR, AMR, and SAS ancestries (i.e., one matched-ancestry PRS, one EUR non-matched ancestry PRS, one EAS non-matched ancestry PRS, and one multi-ancestry PRS), 3 best-trained PRS models for the EUR ancestry (i.e., one single matched-ancestry PRS, one EAS non-matched ancestry PRS, and one multi-ancestry PRS), and 3 best-trained PRS models for the EAS ancestry (i.e., one single matched-ancestry PRS, one EUR non-matched ancestry PRS, and one multi-ancestry PRS) (Supplementary Table 8).

Validation of the PRSs

To validate each of the 18 best-trained PRSs, we applied the posterior variant effect sizes in a second set of unrelated samples and independent cohorts from each ancestry group. We calculated the individual scores in each validation cohort using the --score function in PLINK 1·9[13] and standardized them to have a mean of zero and unit variance. For the multi-ancestry PRS, we combined ancestry-specific standardized scores weighted for the trained ancestry-specific effect sizes as follows:

$$y = \beta_{\theta,AFR}\, PRS_{\theta,AFR} + \beta_{\theta,AMR}\, PRS_{\theta,AMR} + \beta_{\theta,EAS}\, PRS_{\theta,EAS} + \beta_{\theta,EUR}\, PRS_{\theta,EUR} + \beta_{\theta,SAS}\, PRS_{\theta,SAS}$$

Where y is T2D status, $\beta_{\theta,\text{ ancestry group}}$ is the regression coefficient for a given shrinkage prior and ancestry in the training cohort, and $PRS_{\theta,\text{ ancestry group}}$ is the standardized PRS for a given shrinkage prior and ancestry.

To test the predictive performance of the PRS, we calculated i) the iAUC as explained above, ii) the proportion of the variation in the T2D status explained by the PRS using Nagelkerke's $r^2$, iii) the odds ratio per standard deviation (OR per SD) change in the PRS, and iv) the discrimination capacity at the extremes of the PRS distribution by identifying

the individuals at the top 97·5$^{th}$ percentile, 95$^{th}$ percentile and 90$^{th}$ percentile for comparison with the interquartile range.

We applied the DeLong test to statistically assess the difference between AUCs of the single ancestry vs. multi-ancestry PRSs. We combined the PRS estimates across validation cohorts using fixed-effects meta-analyses by ancestry, weighting each cohort's beta coefficient by the inverse of its variance, using the "metafor" package[16] in R (Supplementary Tables 9,10).

Comparison of the best-performing multi-ancestry PRSs to other publishedT2D PRS

Multiple efforts have been made to improve the portability of PRS to apply to individuals from diverse ancestries. Until the preparation of this work (revised on October 07, 2024), 147 PRSs for T2D had been constructed and made publicly available through the PGS catalog.[17] To compare the performance of our best-trained PRS models using PRS-CSx (which we refer to as 'D-PRISM multi-ancestry PRS-CSx' model), we selected 55 PRSs from the PGS catalog that i) were trained for the overall T2D trait and not for any specific subtype of the disease, ii) were trained considering all types of genetic variants and not any specific set of variants for specific biological mechanisms, and iii) were constructed and trained by leveraging genetic information from cohorts other than the AoU, which we used as a validation cohort in this study.

We downloaded the PRSs and extracted the variants from the AoU cohort[18] (release v7, May 2022). Since the variant missingness rate was below 10% for all PRSs, we included all of them for testing. We calculated the individual scores in each of the five ancestries using the --score function in PLINK v1·9[13] and standardized them to mean zero and unit variance. Then, we tested the performance of the PRSs using the same procedure as for the validation cohorts.

To assess the added value of the PRS-CSx approach over constructing a PRS from meta-analyzed multi-ancestry GWAS results, we performed an IVW meta-analysis of D-PRISM ancestry-specific GWAS summary statistics and applied PRS-CS to the resulting meta-analysis. We used the EUR 1KG HM3 LD reference panel and allowed the shrinkage prior to be automatically learned from the data (which we refer to as 'D-PRISM multi-ancestry PRS-CS' model).

Similarly, we also evaluated the performance of a PRS based on the most statistically powered multi-ancestry T2D GWAS to date from Suzuki et al.[19,] including a 16% larger sample size than this study (i.e., Suzuki et al.: 2,535,601 individuals of which 428,452 are T2D cases and 2,107,149 controls vs. D-PRISM: 2,185,548 individuals of which 359,819 are T2D cases and 1,825,729 controls). We first applied the same quality control steps as those used for D-PRISM GWAS summary statistics and then used PRS-CS to construct a PRS using the EUR 1KG HM3 LD reference panel and let the prior shrinkage prior to be automatically learned from the data (which we refer to as 'Suzuki et al., PRS-CS' model).

Additionally, we constructed an rsPRS using the 1,289 distinct variants identified by Suzuki et al.,[19] defined as having an association $p<5 \times 10^{-8}$. For 90 out of 203 palindromic variants, we used proxy variants ($r^2 \geq 0{\cdot}8$ in all the ancestry groups). We excluded 113 variants, as no proxy was available, thereby using 1,176 total variants to construct the rsPRS (which we refer to as 'Suzuki et al., rsPRS' model) (Supplementary Table 11).

