SUPPLEMENTAL MATERIALS

Supplemental methods:

Studies of coronary artery disease and myocardial infarction:

CARDIoGRAMplusC4D consortium: The details of the CARDIoGRAMplusC4D, 1000 genomes imputed dataset have already been published.[1] In brief, the CARDIoGRAMplusC4D dataset consists of merged data from the classic genome-wide association studies (GWAS) CARDIoGRAM and C4D, combining genotype information on 60,801 case subjects and 123,504 control subjects from 48 studies then imputed using the 1000 Genomes phase 1, version 3 reference panel.[1-4] As described, case subjects were defined by an inclusive coronary artery disease (CAD) diagnosis including myocardial infarction (MI), acute coronary syndrome, chronic stable angina, or coronary stenosis >50%. Association data for each contributing study were individually filtered for MAF > 0.5% and an imputation quality metric. For each study, ancestry-informative or other study-specific covariates were included as necessary which was confirmed on submission by review of the study-specific genomic control lambda. Variants that were retained in at least 60% of the studies were submitted for analysis. Following an inverse variance–weighted, fixed-effects meta-analysis, heterogeneity was assessed by Cochran's Q statistic[5] and the I^2 inconsistency index[6] and variants showing marked heterogeneity were reanalyzed using a random-effects model.[7] Overdispersion in the resulting meta-analysis was adjusted for by a second application of the genomic control procedure. 8.6 million single nucleotide polymorphisms (SNPs) and 836,000 insertions/deletions (indels) were included in the analysis. Association testing was performed by logistic regression on additive, recessive, and dominant models of disease susceptibility. Individual studies were combined using a fixed-effects, inverse-variance weighted meta-analysis.

Myocardial Infarction Genetics (MIGen) and CARDIoGRAM Exome consortia: The MIGen and CARDIoGRAM Exome array consortia consists of merged data from 19 studies totaling 42,335 MI case subjects and 78,240 control subjects of European ancestry.[8] Subjects were

genotyped for 220,231 non-synonymous autosomal variants on the Illumina HumanExome BeadChip v1.0 (Illumina, San Diego, Ca). Quality control filters were applied before and after implementation of a zCall algorithm as described.[9] For variants that passed quality control procedures, individual tests for association with CAD were performed within each study. For variants with a MAF greater than 0% in both cases and controls, logistic regression was run on an additive model with the principal components of ancestry as covariates. Individual studies were combined using an inverse-variance weighted meta-analysis. Variants were functionally annotated as published and than restricted to those with a MAF $\geq$ 0.01%, leaving 54,003 variants with reported association testing. While 8 studies in the MIGen and CARDIoGRAM Exome array consortia dataset overlapped completely or partially with the CARDIoGRAMplusC4D meta-analysis, our focus here differs substantively from that of CARDIoGRAMplusC4D given its specific focus on low-frequency variation.

Exome Sequencing Project and Early-Onset Myocardial Infarction (ESP EOMI) consortium: Details of the National Heart, Lung and Blood Institute's GO exome sequencing project (NHLBI ESP) and the ESP early-onset myocardial infarction (ESP EOMI) study have been published.[10,11] Briefly, the ESP EOMI was conducted using 4,703 EOMI case subjects and 5,090 control subjects. EOMI cases were defined as individuals who had an MI at age $\leq$ 50 years for men and at age $\leq$ 60 years for women. Control subjects were selected from individuals without a history of MI at baseline or whom did not have an MI during follow-up surveillance to a pre-specified age. Initial exome sequencing on subjects from 11 studies was performed at the Broad Institute with sequencing, exome capture, read mapping, variant analysis and quality control as published previously.[11] Follow-up sequencing was subsequently performed on samples from three additional studies. These samples were similarly sequenced at the Broad Institute with processing and quality control as published.[11] To test whether rare mutations contribute to EOMI, burden of rare variant association testing was performed on SNPs and

