SUPPLEMENTAL MATERIAL

Supplemental methods

Discovery

The discovery meta-analysis contained 24 studies from CARDIoGRAM*plus*C4D, ENGAGE and SUMMIT consortia of European descent, except for PROMIS, which included individuals of South Asian descent for which full summary statistics were available (Supplementary Table 1). Cases were selected for inclusion following the standard criteria for CAD and myocardial infarction used in the CARDIoGRAM*plus*C4D consortium.[1] There was no sample overlap amongst the studies included from different consortia.

The discovery included both cross-sectional studies and longitudinal studies (analysed as cross-sectional studies). We accounted for the contemporaneous diagnoses of T2D and CAD in longitudinal studies by including CAD cases that had a diagnosis of T2D prior and up to 5 years after the CAD event.

To identify loci that were specific to CAD in the context of diabetes, and to identify loci that interacted with T2D to modify the risk of CAD, analyses were stratified by T2D status, which included 24,259 subjects with T2D (10,014 CAD cases) and 42,384 subjects without diabetes (17,694 CAD cases). Studies provided summary statistics for variants typed on the Cardio Metabochip array or provided GWAS data imputed to either HapMap2 or 1000 Genomes phase 1 reference panels.[2]

Replication

Replication was sought for loci that achieved a discovery p value$<1\times10^{-4}$ for association with CAD in at least one of the following analyses: all individuals combined regardless of T2D status; subjects with T2D only; subjects without diabetes; or the interaction analysis. Replication was conducted in 11,537 subjects with T2D (3,706 CAD cases) and 106,250 subjects without diabetes (12,988 CAD cases) from four studies of European descent (Supplementary Table 2). The samples used in the replication analyses were independent of those used in the discovery analysis.

Joint analysis and combination of evidence

We had access to full summary statistics for the discovery analysis and requested summary statistics for variants selected for replication from replication cohorts. Thus, we performed a joint analysis between the estimates for individual variants from the discovery analyses and summary statistics for a subset of variants selected for replication. Analyses were performed in each study to test the following comparisons: CAD in subjects with T2D and CAD in subjects without diabetes. Association was tested with CAD status in a regression model, adjusted for age, sex and study specific covariates such as principal components to account for population structure, where applicable. Age was defined as age of event for CAD cases and age at sampling for controls.

Genotype characteristics for the discovery cohort are given in Supplementary Table 3. We excluded variants: minor allele frequency (MAF)<1%; 2×N cases ×MAF<10; Hardy-Weinberg equilibrium test *p* value (p_{hwe}) $<5\times10^{-7}$ and MAF>5% or $p_{hwe}<1\times10^{-4}$ and MAF<5% for directly typed variants and imputation information score <0.4 (IMPUTE2)/imputation information score <0.3 (MaCH) for imputed variants or a call rate <95% for directly typed variants.

We used the additive model to generate association summary statistics and combined these statistics in a fixed-effect inverse variance-weighted meta-analysis using GWAMA v2.1.[3] We used a fixed effects model to estimate the allelic effects in individual strata, under the assumptions that any differences in allelic effect between strata were due to type 2 diabetes background. This method did not account for between study variation in allelic effects. To test for heterogeneous allelic effects by T2D status, we used the method outlined by Magi et al., 2010.[3] We double genomic control (GC) corrected association summary statistics both at the study level and in the overall discovery meta-analysis. Variants were excluded from the discovery meta-analysis if the effective sample size < 4000.

We estimated the interaction effects based on comparing the summary allelic effect on CAD for each variant between subjects with and without T2D. This approach allowed us to include more samples in the meta-analyses as studies that did not contain both subjects with and without T2D could be

included in the stratified analyses but would have been excluded from a meta-analysis of the interaction term. By adopting this approach, we were able to increase the sample size but were unable to account for between study variance in allelic effects.

Signal declaration criteria

We selected loci for replication based on a *p* value of association for the lead variant $\leq 1\times10^{-4}$ in the T2D only, non-diabetic only and interaction analyses. The genotype characteristics of the replication cohorts are given in Supplementary Table 4. We estimated the combined effect sizes for variants in a joint analysis based on a fixed-effect inverse variance–weighted meta-analysis using GWAMA v2.1.[3] Novel loci were declared at $p\leq5\times10^{-8}$. For declaring interaction signals, we required that the directions of effect for individual strata were consistent across the discovery and replication analyses and achieved a $p_{interaction}<0.05/175$ (the number of variants selected for replication) in the replication analysis. Suggestive interaction signals were identified as those that showed directional consistency across the discovery and replication analyses, and when combined in joint analysis achieved combined $p_{interaction}$ < discovery $p_{interaction}$.

Power calculations

Stratified and overall analyses

Power calculations were conducted in R statistics using the gap package.[4] For the purpose of the power calculations effective population size was used (4/ (1/Ncases+1/Nctrls) and a disease prevalence of 5%. The calculation also considered allele frequency and effect size. We used an $\alpha\leq5\times10^{-8}$ for novel loci.

Interaction analysis

Statistical interaction was calculated by testing the difference between two estimates of allelic effect on CAD. The allelic effects were estimated in subjects with diabetes and without diabetes separated and were compared using GWAMA v2.1 to calculate a $p_{interaction}$.[3] The power to detect an interaction

depends on how accurately the allelic effect can be estimated in each stratum. We assessed the power to detect an interaction effect of a CAD-risk variant with T2D in three allelic effect scenarios: a) an effect on CAD in subjects with T2D only (i.e. OR is 1 in subjects without diabetes, but varies between 1 and 1.2 in subjects with T2D); b) an effect on CAD in subjects with T2D and without diabetes in the same direction but of differing magnitude (i.e. OR is 1.10 in subjects without diabetes, but varies between 1 and 1.2 in subjects with T2D); and c) an effect on CAD in subjects with T2D and without diabetes but in opposite directions (i.e. OR is 0.90 in subjects without diabetes, but varies between 1 and 1.2 in subjects with T2D). For each scenario, we evaluated a range of risk allele frequencies: 10%, 20% and 50%. Power was calculated for $\alpha \leq 1 \times 10^{-4}$ in discovery (using discovery sample sizes) based on the threshold for replication.