Association of PRSs with diabetes complications

We also evaluated the association of the best-performing D-PRISM multi-ancestry PRS-CSx models with T2D-related microvascular complications (i.e., diabetic nephropathy, diabetic retinopathy, end-stage diabetic nephropathy, and proliferative diabetic retinopathy) and macrovascular complications (i.e., cardiovascular disease and ischemic stroke) in the AoU validation cohort. To comply with the AoU policies, we only considered individuals of the AFR, AMR, and EUR ancestry groups, as there were limited sample sizes for the EAS and SAS ancestries (i.e., <30 individuals with diabetes complications). We defined the traits based on ICD9 and ICD10 codes, as previously described.[19] We restricted microvascular complication analyses to individuals with T2D, as these outcomes are largely diabetes specific. For macrovascular complications, which also occur in those without T2D, we included all individuals and adjusted for T2D status in the models. We tested the association of each T2D-related complication with the standardized PRSs by fitting logistic regression models adjusted for sex, age, and genetics PCs in each ancestry group, separately (Supplementary Table 12).

Method's references

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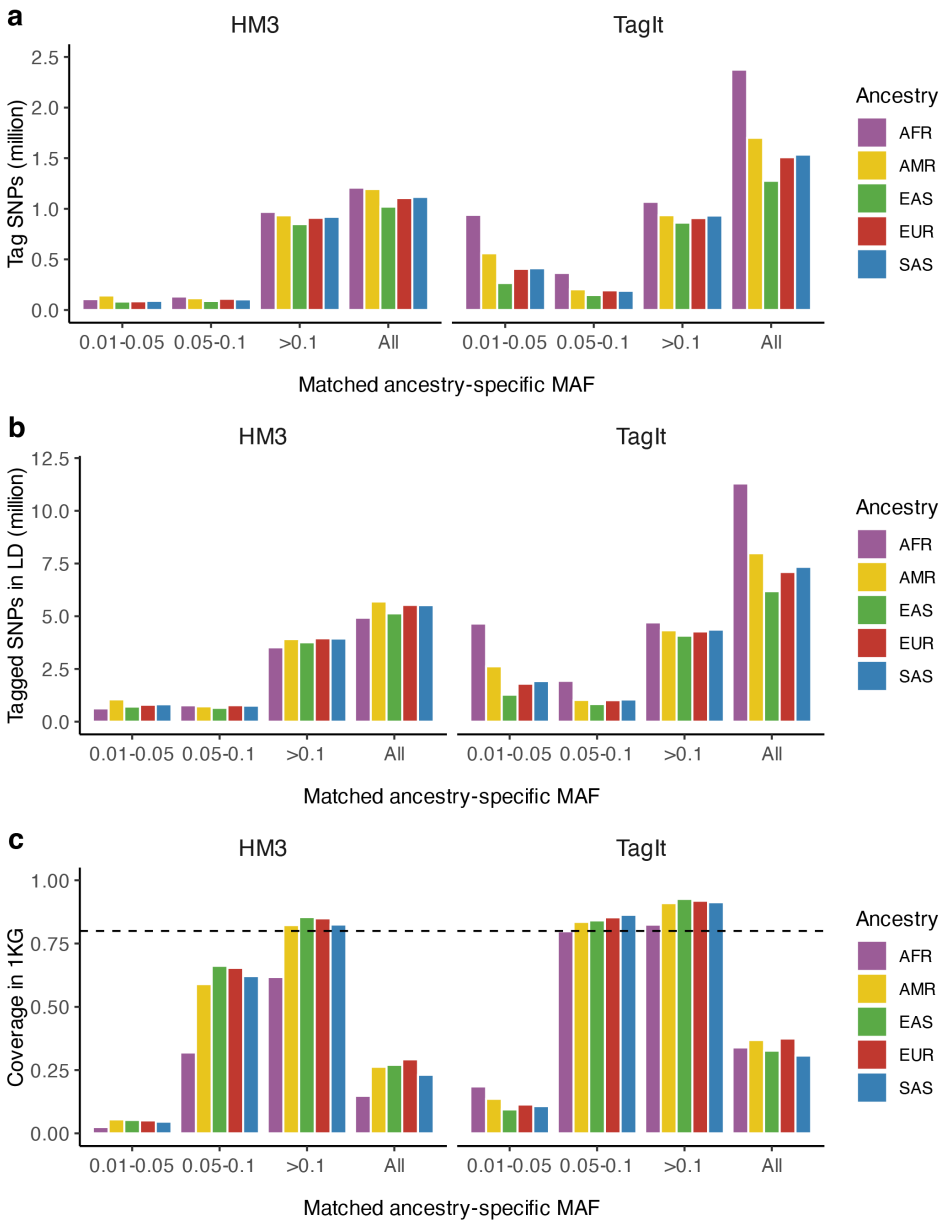
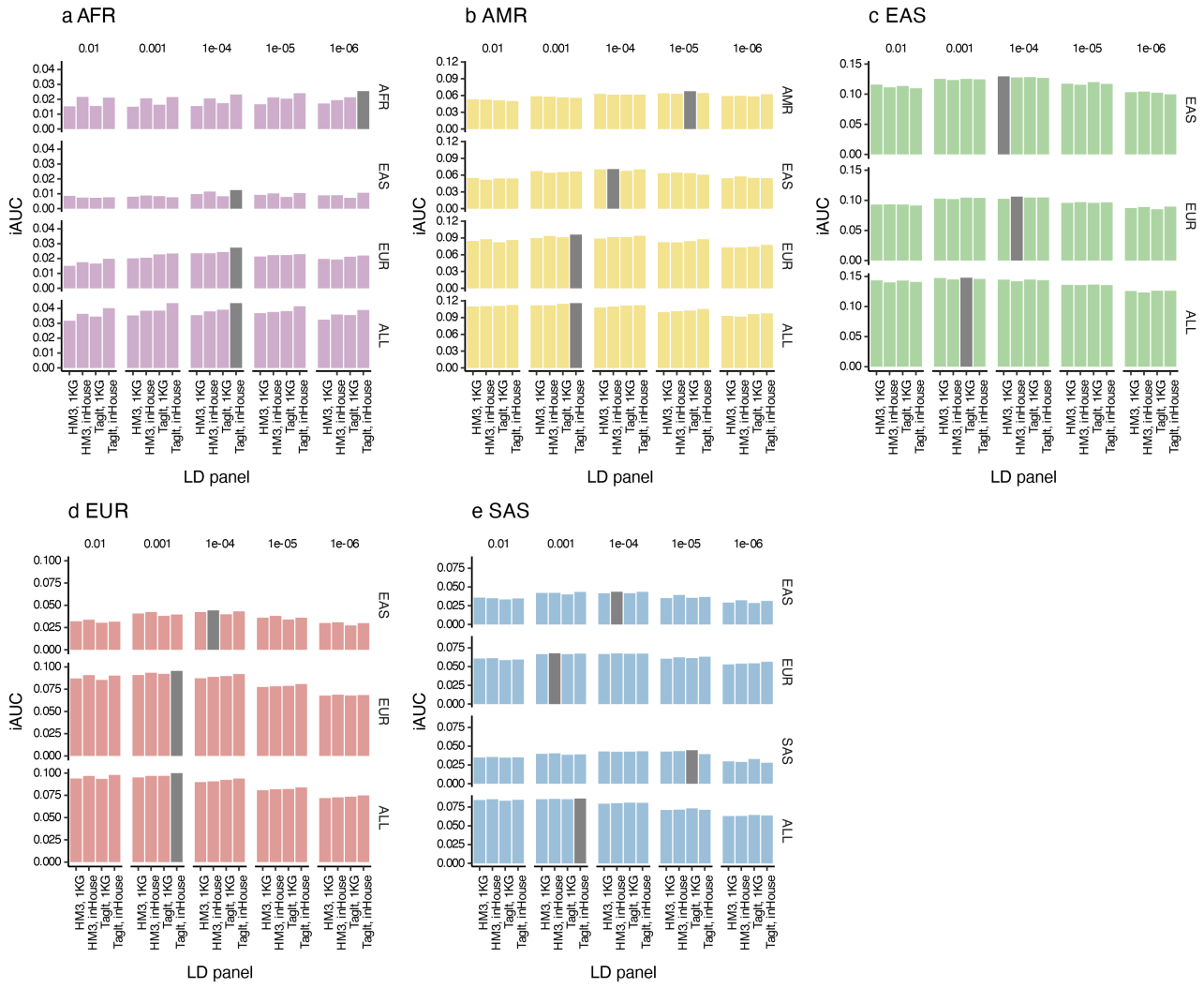
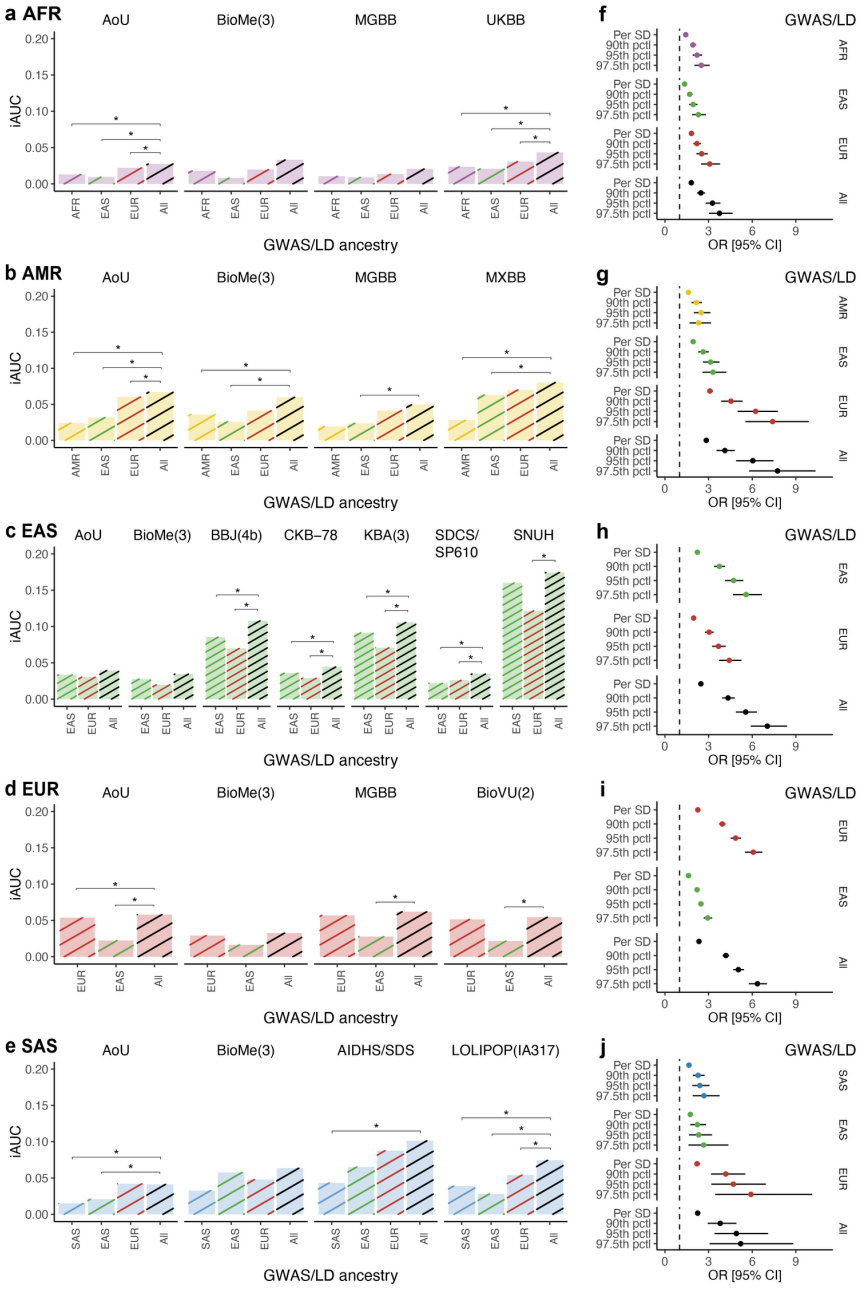
**Supplementary Figures**