indels present in *CCR2, CCR5,* and *CX3CR1.* The analysis was performed using the Efficient Mixed-Model Association eXpedited (EMMAX) Combined Multivariate and Collapsing (CMC) test.[12] The analysis was restricted to variants with a MAF < 1% as calculated using all sequenced samples in the study. Variants were analyzed using seven algorithms: LRT score, MutationTaster, PolyPhen-2 HumVar, PolyPhen-2 HumDiv, SIFT, MutationAssessor, and FATHMM.[13-18] To enrich for harmful alleles, different iterations of rare variant testing were performed using (1) non-synonymous variants; (2) disruptive variants (nonsense, slice-site, and indel frameshift variants); and (3) deleterious variants, defined as disruptive variants in combination with missense variants damaging by five, six, or seven of the aforementioned algorithms. Reported P-values were calculated using the EMMAX CMC test.

The Pakistan Risk of Myocardial Infarction Study (PROMIS): PROMIS is a retrospective case-control study of acute, first MIs in 6 centers in urban Pakistan combining data from 9,058 subjects with an acute MI and 8,378 control subjects.[19,20] Cases were defined as subjects presenting within 24 hours of symptom onset with typical ECG changes and a positive troponin-I. Control subjects were drawn from individuals without self-reported cardiovascular disease identified in the same hospitals as index cases. For each participant, information was collected on demographic factors, lifestyle, personal and family history. Non-fasting blood samples were collected from each participant to allow for measurement of serum biomarkers.

Samples were genotyped on the Illumina 660 and Illumina 770 arrays. Genotypes were imputed using the 1000 phase I integrated reference panel (March, 2012) using SHAPEIT and IMPUTE2.[21-23] SNPs were filtered for HWE $<1x10^{-5}$, imputation quality score (INFO) <0.7, and MAF $\leq$ 1%. Individual tests for association were performed with respect to MI adjusting for the first four principal components. The genomic inflation factor was 1.09.

Studies of glucometabolic traits:

Diabetes Genetics Replication and Meta-analysis (DIAGRAM): DIAGRAM contains information on 12,171 case subjects with type II Diabetes Mellitus (DM) and 56,862 control subjects of European descent combined across 12 GWAS. The details of the study have been published.[24] Sample and SNP quality control were undertaken within each study. Each GWAS was imputed using the phase II CEU HapMap reference panel. SNPs with a MAF>1% passing quality control criteria were tested for association with type II DM under an additive model after adjustment for study-specific covariates. Association summary statistics were combined via a fixed-effects, inverse-variance weighted meta-analysis.

The Genetic Investigation of ANthropometric Traits (GIANT) consortium: The GIANT meta-analysis contains genetic information on 123,865 subjects of European ancestry combined from across 46 studies.[25] All samples were genotyped using the Affymetrix (Affymetrix, Santa Clara, Ca) and Illumina (Illumina, San Diego, CA) whole genome genotyping arrays. Polymorphic SNPs were imputed using the HapMap CEU reference panel.

Association analysis with Body Mass Index (BMI): The GWAS on BMI includes genetic information from subjects across all 46 studies.[25] Each study performed single marker association analyses with BMI under an additive genetic model. BMI was adjusted for age, age^2, and principal components as deemed appropriate and than inverse normally transformed. SNPs with poor imputation quality and a minor allele count less than 3 in each sex- and case-specific stratum were excluded. The meta-analysis was performed in METAL using both the inverse variance method and the weighted *z*-score method.

Association analysis with Waist-Hip Ratio (WHR): The GWAS on WHR includes information on a subset of 77,167 subjects from 32 GWAS.[26] For each cohort, age-adjusted residuals were calculated for men and women separately with BMI adjustment then inverse normally

transformed to ensure comparability across studies. SNP associations for WHR adjusted for BMI were computed by linear regression separately for men and women though these were combined to account for relatedness when appropriate. In addition to study-specific quality control measures, SNPs were excluded for low imputation quality and if the MAF times the number of subjects for a SNP in one study was less than 3.[26] A fixed-effects, inverse-variance weighted model was used to pool β estimates. P-values and standard errors for each study were genomic control corrected and a second genomic control correction was applied to meta-analyzed results.

