and interpret the GWAS results [14]. Regional plot around genome-wide locus were visualized using LocusZoom (http://csg.sph.umich.edu/locuszoom/).

Data are expressed as percentage (*n*), mean and standard deviation, or median (interquartile range) as appropriate. Differences between groups were tested with chi-squared test, Student's *t*-test, or Mann-Whitney test, as appropriate.

Within the cohort, we performed logistic regression to examine the association of genetic markers (e.g., an individual SNP under an additive genetic model or the PRS) with the risks of GDM and AGT after pregnancy, with the adjustments for PCs, age and/or BMI. The results obtained from individual cohorts were combined through meta-analysis using an inverse-variance weighted approach under a fixed-effects model. Heterogeneity of effect across studies was assessed using Cochran's *Q* test. To address potential population stratification and relatedness among individuals, we adjusted for PCs in all association tests, and applied genomic control correction during the meta-analysis analysis. Associations of identified variants with glycemic and metabolic traits measured during pregnancy were tested by linear regression, adjusting for PCs, age, and/or BMI. Odds ratios with their 95% confidence intervals (CIs), or β±standard error were presented in these analyses. *P* values <0.05 and $<5.0\times10^{-8}$ were considered significant and genome-wide significant, respectively. In the candidate gene analysis, we adjusted for multiple testings using Bonferroni correction. Individuals with missing data points for any variables included in the logistic or linear regression model were removed from the analysis.

The area under the receiver operating characteristic curve (AUROC) and continuous net reclassification improvement (NRI) index were used to evaluate the incremental predictive value of PRS in GDM and AGT after pregnancy, over the clinical risk factors and PCs. We calculated the AUC and NRI index based on the predicted risk obtained from logistic regression, using respectively the "concordance.index" and "nricens" functions in R package. Bootstrapping with 10,000 iterations were used to estimate the 95% CI for the NRI index. We compared two correlated AUCs using the paired *t*-test implemented by the "cindex.comp" function in R package.

SUPPLEMENTARY RESULTS

Sensitivity analysis

In the HAPO-HK Study which included data on comprehensive clinical assessment during pregnancy, the associations between the four identified variants and GDM risk persisted after multivariate adjustment for BMI, GWG, blood pressure, smoking status, education year, parity, family history of diabetes and hypertension (Supplementary Table 14).

Associations for glycemic and metabolic traits during pregnancy

In the linear regression analysis adjusted for PCs and age, we observed several significant associations in the HAPO-HK Study: (1) the A-allele of T-box brain transcription factor 1 (*TBR1*)-solute carrier family 4 member 10 (*SLC4A10*) rs117781972 was associated with elevated levels of 1-hour glucose, 2-hour glucose and AUC_{glu} at 0 to 120 minutes ($1.7\times10^{-4}<P<0.0495$); (2) the C-allele of *CDKAL1* rs7754840 demonstrated an association with increased levels of 1-hour glucose and AUC_{glu} at 0 to 120 minutes ($0.0245<P<0.0249$); (3) there was a notable elevation in levels of 1-hour glucose, 2-hour glucose, AUC_{glu} at 0 to 120 minutes, fasting C-peptide and HOMA2 of insulin resistance (HOMA-IR) index per copy of the C-allele of *INS-IGF2*-potassium voltage-gated channel subfamily Q member 1 (*KCNQ1*) rs2237897 ($1.2\times10^{-4}<P<0.0109$); and (4) the C-allele carriers of melatonin receptor 1B (*MTNR1B*) rs7945617 had higher levels of fasting glucose, 1-hour glucose, 2-hour glucose, and AUC_{glu} at 0 to 120 minutes, as well as a reduced of HOMA2 of β-cell function (HOMA2-β) index ($1.1\times10^{-4}<P<0.0119$) (Supplementary Table 13). Adjustment for BMI did not further change these associations (Supplementary Table 13).

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