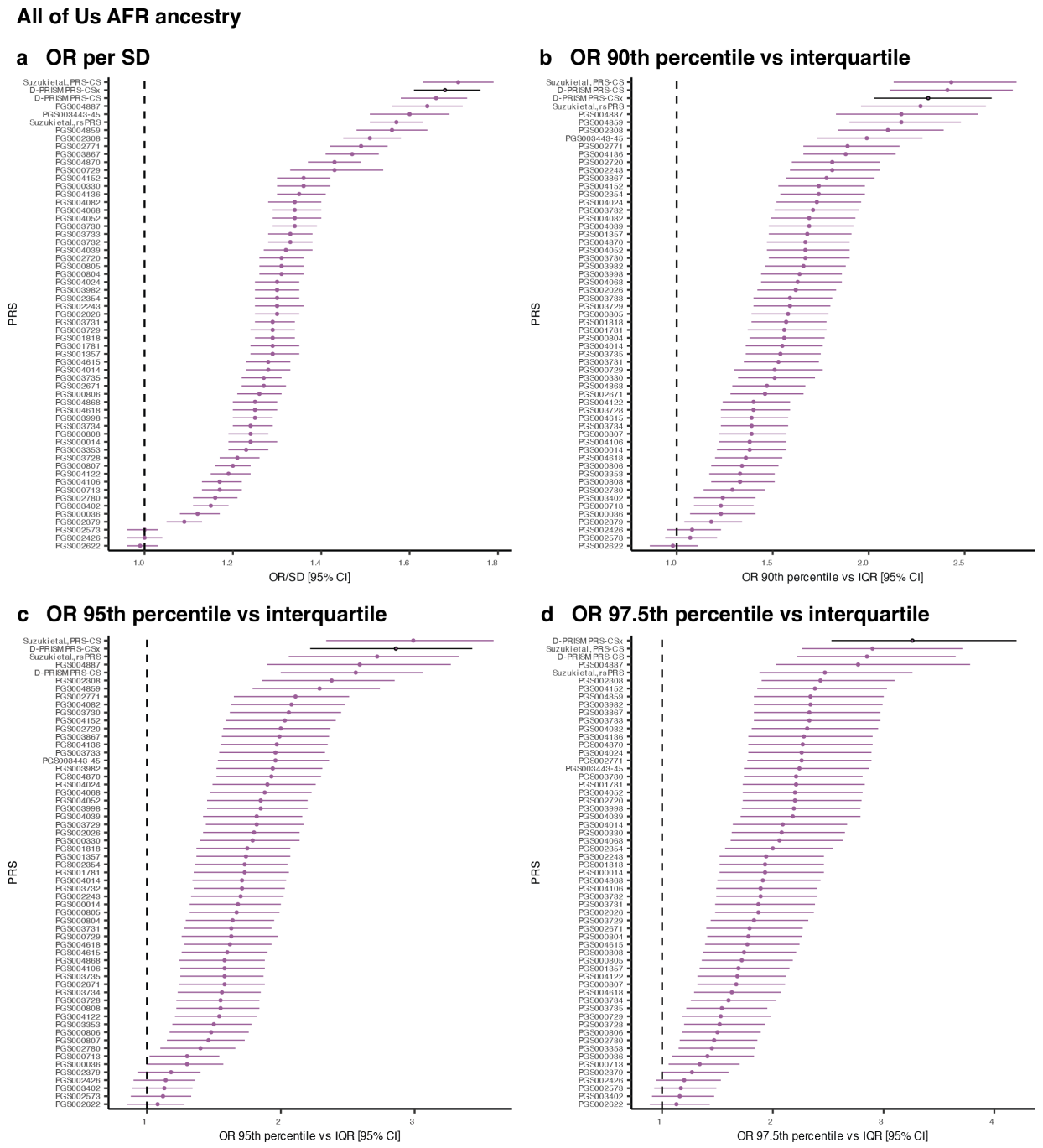

**Supplementary Fig.1 | Tag SNPs informativeness across ancestries.** We considered two sets of reference SNPs: one was based on the HapMap3 (HM3) set of variants, and the other was selected using the TagIt program. **a,** Number of tag SNPs in the LD reference panels, stratified by minor allele frequency and ancestry (*left*, HM3-based; *right*, TagIt-based), **b,** Number of SNPs being tagged in the LD reference panels, stratified by minor allele frequency and ancestry for a minimum pairwise correlation threshold of $r^2$>0.8 (*left*, HM3-based; *right*, TagIt-based), **c,** Proportion of SNPs that are either tags or are tagged ($r^2$>0.8) in the LD reference panels, stratified