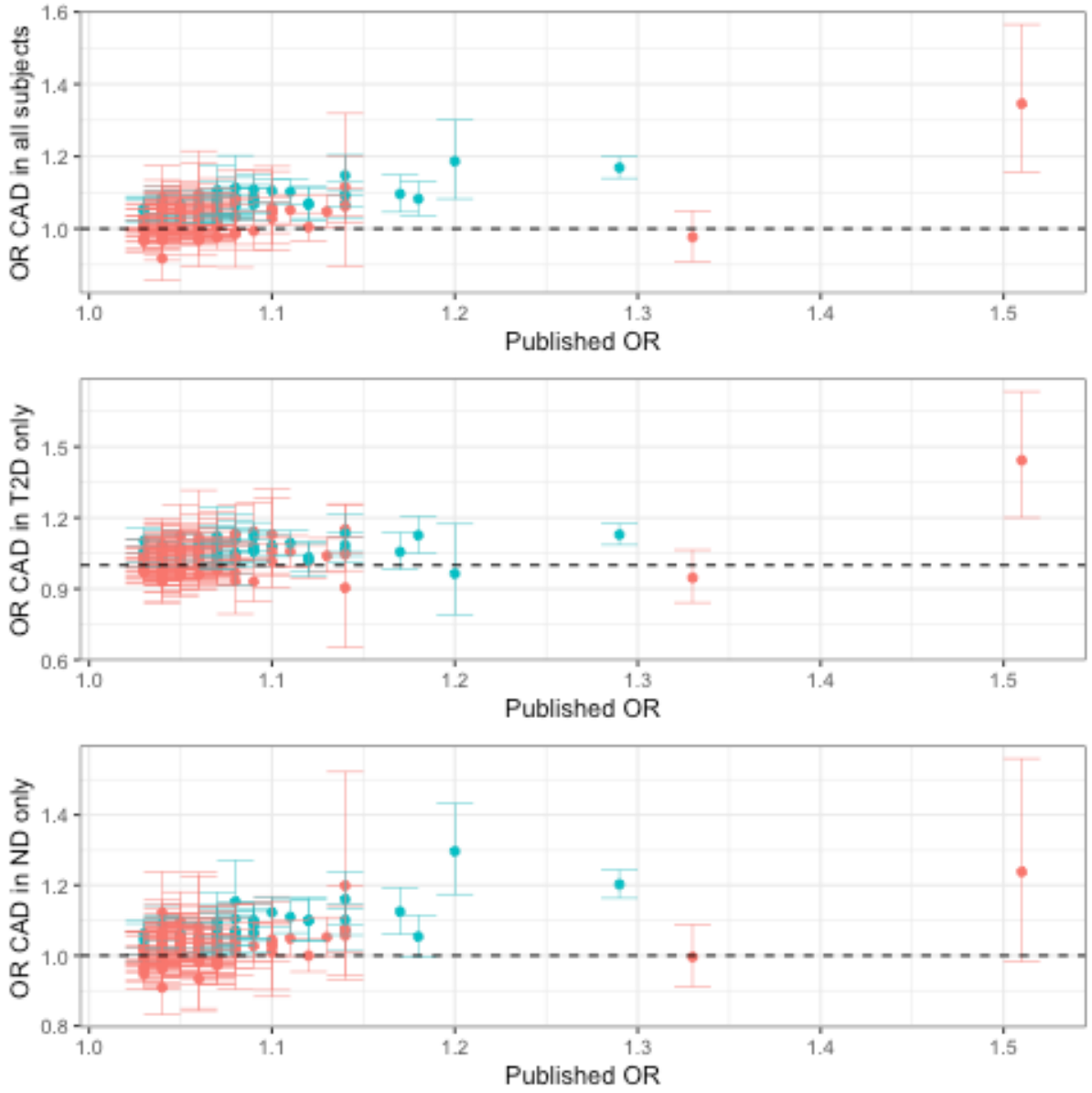
Genetic correlation with related risk factors

We assessed the genetic correlation of CAD by diabetes subgroup with related risk factors using LDHub.[5] Genetic correlation was calculated by taking the slope of the regression of the product of trait 1 z scores on trait 2 z scores on the LD score for a SNP. Z scores were derived from the allelic effects and standard error for that trait. We restricted the analysis to 106 traits available in LDHub that are known risk factors for T2D and CAD.