Global Lipids Genetics Consortium (GLGC): The 2010 GLGC meta-analysis includes information on 100,184 individuals of European descent from 46 GWAS of lipids and lipid-related traits.[27] Each study performed genotype imputation with respect to the phase II CEU HapMap reference panel. Residual lipoprotein concentrations were determined after regression adjustment for the covariates age, age^2, and sex. Each genotyped or imputed SNP was tested for association with each trait assuming an additive genetic model. Linear regression was employed for studies of unrelated individuals and linear mixed effects models were used to account for family structure in family-based studies. SNPs with a MAF < 0.01 and poor imputation quality were excluded. Results were combined using a fixed effects meta-analysis in METAL for each of the lipid traits.

Meta-Analysis of glucose and Insulin-related traits consortium (MAGIC): The MAGIC consortium contains information on glycemic traits from non-diabetic individuals of European descent. The results have been published and are freely available online.[28,29] Polymorphic SNPs were imputed using the HapMap CEU reference panel. HgbA1c association results were available for 46,368 non-diabetic adults of European descent from 23 GWAS**.** The fasting insulin and fasting glucose datasets were generated by performing a meta-analysis of up to 21 GWAS

informative for fasting glucose, fasting insulin and indices of β-cell function (HOMA-B) and insulin resistance (HOMA-IR) in 46,186 non-diabetic participants.[29] Trait values for fasting insulin, HOMA-IR, and HOMA-B were naturally log transformed. Datasets were adjusted for age, sex and study-specific covariates and then combined using a fixed-effects, inverse-variance approach.

The Pakistan Risk of Myocardial Infarction Study (PROMIS): The PROMIS resource is described above.[19,20] In addition to MI, association tests were performed for type II DM and lipid levels and summary data extracted for SNPs within 5,000bps of the start and end positions of *CCR2, CCR5,* and *CX3CR1*.

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Gene	Position (hg19)	rs#	Location	Alternate Allele	EAF	Beta	P-value
CX3CR1	3:39300023	rs140374643	Intergenic	A	0.98	-0.02	0.63
CX3CR1	3:39300157	rs17038640	Intergenic	C	0.65	0.004	0.71
CX3CR1	3:39300476	rs75540383	Intergenic	C	0.95	-0.02	0.34
CX3CR1	3:39300686	rs62244210	Intergenic	G	0.95	0.04	0.15
CX3CR1	3:39300845	rs73060520	Intergenic	C	0.91	0.01	0.59
CX3CR1	3:39301123	rs73060524	Intergenic	A	0.76	0.002	0.83
CX3CR1	3:39301287	rs151027349	Intergenic	G	0.98	-0.05	0.17
CX3CR1	3:39302355	rs11711922	Intergenic	A	0.69	-0.01	0.56
CX3CR1	3:39302415	rs112580659	Intergenic	G	0.99	-0.05	0.25
CX3CR1	3:39302659	rs79659083	Intergenic	A	0.94	0.02	0.43
CX3CR1	3:39303147	rs4676487	Intergenic	T	0.56	-0.01	0.45
CX3CR1	3:39304464	rs1877563	Downstream	A	0.88	-0.004	0.76
CX3CR1	3:39304549	rs11713282	Downstream	C	0.76	0.002	0.84
CX3CR1	3:39304570	rs17038645	Downstream	A	0.96	-0.02	0.30
CX3CR1	3:39304588	rs17038647	Downstream	C	0.94	-0.02	0.28
CX3CR1	3:39304602	rs11129819	Downstream	T	0.76	0.003	0.77
CX3CR1	3:39304794	rs11129820	Downstream	T	0.69	0.0003	0.98
CX3CR1	3:39304818	rs9826296	Downstream	A	0.86	0.001	0.96
CX3CR1	3:39305304	rs17038663	3' UTR	T	0.95	-0.02	0.37
CX3CR1	3:39306134	rs76874165	3' UTR	T	0.98	-0.05	0.18
CX3CR1	3:39306219	rs11710546	3' UTR	A	0.76	0.002	0.84
CX3CR1	3:39306605	rs17038674	3' UTR	T	0.96	-0.02	0.29
CX3CR1	3:39306784	rs1050592	3' UTR	G	0.76	0.003	0.79
CX3CR1	3:39307162	rs3732378	Exonic (Nonsynonymous)	A	0.85	0.01	0.63
CX3CR1	3:39307256	rs3732379	Exonic (Nonsynonymous)	T	0.76	0.002	0.88
CX3CR1	3:39307962	rs41535248	Exonic (Nonsynonymous)	A	0.99	-0.05	0.24
CX3CR1	3:39308205	rs55955702	Intronic	A	0.99	-0.05	0.26
CX3CR1	3:39308293	rs4271863	Intronic	T	0.50	0.01	0.49
CX3CR1	3:39308298	rs141909558	Intronic	T	0.99	0.01	0.77
CX3CR1	3:39309108	rs2669850	Intronic	C	0.58	-0.01	0.47
CX3CR1	3:39309215	rs17793056	Intronic	C	0.52	-0.01	0.16
CX3CR1	3:39310269	rs57345776	Intronic	T	0.50	-0.01	0.57
CX3CR1	3:39310764	rs116395001	Intronic	C	0.98	0.005	0.90
CX3CR1	3:39310793	rs145883535	Intronic	G	0.99	0.03	0.65
CX3CR1	3:39311087	rs140133131	Intronic	T	0.99	-0.02	0.79
CX3CR1	3:39311345	rs56379504	Intronic	G	0.81	-0.01	0.42
CX3CR1	3:39311583	rs9862876	Intronic	G	0.78	-0.01	0.48
CX3CR1	3:39311657	rs7615733	Intronic	G	0.69	-0.003	0.78
CX3CR1	3:39311666	rs13077357	Intronic	T	0.61	0.001	0.92
CX3CR1	3:39312017	rs78796740	Intronic	A	0.99	-0.08	0.07
CX3CR1	3:39312941	rs9868689	Intronic	T	0.81	-0.01	0.35
CX3CR1	3:39313391	rs35660161	Intronic	G	0.98	-0.02	0.54
CX3CR1	3:39313443	rs56110221	Intronic	G	0.01	-0.11	0.07
CX3CR1	3:39313524	rs34808142	Intronic	T	0.97	-0.06	0.04