by minor allele frequency and ancestry in the individuals from 1KG. The dashed line represents 80% coverage.



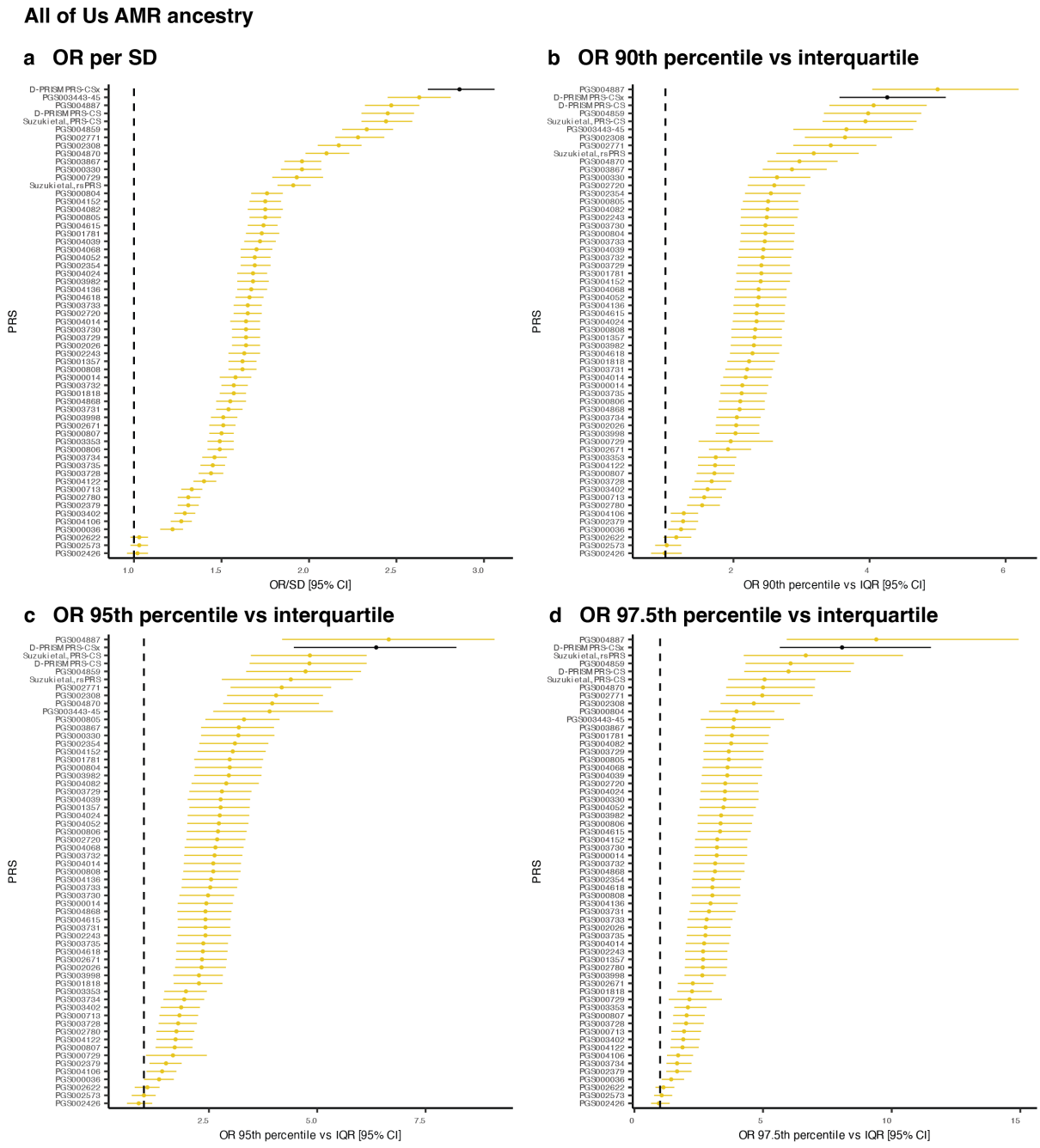

**Supplementary Fig.2 | Performance of the T2D PRSs in the training cohorts across ancestry groups.** Incremental AUC (iAUC) of the T2D PRS in the training cohorts: **a**, AFR, **b,** AMR, **c,** EAS, **d,** EUR, **e,** SAS. For each ancestry, we trained single-ancestry and multi-ancestry (ALL) PRSs using four LD panels and 5 phi continuous shrinkage priors. Bar colors represent the ancestry group: purple for AFR, yellow for AMR, green for EAS, red for EUR, and blue for SAS. The grey color highlights the best-trained PRS models that maximize the iAUC.