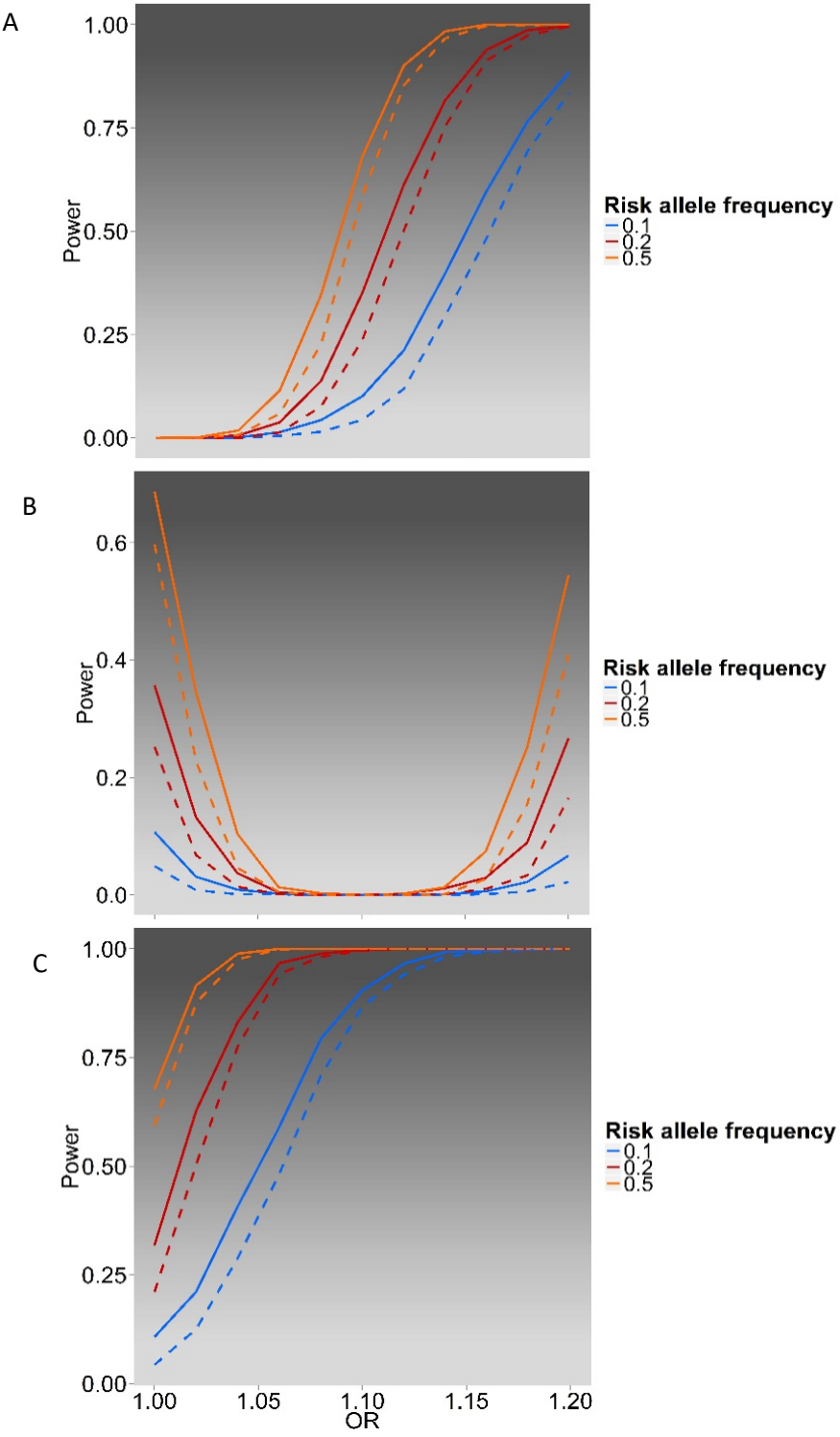
Genetic risk score analysis

SNPs associated with Waist-Hip-ratio (adjusted for body mass index [BMI]), number of SNPs [N_{SNPs}]=53),[6] BMI (untransformed, N_{SNPs}=95 and z-transformed, N_{SNPs}=23),[7,8] systolic blood pressure (SBP) (N_{SNPs}=21),[9,10] LDL-C (N_{SNPs}=143), HDL-C (N_{SNPs}=143), triglycerides (N_{SNPs}=143),[11] T1D, T2D (N_{SNPs}=403),[12] 2-hr glucose (adjusted for BMI, N_{SNPs}=15),[13] fasting glucose (FG, adjusted for BMI, N_{SNPs}=21),[14] glycated haemoglobin (N_{SNPs}=15),[15] fasting insulin (natural log transformed and adjusted for BMI, N_{SNPs}=13),[14] fasting pro-insulin (adjusted for BMI and FG, N_{SNPs}=10),[16] HOMA-B(N_{SNPs}=15), HOMA-IR(N_{SNPs}=15)[17] and insulin resistance (N_{SNPs}=10)[18] at genome-wide significance ($p \leq 5 \times 10^{-8}$) were included in a genetic risk score (GRS) for each trait respectively. To account for the pleiotropic effects

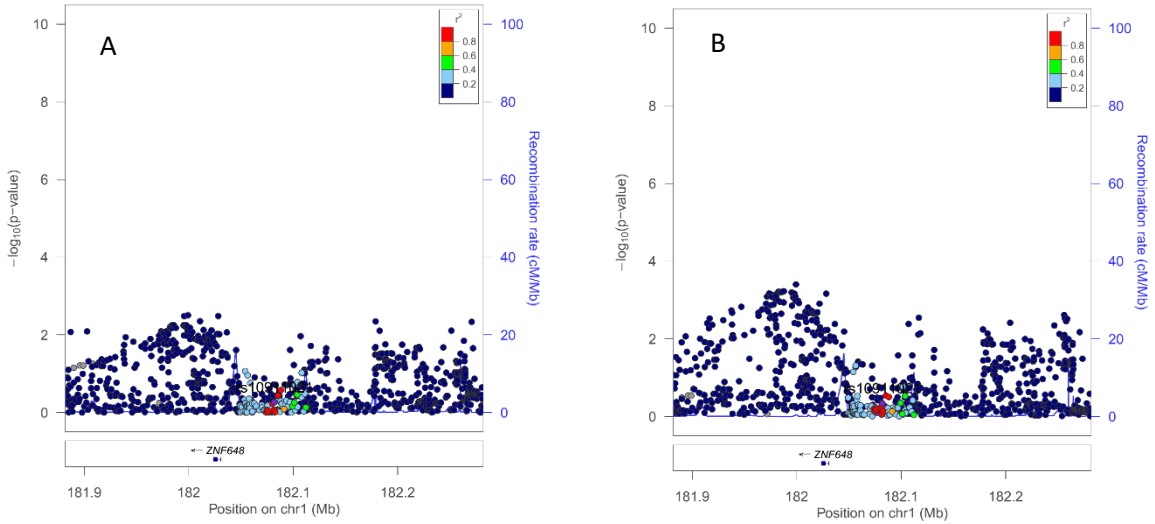
amongst the lipid associated loci a multivariable model was employed from in R using the TwoSampleMR package.[19] For all other GRS the inverse variance weighted method was used to associate each of the GRS with CAD summary statistics.[20]



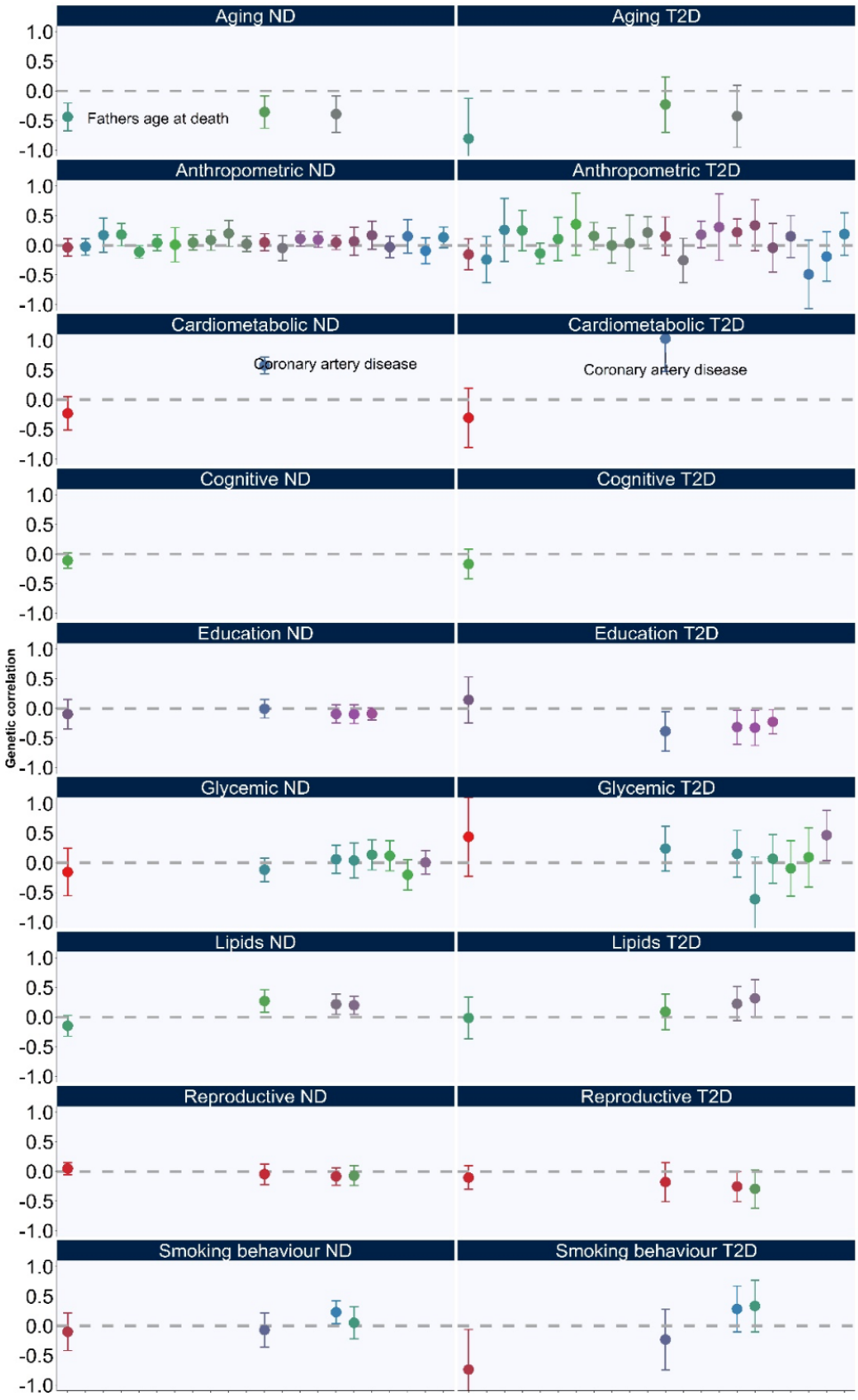
Supplementary Figure 1: Odds ratios (OR) for 160 known coronary artery disease (CAD) loci from this study compared to the published OR in the combined analysis of CAD, CAD in subjects with T2D and for CAD in subjects without diabetes. The blue colour indicates a p<5x10-3 in at least one of the analyses. For these SNPs the odds ratios >/= 1.00 for the published risk allele.