CX3CR1	3:39314574	rs4423707	Intronic	T	0.91	0.01	0.57
CX3CR1	3:39315319	rs55675170	Intronic	G	0.99	-0.03	0.50
CX3CR1	3:39315901	rs56391246	Intronic	T	0.90	-0.001	0.97
CX3CR1	3:39316416	rs4676624	Intronic	C	0.94	-0.07	0.05
CX3CR1	3:39316828	rs12636547	Intronic	C	0.91	0.01	0.50
CX3CR1	3:39316976	rs4676625	Intronic	A	0.68	-0.01	0.17
CX3CR1	3:39317913	rs13062158	Intronic	C	0.69	0.01	0.23
CX3CR1	3:39318001	rs56095464	Intronic	G	0.59	-0.01	0.52
CX3CR1	3:39318192	rs56039226	Intronic	A	0.96	-0.02	0.32
CX3CR1	3:39318238	rs56386815	Intronic	T	0.99	-0.04	0.45
CX3CR1	3:39318288	rs2853712	Intronic	C	0.57	-0.01	0.38
CX3CR1	3:39318704	rs2669841	Intronic	T	0.73	-0.003	0.80
CX3CR1	3:39318797	rs2853711	Intronic	T	0.73	-0.003	0.80
CX3CR1	3:39319037	rs72865917	Intronic	G	0.91	0.01	0.49
CX3CR1	3:39319197	rs6796033	Intronic	G	0.57	-0.01	0.36
CX3CR1	3:39319288	rs56239258	Intronic	G	0.99	-0.06	0.26
CX3CR1	3:39319510	rs56035529	Intronic	T	0.91	0.003	0.86
CX3CR1	3:39320000	rs11720041	Intronic	T	0.82	-0.01	0.64
CX3CR1	3:39320055	rs2669843	Intronic	G	0.87	-0.01	0.70
CX3CR1	3:39320511	rs7622254	Intronic	T	0.98	-0.03	0.41
CX3CR1	3:39320598	rs116583694	Intronic	A	0.90	-0.01	0.74
CX3CR1	3:39320644	rs75098903	Intronic	T	0.97	-0.06	0.03
CX3CR1	3:39321218	rs2669845	Intronic	T	0.87	-0.01	0.70
CX3CR1	3:39321373	rs41376750	Intronic	A	0.98	-0.04	0.39
CX3CR1	3:39321412	rs41336745	Intronic	T	0.97	0.03	0.49
CX3CR1	3:39321516	rs36230801	5' UTR	A	0.98	-0.03	0.42
CX3CR1	3:39321710	rs35500272	Intronic	T	0.89	0.005	0.74
CX3CR1	3:39321770	rs9813187	5' UTR	A	0.83	-0.01	0.69
CX3CR1	3:39321805	rs36230797	5' UTR	C	0.89	-0.002	0.91
CX3CR1	3:39321867	rs871610	5' UTR	T	0.72	0.01	0.16
CX3CR1	3:39322466	rs871144	Intronic	T	0.68	-0.01	0.23
CX3CR1	3:39322483	rs55695898	Intronic	T	0.99	-0.04	0.37
CX3CR1	3:39322542	rs56156211	5' UTR	A	0.99	-0.04	0.36
CX3CR1	3:39322665	rs938203	5' UTR	A	0.84	-0.003	0.84
CX3CR1	3:39322826	rs2669846	Intronic	T	0.66	0.01	0.15
CX3CR1	3:39322843	rs2853708	Intronic	C	0.60	-0.01	0.29
CX3CR1	3:39323163	rs11715522	Exonic (Nonsynonymous)	C	0.61	0.01	0.14
CX3CR1	3:39323177	rs147724093	Exonic (Nonsynonymous)	C	0.99	-0.02	0.81
CX3CR1	3:39323423	rs11917223	Upstream	G	0.71	0.01	0.21
CX3CR1	3:39323542	rs62244246	Upstream	A	0.97	0.06	0.11
CX3CR1	3:39323765	rs11716530	Upstream	T	0.78	0.01	0.61
CX3CR1	3:39323843	rs13098237	Upstream	C	0.72	0.01	0.39
CX3CR1	3:39323847	rs13098239	Upstream	C	0.71	0.01	0.42
CX3CR1	3:39323992	rs9861437	Upstream	G	0.89	0.001	0.92
CX3CR1	3:39324065	rs6783639	Upstream	G	0.61	0.01	0.20
CX3CR1	3:39324246	rs76897474	Intergenic	C	0.89	0.01	0.58
CX3CR1	3:39324283	rs2853707	Intergenic	G	0.77	-0.02	0.13