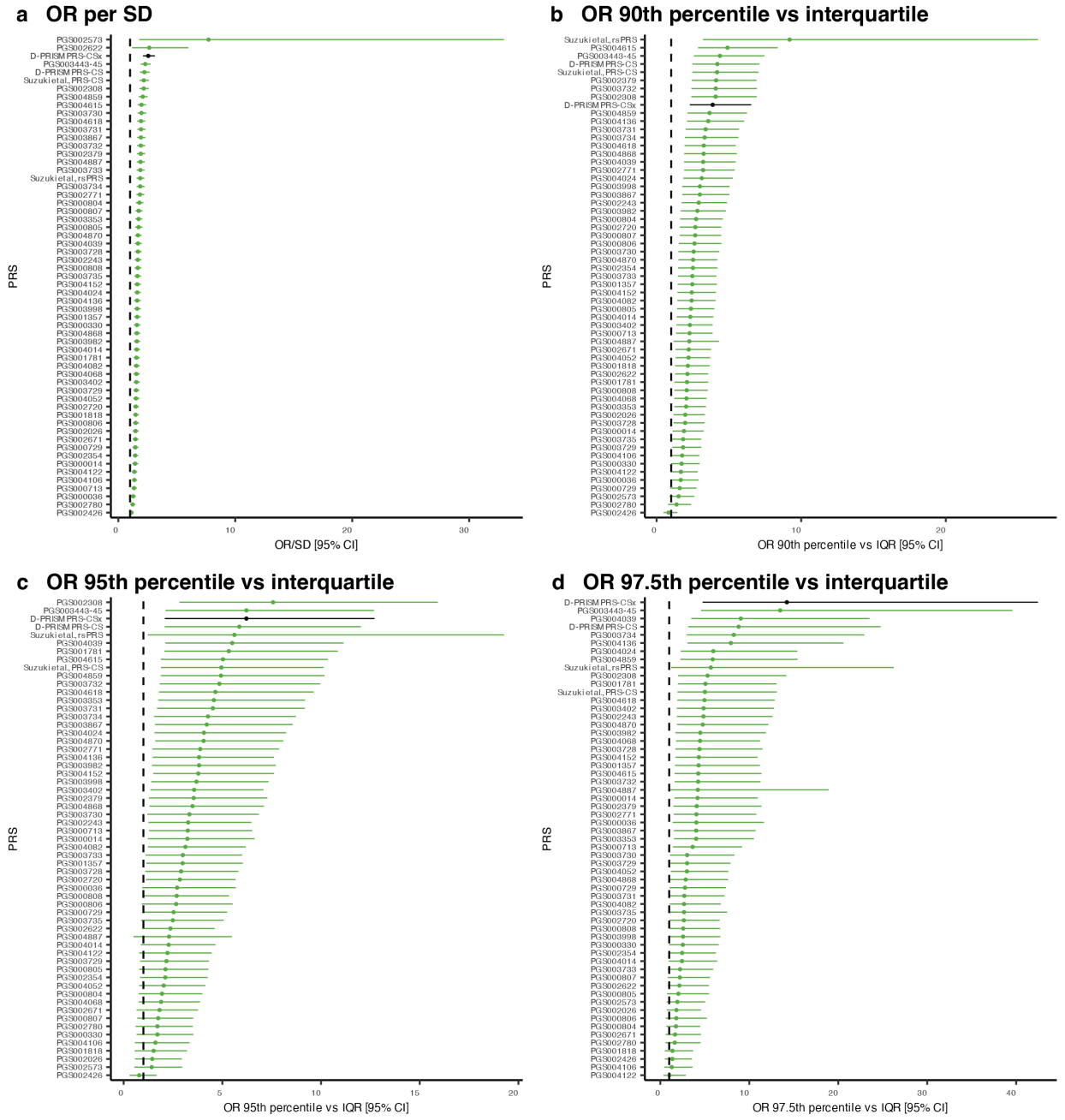

**Supplementary Fig. 3 | Performance of the T2D PRSs adjusted for BMI in the validation cohorts across ancestry groups. a-e:** Incremental AUC (iAUC) of the T2D PRS in the validation cohorts across ancestry groups: **a,** AFR, **b,** AMR, **c,** EAS, **d,** EUR, **e,** SAS. For each ancestry, the best-performing single-ancestry and multi-ancestry (All) PRSs were evaluated. Each bar represents a single cohort. Bar colors represent the ancestry group: purple for AFR, yellow for AMR, green for EAS, red for EUR, and blue for SAS. Line colors represent the ancestry of the T2D GWAS summary statistics and LD panels used to train the PRS, using the same color codes for single-ancestry PRSs, and black for multi-ancestry PRSs. **f-j:** Odds ratio (OR) from the meta-analysis of validation cohorts across ancestry groups: **f,** AFR, **g,** AMR, **h,** EAS, **i,** EUR, **j,** SAS. Points represent the odds ratio per standard deviation of the PRS distribution or the odds ratio comparing different PRS distribution extremes relative to the interquartile range. Error bars show the 95% confidence intervals (95% CI). Point colors represent the ancestry of the T2D GWAS summary statistics and LD panels used to train the PRS. * De Long $p<0.05$.