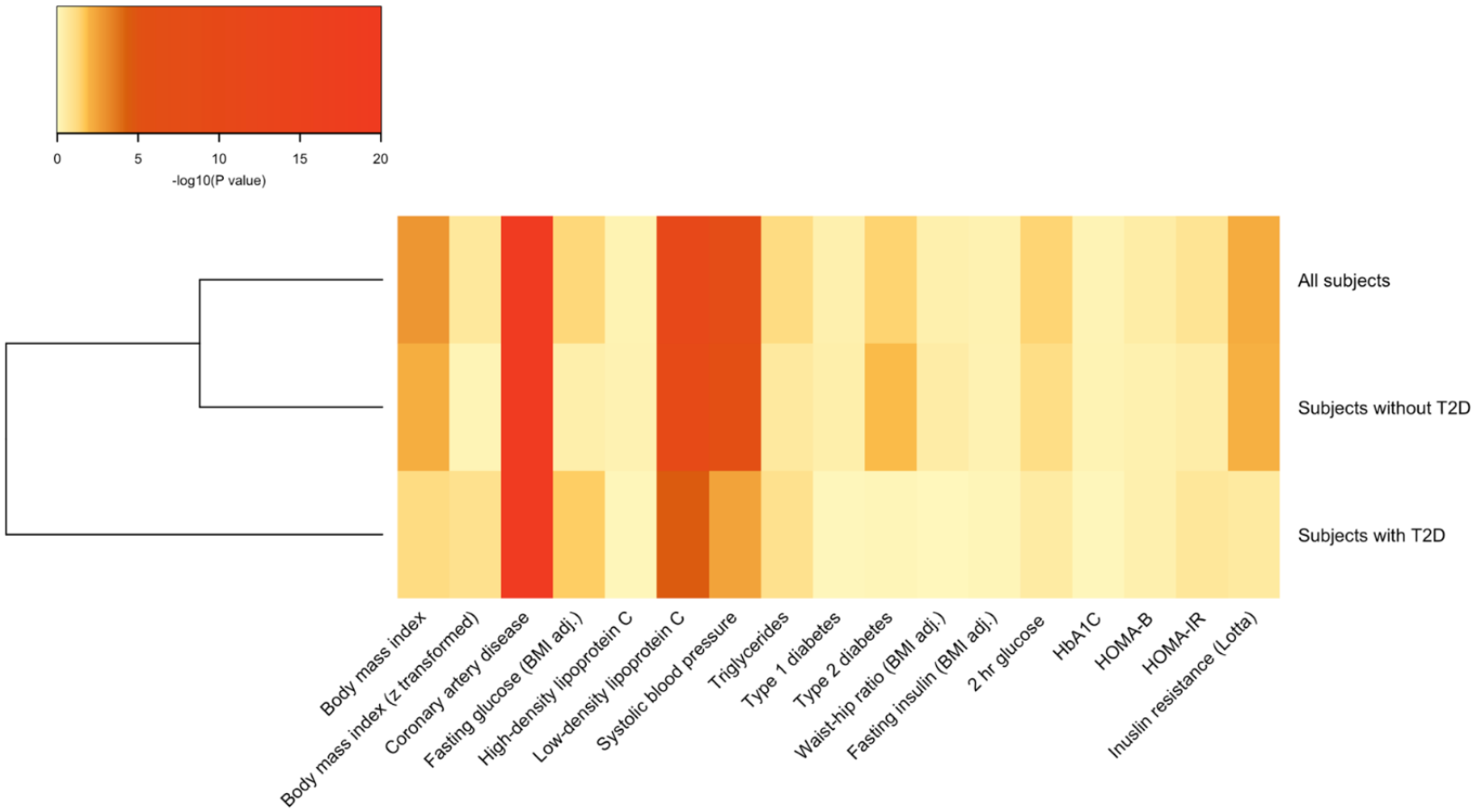
Supplementary Figure 2: Interactions can be broadly classified into three classes: A) effect on coronary artery disease (CAD) is specific to subjects with type 2 diabetes (T2D) (i.e. OR is 1.0 in diabetes free subjects, but varies between 1.0 and 1.2 in subjects with T2D); B) effect on CAD is heterogeneous, but is in the same direction irrespective of diabetes status (i.e. OR is 1.10 in diabetes free subjects, but varies between 1.0 and 1.2 in subjects with T2D); and C) effect on CAD is heterogeneous, and is in the opposite direction in subjects with T2D and diabetes free subjects (i.e. OR is 0.9 in diabetes free subjects, but varies between 1.0 and 1.2 in subjects with T2D). The continuous lines represent the power to detect an interaction $p<1\times10^{-4}$ in discovery and the dashed line a $p<0.05$ in the replication.



Supplementary Figure 3: Locuszoom plots of association statistics in the region of *ZNF648* for a published variant rs10911021 with coronary artery disease (A) and the interaction p value with type 2 diabetes (B). This variant has been previously reported to interact with T2D to modify the risk of coronary artery disease.