CX3CR1	3:39325104	rs10865886	Intergenic	T	0.67	0.01	0.32
CX3CR1	3:39325126	rs192343698	Intergenic	C	0.02	0.02	0.56
CX3CR1	3:39325128	rs149810846	Intergenic	G	0.88	0.01	0.60
CX3CR1	3:39325227	rs188646763	Intergenic	T	0.99	0.01	0.92
CX3CR1	3:39325489	rs4256069	Intergenic	G	0.45	-0.01	0.32
CX3CR1	3:39325523	rs3020453	Intergenic	C	0.77	-0.02	0.10
CX3CR1	3:39325614	rs2965057	Intergenic	G	0.82	0.01	0.44
CX3CR1	3:39325677	rs3926044	Intergenic	T	0.59	-0.01	0.27
CX3CR1	3:39326084	rs11720953	Intergenic	A	0.90	-0.003	0.86
CX3CR1	3:39326283	rs1014638	Intergenic	G	0.67	0.01	0.50
CX3CR1	3:39326317	rs111791069	Intergenic	A	0.99	0.05	0.29
CX3CR1	3:39326511	rs938200	Intergenic	A	0.62	-0.02	0.10
CX3CR1	3:39327174	rs938199	Intergenic	A	0.62	-0.02	0.10
CX3CR1	3:39327376	rs13062901	Intergenic	T	0.67	0.01	0.50
CX3CR1	3:39327449	rs12486535	Intergenic	T	0.67	0.01	0.52
CX3CR1	3:39327556	rs187302965	Intergenic	G	0.99	-0.04	0.57
CX3CR1	3:39327676	rs4270454	Intergenic	T	0.67	0.01	0.50
CX3CR1	3:39327736	rs4271864	Intergenic	C	0.98	-0.07	0.07
CX3CR1	3:39327784	rs4271865	Intergenic	A	0.67	0.01	0.52
CX3CR1	3:39328082	rs190087508	Intergenic	A	0.99	-0.03	0.63
CCR2	3:46390228	rs35728689	Intergenic	A	0.89	-0.001	0.96
CCR2	3:46391071	rs6441971	Intergenic	C	0.76	-0.004	0.70
CCR2	3:46391390	rs6441972	Intergenic	A	0.67	-0.01	0.24
CCR2	3:46391648	rs35943069	Intergenic	G	0.91	-0.02	0.23
CCR2	3:46391788	rs17141006	Intergenic	G	0.89	-0.001	0.97
CCR2	3:46392060	rs17141010	Intergenic	T	0.89	-0.001	0.97
CCR2	3:46392089	rs1894387	Intergenic	T	0.91	-0.02	0.17
CCR2	3:46392131	rs1894388	Intergenic	T	0.91	-0.02	0.18
CCR2	3:46392162	rs62242995	Intergenic	A	0.89	-0.0001	1.00
CCR2	3:46392265	rs768539	Intergenic	A	0.76	-0.004	0.70
CCR2	3:46392976	rs34473395	Intergenic	T	0.91	-0.02	0.17
CCR2	3:46393463	rs3918354	Intergenic	G	0.91	-0.02	0.17
CCR2	3:46393827	rs3918355	Intergenic	T	0.96	0.003	0.92
CCR2	3:46393970	rs3918357	Intergenic	A	0.89	0.00001	1.00
CCR2	3:46394419	rs3918358	Upstream	C	0.67	-0.01	0.24
CCR2	3:46394680	rs3918359	Upstream	A	0.76	-0.004	0.70
CCR2	3:46395313	rs3749461	5' UTR	G	0.91	-0.02	0.16
CCR2	3:46395585	rs3092964	5' UTR	G	0.76	-0.004	0.71
CCR2	3:46395615	rs3918376	5' UTR	C	0.99	-0.02	0.76
CCR2	3:46395786	rs3918361	Intronic	A	0.78	0.002	0.83
CCR2	3:46395930	rs3918362	Intronic	T	0.91	-0.02	0.16
CCR2	3:46396616	rs3762823	Intronic	A	0.78	0.002	0.83
CCR2	3:46396938	rs3092963	Intronic	G	0.68	-0.01	0.49
CCR2	3:46397039	rs3092962	Intronic	A	0.78	0.003	0.82
CCR2	3:46397440	rs3092961	Intronic	A	0.43	0.01	0.45
CCR2	3:46398159	rs3918363	Intronic	T	0.89	-0.0003	0.99
CCR2	3:46398291	rs3918364	Intronic	T	0.89	-0.0003	0.99
CCR2	3:46398364	rs3918365	Intronic	G	0.91	-0.02	0.16
CCR2	3:46399174	rs3918367	Exonic	T	0.99	-0.08	0.20