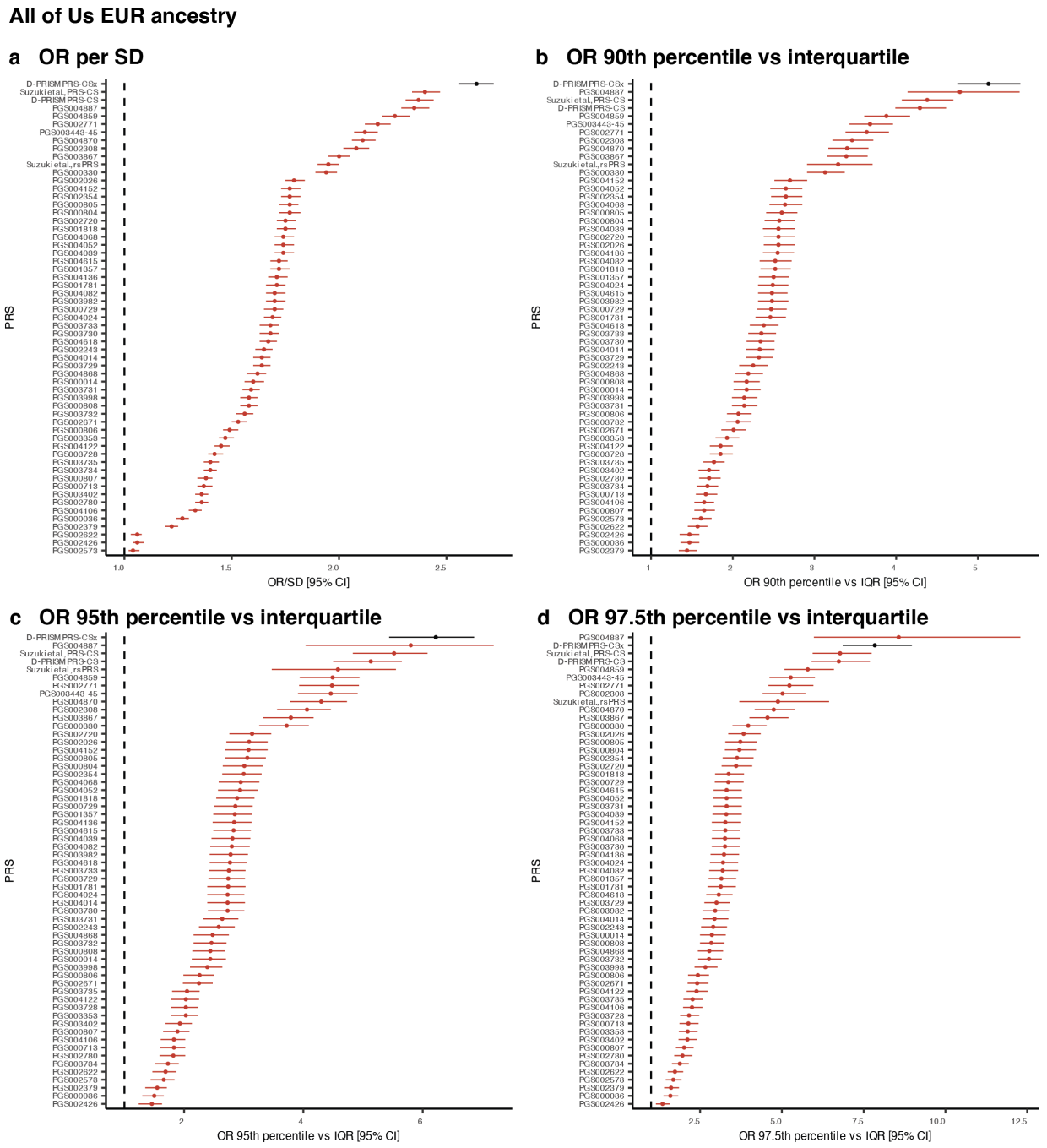

**Supplementary Fig.4 | Performance of D-PRISM multi-ancestry PRS-CSx compared to the published T2D PRSs from the PGS Catalog and others in individuals of AFR ancestry from the All of Us validation cohort. a,** Odds ratio per standard deviation (OR per SD) of the PRS distribution, **b,** OR comparing the 90$^{th}$ percentile of the PRS relative to the interquartile range, **c,** OR comparing the 95$^{th}$ percentile of the PRS relative to the interquartile range, **d,** OR comparing the 97.5$^{th}$ percentile of the PRS relative to the interquartile range.