Supplementary Figure 4: Genetic correlation of coronary artery disease stratified by type 2 diabetes with known risk factors for CAD. Asterisks indicate a p value < 4.1×10^{-4} (0.05/121) for accuracy in the estimation of genetic correlation. Where the error bars do not cross zero-line p<0.05. Genetic correlations are calculated from all variants rather than those reaching genome-wide significance and give a broader picture of the overall genetic overlap. A different colour point is assigned to each trait.



Supplementary Figure 5: The heat map of genetic risk scores for known coronary artery disease (CAD) risk factors does not show any significant differences between CAD in subjects with and without diabetes. Genetic risk scores (GRS) were for known CAD risk factors were constructed and associated with CAD in all individuals, CAD in subjects without T2D and with CAD in subjects with T2D. This was to determine if there was a different effect of risk factors on CAD by T2D background. Similar colours indicate a similar strength of association between the GRS for the known CAD risk factor and CAD in the different contexts examined.

Supplementary Table 1: The discovery meta-analysis included 24 studies and the sample characteristics of those studies are provided in the accompanying excel file.

Supplementary Table 2: Phenotypic characteristics of the 4 studies that were included in the replication meta-analyses

STUDY	HPFS	NHS	METSIM	DECODE
FULL NAME	The Health Professionals Follow-Up Study	The Nurses' Health Study	The Metabolic Syndrome In Men Study	deCODE Study
REFERENCE	PMID: 23982368	PMID: 23982368	PMID: 19223598	PMID: 27192541
ETHNICITY	Caucasian	Caucasian	European	European
REGION OF RECRUITMENT	The U.S.	The U.S.	Finland	Iceland
PHENOTYPE	CHD	CHD	CAD	CAD
PHENOTYPE DEFINITION	Nonfatal CHD was confirmed using the criteria of the World Health Organization, specifically, on the basis of symptoms and either electrocardiographic changes or elevated cardiac enzyme concentrations. Fatal CHD was defined as fatal myocardial infarction if this was confirmed by hospital records or autopsy, or if CHD was listed as the cause of death on the certificate and this was the underlying and only plausible cause, and evidence of previous CHD was available.	Nonfatal CHD was confirmed using the criteria of the World Health Organization, specifically, on the basis of symptoms and either electrocardiographic changes or elevated cardiac enzyme concentrations. Fatal CHD was defined as fatal myocardial infarction if this was confirmed by hospital records or autopsy, or if CHD was listed as the cause of death on the certificate and this was the underlying and only plausible cause, and evidence of previous CHD was available.	Hopsital admission codes and death records for: MI/Unstable Angina/Corrective procedures (PTCA & CABG)	Coronary artery disease (CAD) was defined as a) individuals in the MONICA registry who suffered myocardial infarction (MI) before the age of 75 in Iceland between 1981 and 2002 and satisfied the MONICA criteria1, b) subjects with CAD discharge diagnoses (ICD 9 codes 410.*, 411.*, 412.*, 414.* or ICD 10 codes I20.0, I21.*, I22.*. I23.*, I24.*, I25.*) from LUH, c) subjects diagnosed with significant angiographic CAD (see below) identified from a nationwide clinical registry of coronary

STUDY	HPFS	NHS	METSIM	DECODE
				angiography and percutaneous coronary interventions at LUH between the years 1987 and 2012, d) subjects undergoing coronary artery bypass grafting (CABG) procedures at LUH between the years 2002 and 2011 and e) cause of death or contributing cause of death listed as MI or CAD (ICD 9 or 10 codes) on death registries between the years 1996 and 2009. Coronary angiograms in the nationwide registry were evaluated by an interventional cardiologist. Patients were considered to have significant angiographic CAD if one or more of the three major epicardial coronary vessels or the left main coronary artery was found to have at least 50% stenosis by visual estimation.

STUDY	HPFS	NHS	METSIM	DECODE
CONTROL DEFINITION	CHD controls were selected randomly and matched in a 1:2 ratio on age, smoking, and month of blood return, among participants who were free of CVD and T2D when CHD was diagnosed in the case.	CHD controls were selected randomly and matched in a 1:2 ratio on age, smoking, and month of blood return, among participants who were free of CVD and T2D when CHD was diagnosed in the case.	Controls free of CAD	Population controls without known history of CAD.
MEAN AGE (SD) YEARS [CASES/CONTROLS] - AGE AT CATH			62.12 (6.40) / 57.07 (7.00)	66.9 (12.9) / 57.7 (17.6)
N CAD/MI [CASES/CONTROLS]	750/1403	644/1689	1173/8999	14156 / 89340
N DIABETIC CAD/MI [CASES/CONTROLS]	385/646	314/918	391/1579	2619 / 4712
N NON-DIABETIC CAD/MI CASES [CASES/CONTROLS]	365/756	330/771	782/7420	11537 / 84628
% EVER SMOKER CAD/MI [CASES/CONTROLS]			17.0%/18.2 %	54.3% / 38.8%
BMI DIABETIC CAD/MI [CASES/CONTROLS]			29.78 (4.64) / 29.90 (5.05)	29.3 (5.3) / 30.3 (6.1)
BMI NON-DIABETIC CAD/MI [CASES/CONTROLS]			27.01 (3.83) / 26.63 (3.71)	26.5 (4.4) / 26.5 (5.0)

Supplementary Table 3: The phenotypic characteristics of studies included in the discovery meta-analysis can be found in the accompanying excel file.

Supplementary Table 4: Genotypic characteristics of the four studies that were included in the replication analyses.

STUDY NAME	HPFS	NHS	METSIM	DECODE
GENOTYPING CENTRE	**The Broad Center for Genotyping and Analysis**	**The Broad Center for Genotyping and Analysis**	**NHGRI**	**deCODE**
GENOTYPING ARRAY	**Affymetrix Genome-Wide Human 6.0 array**	**Affymetrix Genome-Wide Human 6.0 array**	**HumanOmniExpress-12v1 or HumanExome-12v1_A**	**Illumina HumanHap300, HumanCNV370, HumanHap610, HumanHap1M, HumanHap660, Omni-1, Omni 2.5 or Omni Express bead chips**
CALLING ALGORITHM	**the Birdseed calling algorithm**	**the Birdseed calling algorithm**	**GenomeStudio version 2011.1,Genotyping Module version 1.9.4, GenTrain version 1.0**	**GenomeStudio**