			(Synonymous)				
CCR2	3:46399208	rs1799864	Exonic (Nonsynonymous)	A	0.90	0.0001	0.99
CCR2	3:46399798	rs1799865	Exonic (Synonymous)	C	0.68	-0.01	0.48
CCR2	3:46400062	rs3092960	Exonic (Synonymous)	A	0.88	-0.01	0.39
CCR2	3:46401032	rs3138042	Intronic	G	0.68	-0.01	0.47
CCR2	3:46401606	rs140253702	3' UTR	G	0.99	-0.01	0.86
CCR2	3:46402018	rs743660	3' UTR	A	0.78	0.002	0.85
CCR2	3:46402053	rs34138562	3' UTR	G	0.89	-0.001	0.94
CCR2	3:46402431	rs11575062	Downstream	T	0.91	-0.02	0.16
CCR2	3:46402564	rs762788	Downstream	T	0.78	0.002	0.88
CCR2	3:46402627	rs762789	Downstream	A	0.66	-0.01	0.56
CCR2	3:46402645	rs71327057	Downstream	C	0.91	-0.02	0.16
CCR2	3:46402688	rs762790	Downstream	G	0.87	-0.01	0.44
CCR2	3:46402734	rs34041956	Downstream	A	0.91	-0.02	0.16
CCR2	3:46403240	rs3918368	Downstream	A	0.91	-0.02	0.20
CCR2	3:46403315	rs6441973	Downstream	A	0.43	0.01	0.44
CCR2	3:46403401	rs1034382	Downstream	T	0.77	0.002	0.89
CCR2	3:46403468	rs3092959	Intergenic	A	0.91	-0.02	0.16
CCR2	3:46403681	rs3092958	Intergenic	A	0.91	-0.02	0.18
CCR2	3:46403961	rs3092957	Intergenic	A	0.91	-0.02	0.16
CCR2	3:46404163	rs2373226	Intergenic	T	0.77	0.003	0.81
CCR2	3:46404270	rs150203971	Intergenic	G	0.99	-0.04	0.51
CCR2	3:46404740	rs34944500	Intergenic	T	0.84	0.005	0.72
CCR2	3:46404742	rs34030880	Intergenic	T	0.64	-0.003	0.81
CCR2	3:46404744	rs35893284	Intergenic	A	0.47	0.001	0.89
CCR2	3:46404897	rs139885889	Intergenic	A	0.96	0.02	0.43
CCR2	3:46406367	rs2213290	Intergenic	T	0.60	0.01	0.17
CCR2	3:46406546	rs143226343	Intergenic	T	0.99	0.004	0.94
CCR2	3:46406578	rs2373227	Intergenic	C	0.83	0.02	0.12
CCR5	3:46408180	rs35513549	Intergenic	A	0.91	-0.02	0.14
CCR5	3:46408731	rs2040388	Intergenic	G	0.59	0.003	0.75
CCR5	3:46409113	rs3136535	Intergenic	A	0.87	-0.01	0.42
CCR5	3:46410036	rs7637813	Intergenic	G	0.70	0.01	0.51
CCR5	3:46410137	rs41490645	Intergenic	C	0.88	-0.01	0.36
CCR5	3:46410494	rs2856757	Intergenic	C	0.62	0.02	0.11
CCR5	3:46410936	rs2734225	Upstream	T	0.64	0.02	0.07
CCR5	3:46411542	rs2227010	Upstream	G	0.57	0.0005	0.96
CCR5	3:46411661	rs2856758	5' UTR	G	0.87	-0.01	0.42
CCR5	3:46411840	rs2734648	Intronic	T	0.63	0.02	0.09
CCR5	3:46411935	rs1799987	Intronic	A	0.47	-0.01	0.55
CCR5	3:46412259	rs1799988	5' UTR	C	0.47	-0.01	0.48
CCR5	3:46412308	rs1800023	5' UTR	G	0.64	0.02	0.06
CCR5	3:46412559	rs1800024	Intronic	T	0.89	0.0003	0.99
CCR5	3:46413334	rs2856762	Intronic	T	0.90	-0.02	0.12
CCR5	3:46413418	rs2254089	Intronic	T	0.64	0.02	0.06
CCR5	3:46413676	rs181867134	Intronic	T	0.98	-0.05	0.41