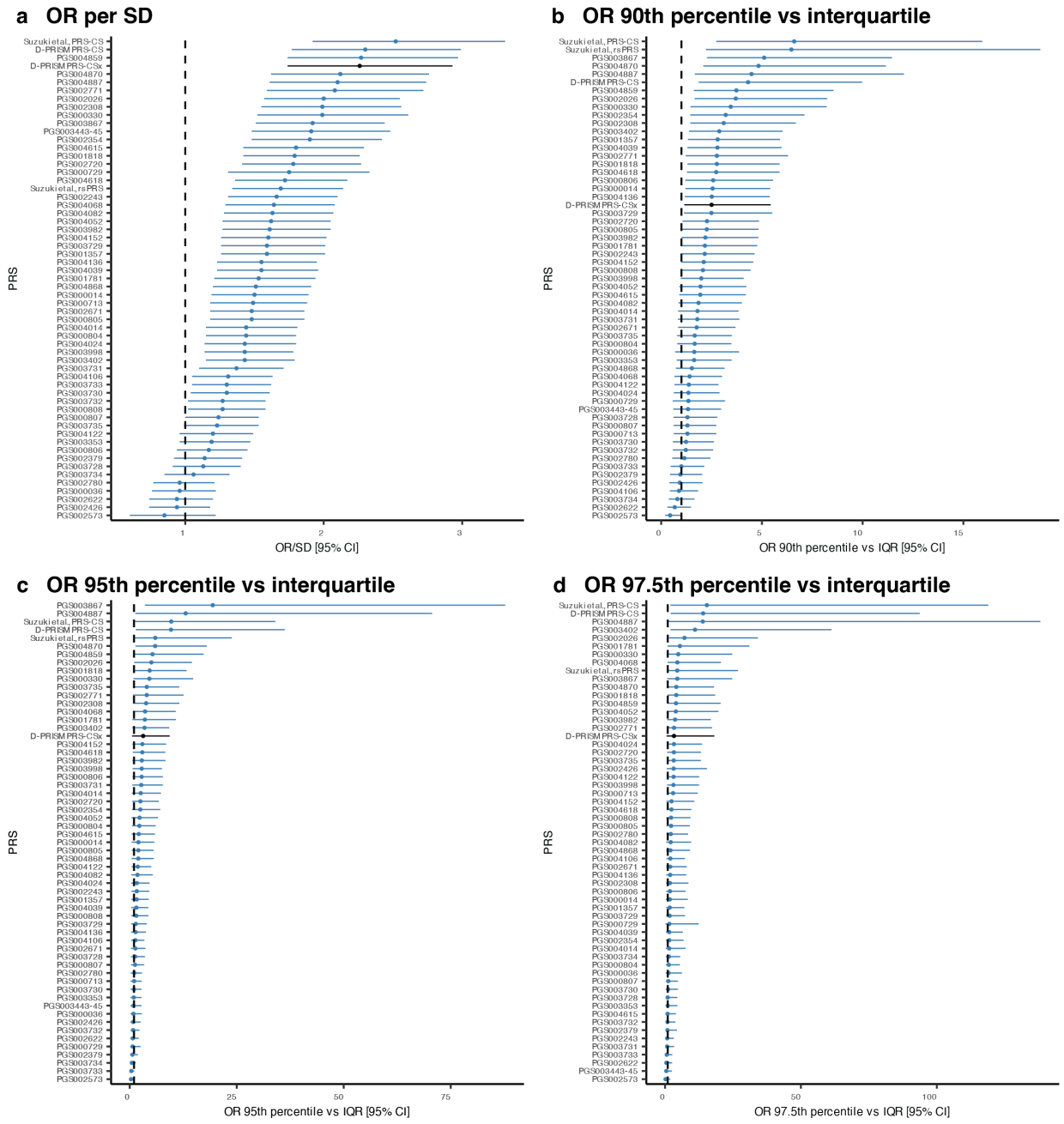

**Supplementary Fig.5 | Performance of D-PRISM multi-ancestry PRS-CSx compared to the published T2D PRSs from the PGS Catalog and others in individuals of AMR ancestry from the All of Us validation cohort. a,** Odds ratio per standard deviation (OR per SD) of the PRS distribution, **b,** OR comparing the 90$^{th}$ percentile of the PRS relative to the interquartile range, **c,** OR comparing the 95$^{th}$ percentile of the PRS relative to the interquartile range, **d,** OR comparing the 97.5$^{th}$ percentile of the PRS relative to the interquartile range.




**Supplementary Fig.6 | Performance of D-PRISM multi-ancestry PRS-CSx compared to the published T2D PRSs from the PGS Catalog and others in individuals of EAS ancestry from the All of Us validation cohort. a,** Odds ratio per standard deviation (OR per SD) of the PRS distribution, **b,** OR comparing the 90$^{th}$ percentile of the PRS relative to the interquartile range, **c,** OR comparing the 95$^{th}$ percentile of the PRS relative to the interquartile range, **d,** OR comparing the 97.5$^{th}$ percentile of the PRS relative to the interquartile range.




**Supplementary Fig.7 | Performance of D-PRISM multi-ancestry PRS-CSx compared to the published T2D PRSs from the PGS Catalog and others in individuals of EUR ancestry from the All of Us validation cohort. a,** Odds ratio per standard deviation (OR per SD) of the PRS distribution, **b,** OR comparing the 90$^{th}$ percentile of the PRS relative to the interquartile range, **c,** OR comparing the 95$^{th}$ percentile of the PRS relative to the interquartile range, **d,** OR comparing the 97.5$^{th}$ percentile of the PRS relative to the interquartile range.




**Supplementary Fig.8 | Performance of D-PRISM multi-ancestry PRS-CSx compared to the published T2D PRSs from the PGS Catalog and others in individuals of SAS ancestry from the All of Us validation cohort. a,** Odds ratio per standard deviation (OR per SD) of the PRS distribution, **b,** OR comparing the 90$^{th}$ percentile of the PRS relative to the interquartile range, **c,** OR comparing the 95$^{th}$ percentile of the PRS relative to the interquartile range, **d,** OR comparing the 97.5$^{th}$ percentile of the PRS relative to the interquartile range.

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