STUDY NAME	HPFS	NHS	METSIM	DECODE
PRE-IMPUTATION QC - EXCLUSION CRITERIA	Genotypic data first passed Broad's initial QC which included SNP fingerprints for sample tracking and early detection of sample misidentification, missing call rates of ≥ 5%, the use of a HapMap control to check genotype quality independent of study samples and tracking of reagent and instrumental performance	Genotypic data first passed Broad's initial QC which included SNP fingerprints for sample tracking and early detection of sample misidentification, missing call rates of ≥ 5%, the use of a HapMap control to check genotype quality independent of study samples and tracking of reagent and instrumental performance		Chip SNPs were excluded if they had (i) yield less than 95%, (ii) minor allele frequency (MAF) less than 1% in the population or (iii) significant deviation from Hardy-Weinberg equilibrium (P < 0.001), (iv) if they produced an excessive inheritance error rate (over 0.001), (v) if there was substantial difference in allele frequency between chip types (from just a single chip if that resolved all differences, but from all chips otherwise). All samples with a call rate below 97% were excluded from the

STUDY NAME	HPFS	NHS	METSIM	DECODE
				analysis.Individuals not of Icelandic origin were excluded.
SAMPLE CALL RATE	>98%	>98%	99%	>97%
SNP CALL RATE	>99%	>99%	>95%	>95%
HWE			>0.00029	P > 0.001
IMPUTATION SOFTWARE	MACH	MACH		deCode's imputation pipeline
REFERENCE PANEL	NCBI build 37	NCBI build 37		2636 WGS Icelanders
ANALYSIS SOFTWARE	Plink	Plink	Plink	R
ANALYSIS MODEL	Logistic regression	Logistic regression	Logistic regression	Logistic regression
TOTAL SNPS INCLUDED IN ANALYSIS DIABETIC ONLY [GENOTYPED/IMPUTED]	456	456	290	646 / 0
TOTAL SNPS INCLUDED IN ANALYSIS NON-DIABETIC ONLY [GENOTYPED/IMPUTED]	548	548	330	646 / 0

Supplementary Table 5**:** Results from the T2D stratified analysis for SNPs in known coronary artery disease loci (N=160) in the combined, T2D only and non-diabetic analyses. This table shows the odds ratios from each of the analyses performed and reports the published odds ratio.

The table is available in the supplementary excel file

Supplementary Table 6: Pairwise genetic correlation between 106 traits and coronary artery disease (CAD) stratified by type 2 diabetes (T2D) status using data from LDScore hub. Genetic correlations indicate the overall genetic overlap between traits. Here we wanted to understand the genetic overlap of known CAD risk factors with CAD in the context of T2D.

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Aging	Fathers age at death	27015805	-0.81	0.35	0.02
In subjects without diabetes	Aging	Fathers age at death	27015805	-0.44	0.12	2.0×10^{-4}
In subjects with diabetes	Aging	Mothers age at death	27015805	-0.23	0.24	0.34
In subjects without diabetes	Aging	Mothers age at death	27015805	-0.36	0.14	0.01
In subjects with diabetes	Aging	Parents age at death	27015805	-0.43	0.27	0.11
In subjects without diabetes	Aging	Parents age at death	27015805	-0.39	0.16	0.01
In subjects with diabetes	Anthropometric	Birth weight	27680694	-0.15	0.13	0.25
In subjects without diabetes	Anthropometric	Birth weight	27680694	-0.04	0.08	0.64
In subjects with diabetes	Anthropometric	Body fat	26833246	0.15	0.17	0.36
In subjects without diabetes	Anthropometric	Body fat	26833246	0.05	0.07	0.50
In subjects with diabetes	Anthropometric	Body mass index	20935630	0.22	0.12	0.06
In subjects without diabetes	Anthropometric	Body mass index	20935630	0.05	0.06	0.44

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Anthropometric	Child birth length	25281659	0.34	0.22	0.13
In subjects without diabetes	Anthropometric	Child birth length	25281659	0.07	0.12	0.57
In subjects with diabetes	Anthropometric	Child birth weight	23202124	-0.04	0.21	0.84
In subjects without diabetes	Anthropometric	Child birth weight	23202124	0.17	0.12	0.16
In subjects with diabetes	Anthropometric	Childhood obesity	22484627	0.15	0.18	0.41
In subjects without diabetes	Anthropometric	Childhood obesity	22484627	-0.03	0.09	0.74
In subjects with diabetes	Anthropometric	Difference in height between adolescence and adulthood; age 14	23449627	-0.49	0.30	0.10
In subjects without diabetes	Anthropometric	Difference in height between adolescence and adulthood; age 14	23449627	0.15	0.15	0.30
In subjects with diabetes	Anthropometric	Difference in height between childhood and adulthood; age 8	23449627	-0.19	0.21	0.37
In subjects without diabetes	Anthropometric	Difference in height between childhood and adulthood; age 8	23449627	-0.09	0.11	0.40
In subjects with diabetes	Anthropometric	Extreme bmi	23563607	0.19	0.18	0.31
In subjects without diabetes	Anthropometric	Extreme bmi	23563607	0.13	0.09	0.13
In subjects with diabetes	Anthropometric	Extreme height	23563607	-0.24	0.20	0.22
In subjects without diabetes	Anthropometric	Extreme height	23563607	-0.03	0.07	0.73

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Anthropometric	Extreme waist-to-hip ratio	23563607	0.26	0.27	0.34
In subjects without diabetes	Anthropometric	Extreme waist-to-hip ratio	23563607	0.17	0.15	0.24
In subjects with diabetes	Anthropometric	Height 2010	20881960	-0.14	0.09	0.13
In subjects without diabetes	Anthropometric	Height 2010	20881960	-0.11	0.05	0.04
In subjects with diabetes	Anthropometric	Height; Females at age 10 and males at age 12	23449627	0.25	0.17	0.15
In subjects without diabetes	Anthropometric	Height; Females at age 10 and males at age 12	23449627	0.18	0.10	0.06
In subjects with diabetes	Anthropometric	Hip circumference	25673412	0.11	0.19	0.57
In subjects without diabetes	Anthropometric	Hip circumference	25673412	0.04	0.07	0.52
In subjects with diabetes	Anthropometric	Infant head circumference	22504419	0.35	0.27	0.18
In subjects without diabetes	Anthropometric	Infant head circumference	22504419	0.01	0.15	0.95
In subjects with diabetes	Anthropometric	Obesity class 1	23563607	0.16	0.12	0.18
In subjects without diabetes	Anthropometric	Obesity class 1	23563607	0.05	0.07	0.48
In subjects with diabetes	Anthropometric	Obesity class 2	23563607	0.00	0.15	0.99
In subjects without diabetes	Anthropometric	Obesity class 2	23563607	0.09	0.09	0.32