CCR5	3:46413743	rs2856764	Intronic	T	0.64	0.02	0.06
CCR5	3:46413950	rs2856765	Intronic	A	0.64	0.02	0.06
CCR5	3:46414035	rs41515644	Intronic	G	0.66	0.02	0.10
CCR5	3:46414557	rs1799863	Exonic (Nonsynonymous)	A	0.98	-0.003	0.94
CCR5	3:46414975	rs62625034	Exonic (Nonsynonymous)	T	0.96	-0.04	0.23
CCR5	3:46416216	rs17765882	3' UTR	T	0.90	-0.02	0.11
CCR5	3:46416470	rs1800874	3' UTR	T	0.64	0.02	0.05
CCR5	3:46416686	rs41526948	3' UTR	G	0.98	0.02	0.62
CCR5	3:46417069	rs41442546	3' UTR	A	0.97	0.02	0.64
CCR5	3:46417312	rs746492	3' UTR	G	0.48	-0.01	0.35
CCR5	3:46418342	rs3087251	Downstream	A	0.57	-0.002	0.85
CCR5	3:46418417	rs3087252	Downstream	T	0.64	0.02	0.03
CCR5	3:46418689	rs3087253	Downstream	C	0.57	-0.002	0.84
CCR5	3:46420104	rs11575816	Intergenic	T	0.64	0.02	0.04
CCR5	3:46420170	rs11575815	Intergenic	T	0.64	0.02	0.03
CCR5	3:46420618	rs181392199	Intergenic	G	0.99	-0.06	0.25
CCR5	3:46420781	rs71327059	Intergenic	T	0.91	-0.02	0.14
CCR5	3:46420799	rs3181038	Intergenic	C	0.91	-0.03	0.11
CCR5	3:46421838	rs3181039	Intergenic	C	0.64	0.02	0.04
CCR5	3:46422355	rs11575821	Intergenic	A	0.86	-0.01	0.45
CCR5	3:46422645	rs17715106	Intergenic	T	0.90	-0.02	0.11