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Anthropometric	Obesity class 3	23563607	0.04	0.24	0.88
In subjects without diabetes	Anthropometric	Obesity class 3	23563607	0.20	0.11	0.08
In subjects with diabetes	Anthropometric	Overweight	23563607	0.21	0.14	0.12
In subjects without diabetes	Anthropometric	Overweight	23563607	0.02	0.07	0.73
In subjects with diabetes	Anthropometric	Sitting height ratio	25865494	-0.25	0.19	0.18
In subjects without diabetes	Anthropometric	Sitting height ratio	25865494	-0.05	0.11	0.67
In subjects with diabetes	Anthropometric	Waist circumference	25673412	0.31	0.29	0.28
In subjects without diabetes	Anthropometric	Waist circumference	25673412	0.09	0.07	0.15
In subjects with diabetes	Anthropometric	Waist-to-hip ratio	25673412	0.18	0.11	0.12
In subjects without diabetes	Anthropometric	Waist-to-hip ratio	25673412	0.11	0.06	0.09
In subjects with diabetes	Cardiometabolic	Adiponectin	22479202	-0.31	0.26	0.23
In subjects without diabetes	Cardiometabolic	Adiponectin	22479202	-0.23	0.14	0.11
In subjects with diabetes	Cardiometabolic	Coronary artery disease	26343387	1.03	0.28	3.0×10^{-4}
In subjects without diabetes	Cardiometabolic	Coronary artery disease	26343387	0.58	0.07	1.1×10^{-16}

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Cognitive	Intelligence	28530673	-0.17	0.13	0.19
In subjects without diabetes	Cognitive	Intelligence	28530673	-0.11	0.06	0.09
In subjects with diabetes	Education	Childhood IQ	23358156	0.14	0.20	0.47
In subjects without diabetes	Education	Childhood IQ	23358156	-0.10	0.13	0.45
In subjects with diabetes	Education	College completion	23722424	-0.39	0.17	0.02
In subjects without diabetes	Education	College completion	23722424	-0.01	0.08	0.92
In subjects with diabetes	Education	Years of schooling (proxy cognitive performance)	25201988	-0.32	0.15	0.03
In subjects without diabetes	Education	Years of schooling (proxy cognitive performance)	25201988	-0.09	0.08	0.22
In subjects with diabetes	Education	Years of schooling 2013	23722424	-0.33	0.15	0.03
In subjects without diabetes	Education	Years of schooling 2013	23722424	-0.10	0.08	0.21
In subjects with diabetes	Education	Years of schooling 2016	27225129	-0.23	0.10	0.03
In subjects without diabetes	Education	Years of schooling 2016	27225129	-0.09	0.05	0.09
In subjects with diabetes	Glycemic	2hr glucose adjusted for BMI	20081857	0.44	0.34	0.20
In subjects without diabetes	Glycemic	2hr glucose adjusted for BMI	20081857	-0.16	0.20	0.44

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Glycemic	Fasting glucose main effect	22581228	0.24	0.19	0.22
In subjects without diabetes	Glycemic	Fasting glucose main effect	22581228	-0.12	0.10	0.25
In subjects with diabetes	Glycemic	Fasting insulin main effect	22581228	0.15	0.20	0.46
In subjects without diabetes	Glycemic	Fasting insulin main effect	22581228	0.06	0.12	0.62
In subjects with diabetes	Glycemic	Fasting proinsulin	20081858	-0.62	0.36	0.09
In subjects without diabetes	Glycemic	Fasting proinsulin	20081858	0.04	0.15	0.79
In subjects with diabetes	Glycemic	HbA1C	20858683	0.07	0.21	0.75
In subjects without diabetes	Glycemic	HbA1C	20858683	0.14	0.13	0.30
In subjects with diabetes	Glycemic	HOMA-B	20081858	-0.09	0.24	0.69
In subjects without diabetes	Glycemic	HOMA-B	20081858	0.12	0.13	0.36
In subjects with diabetes	Glycemic	HOMA-IR	20081858	0.09	0.26	0.72
In subjects without diabetes	Glycemic	HOMA-IR	20081858	-0.20	0.13	0.12
In subjects with diabetes	Glycemic	Type 2 Diabetes	22885922	0.46	0.22	0.03
In subjects without diabetes	Glycemic	Type 2 Diabetes	22885922	0.01	0.10	0.96

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Lipids	HDL cholesterol	20686565	-0.01	0.18	0.94
In subjects without diabetes	Lipids	HDL cholesterol	20686565	-0.15	0.09	0.10
In subjects with diabetes	Lipids	LDL cholesterol	20686565	0.09	0.15	0.56
In subjects without diabetes	Lipids	LDL cholesterol	20686565	0.27	0.10	0.01
In subjects with diabetes	Lipids	Total Cholesterol	20686565	0.23	0.15	0.12
In subjects without diabetes	Lipids	Total Cholesterol	20686565	0.22	0.09	0.01
In subjects with diabetes	Lipids	Triglycerides	20686565	0.32	0.16	0.05
In subjects without diabetes	Lipids	Triglycerides	20686565	0.20	0.08	0.01
In subjects with diabetes	Reproductive	Age at Menarche	25231870	-0.10	0.10	0.31
In subjects without diabetes	Reproductive	Age at Menarche	25231870	0.05	0.05	0.35
In subjects with diabetes	Reproductive	Age at Menopause	26414677	-0.18	0.17	0.29
In subjects without diabetes	Reproductive	Age at Menopause	26414677	-0.05	0.09	0.61
In subjects with diabetes	Reproductive	Age of first birth	27798627	-0.25	0.13	0.05
In subjects without diabetes	Reproductive	Age of first birth	27798627	-0.08	0.07	0.27

Coronary Artery Disease	Category	Trait	PMID	rg	se	p
In subjects with diabetes	Reproductive	Number of children ever born	27798627	-0.29	0.16	0.07
In subjects without diabetes	Reproductive	Number of children ever born	27798627	-0.07	0.09	0.40
In subjects with diabetes	Smoking behaviour	Age of smoking initiation	20418890	-0.74	0.35	0.03
In subjects without diabetes	Smoking behaviour	Age of smoking initiation	20418890	-0.10	0.16	0.53
In subjects with diabetes	Smoking behaviour	Cigarettes smoked per day	20418890	-0.23	0.26	0.37
In subjects without diabetes	Smoking behaviour	Cigarettes smoked per day	20418890	-0.07	0.15	0.63
In subjects with diabetes	Smoking behaviour	Ever vs never smoked	20418890	0.28	0.20	0.15
In subjects without diabetes	Smoking behaviour	Ever vs never smoked	20418890	0.23	0.10	0.02
In subjects with diabetes	Smoking behaviour	Former vs Current smoker	20418890	0.34	0.22	0.13
In subjects without diabetes	Smoking behaviour	Former vs Current smoker	20418890	0.05	0.14	0.71

Supplementary Table 7: Coronary artery disease (CAD) has several risk factors that are in part genetically determined. We constructed genetic risk scores for known CAD risk factors and associated them with CAD by type 2 diabetes status.

CAD score	Risk Factor	OR	L95	U95	P
All subjects	Body mass index	1.06	1.02	1.09	1.6×10^{-3}
Subjects with T2D	Body mass index	1.05	1.00	1.10	0.07
Subjects without T2D	Body mass index	1.06	1.02	1.11	7.1×10^{-3}
All subjects	Body mass index (z transformed)	1.08	0.95	1.22	0.23
Subjects with T2D	Body mass index (z transformed)	1.16	0.96	1.41	0.12
Subjects without T2D	Body mass index (z transformed)	1.03	0.88	1.20	0.75
All subjects	Coronary artery disease	2.12	2.01	2.24	6.4×10^{-171}
Subjects with T2D	Coronary artery disease	2.05	1.88	2.23	1.1×10^{-59}
Subjects without T2D	Coronary artery disease	2.18	2.04	2.33	8.5×10^{-117}
All subjects	Fasting glucose (BMI adj.)	1.56	1.00	2.42	0.05
Subjects with T2D	Fasting glucose (BMI adj.)	2.13	1.08	4.21	0.03
Subjects without T2D	Fasting glucose (BMI adj.)	1.23	0.70	2.18	0.47
All subjects	High-density lipoprotein C	0.97	0.86	1.10	0.66
Subjects with T2D	High-density lipoprotein C	0.99	0.84	1.17	0.89
Subjects without T2D	High-density lipoprotein C	0.96	0.83	1.12	0.62
All subjects	Low-density lipoprotein C	1.37	1.25	1.50	1.0×10^{-11}
Subjects with T2D	Low-density lipoprotein C	1.32	1.16	1.50	3.3×10^{-5}
Subjects without T2D	Low-density lipoprotein C	1.43	1.28	1.60	6.6×10^{-10}
All subjects	Systolic blood pressure	1.66	1.39	1.98	1.7×10^{-8}
Subjects with T2D	Systolic blood pressure	1.58	1.16	2.16	3.7×10^{-3}
Subjects without T2D	Systolic blood pressure	1.70	1.37	2.10	9.9×10^{-7}
All subjects	Triglycerides	1.14	0.99	1.30	0.07
Subjects with T2D	Triglycerides	1.16	0.96	1.41	0.12

CAD score	Risk Factor	OR	L95	U95	P
Subjects without T2D	Triglycerides	1.11	0.93	1.33	0.26
All subjects	Type 1 diabetes	1.00	0.99	1.02	0.53
Subjects with T2D	Type 1 diabetes	1.00	0.98	1.02	0.95
Subjects without T2D	Type 1 diabetes	1.01	0.99	1.02	0.46
All subjects	Type 2 diabetes	1.04	1.00	1.08	0.04
Subjects with T2D	Type 2 diabetes	0.99	0.94	1.05	0.85
Subjects without T2D	Type 2 diabetes	1.06	1.01	1.11	0.01
All subjects	Waist-hip ratio (BMI adj.)	0.80	0.41	1.57	0.51
Subjects with T2D	Waist-hip ratio (BMI adj.)	1.01	0.36	2.82	0.99
Subjects without T2D	Waist-hip ratio (BMI adj.)	0.66	0.27	1.60	0.36
All subjects	Fasting insulin (BMI adj.)	1.17	0.64	2.14	0.62
Subjects with T2D	Fasting insulin (BMI adj.)	1.12	0.44	2.84	0.82
Subjects without T2D	Fasting insulin (BMI adj.)	1.20	0.55	2.63	0.65
All subjects	2 hr glucose	0.89	0.80	1.00	0.04
Subjects with T2D	2 hr glucose	0.91	0.76	1.09	0.31
Subjects without T2D	2 hr glucose	0.89	0.77	1.02	0.08
All subjects	HbA1C	1.09	0.69	1.70	0.72
Subjects with T2D	HbA1C	1.02	0.50	2.09	0.95
Subjects without T2D	HbA1C	1.12	0.64	1.98	0.69
All subjects	HOMA-B	1.18	0.82	1.70	0.38
Subjects with T2D	HOMA-B	1.21	0.68	2.15	0.52
Subjects without T2D	HOMA-B	1.15	0.72	1.84	0.55
All subjects	HOMA-IR	0.67	0.39	1.17	0.16
Subjects with T2D	HOMA-IR	0.55	0.22	1.40	0.21
Subjects without T2D	HOMA-IR	0.75	0.38	1.47	0.40
All subjects	Insulin resistance (Lotta)	1.01	1.00	1.01	6.3×10^{-3}
Subjects with T2D	Insulin resistance (Lotta)	1.00	1.00	1.01	0.28

CAD score	Risk Factor	OR	L95	U95	P
Subjects without T2D	Insulin resistance (Lotta)	1.01	1.00	1.01	0.01

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	Heike Zimdahl-Gelling	**Pharmacogenomics, Head of Lab**
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	Carl Whatling	**Bioscientist**
23	**Everson Nogoceke**	**Pre-clinical and clinical aspects of metabolic and vascular disease; PI; WP2 leader**
Roche	**Gonzalo Durán Pacheco**	**Senior Research Statistician**
	Ivan Formentini	**Biomarker & Experimental Medicine Leader**
	Thomas Schindler	**Pre-clinical and clinical and clinical biomarkers**
24	**Piero Tortoli**	**Professor of Electronics**
University of Florence	**Luca Bassi**	**Postdoctoral fellow**
	Enrico Boni	**Postdoctoral fellow**
	Alessandro Dallai	**Postdoctoral fellow**
	Francesco Guidi	**Technician**

	Matteo Lenge	**PhD student**
	Riccardo Matera	**PhD student**
	Alessandro Ramalli	**PhD student**
	Stefano Ricci	**Assist. Prof.**
	Jacopo Viti	**PhD student**
25	**Bernd Jablonka**	**SAD internal IMI coordinator**
Sanofi-aventis	**Dan Crowther**	**Biomarker researcher**
	Johan Gassenhuber	**Biostatistician**
	Sibylle Hess	**Biomarker researcher**
	Thomas Hübschle	**Pharmacologist Diabetes**
	Hans-Paul Juretschke	**Imaging**
	Hartmut Rütten	**Head Translational Medicine**
	Thorsten Sadowski	**Pharmacologist Diabetes**
	Paulus Wohlfart	**Pharmacologist Diabetes**
26	**Julia Brosnan**	**Biochemist, (pre)clinical research CVD, Pfizer US; WP2 leader**
Pfizer	**Valerie Clerin**	**Cardio-renal biologist, WP2**
	Eric Fauman	**Computational biologist**
	Craig Hyde	**Statistician**
	Anders Malarstig	**Human genetics, Pfizer Europé; WP1 leader**
	Nick Pullen	**Renal Disease Research Director**
	Mera Tilley	
	Theresa Tuthill	**Imaging specialist**
	Ciara Vangjeli	**Cardiovascular genetic epidemiologist, Pfizer Europe**
	Daniel Ziemek	**Computational biologist**

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