Supplemental Table 1: No SNPs within *CCR2, CCR5*, or *CX3CR1* were significantly associated with CAD in the CARDIoGRAMplusC4D meta-analysis. Presented are the 206 SNPs within *CCR2, CCR5*, and *CX3CR1* captured in the CARDIoGRAMplusC4D meta-analysis. None of these SNPs nor the 14 indels in the corresponding genes were significantly associated with CAD after correction for multiple testing. Key: SNP = Single nucleotide polymorphism; CAD = Coronary artery disease; EAF = Effect Allele Frequency.

Gene	Position (hg19)	Change	AA Change	Minor Allele (MAF %)	P-value	Subjects
CCR2	3:46399208	Missense	V64I	A (9.0)	0.43	120565
CCR2	3:46399158	Missense	P47L	T (0.3)	0.82	68833
CCR5	3:46414947	Missense	S185I	T (11.1)	0.012	120557
CCR5	3:46414975	Missense	Q194H	T (11.1)	0.013	120573
CCR5	3:46414696	Nonsense	C101X	A (0.2)	0.032	112293
CCR5	3:46414557	Missense	L55Q	A (2.3)	0.14	120555
CCR5	3:46414573	Missense	R60S	T (0.2)	0.19	105867
CCR5	3:46414611	Missense	A73V	T (0.2)	0.43	119401
CCR5	3:46415066	Nonsense	R225X	T (0.1)	0.75	38938
CCR5	3:46415255	Missense	T288A	G (0.4)	0.78	47885
CCR5	3:46415061	Missense	R223Q	A (0.2)	0.88	108979
CX3CR1	3:39307832	Missense	T57A	C (0.5)	0.009	119734
CX3CR1	3:39323163	Missense	F8L	C (38.5)	0.39	120570
CX3CR1	3:39307962	Missense	E13D	A (1.2)	0.44	120573
CX3CR1	3:39307162	Missense	T280M	A (17.1)	0.45	120575
CX3CR1	3:39307256	Missense	V249I	T (27.8)	0.48	120558
CX3CR1	3:39307927	Missense	D25G	C (0.2)	0.49	114667
CX3CR1	3:39307637	Missense	V122I	T (0.1)	0.60	112828
CX3CR1	3:39307125	Missense	I292M	C (0.1)	0.84	77082
CX3CR1	3:39323177	Missense	P4A	C (0.9)	0.99	120516

Supplemental Table 2: No SNPs within *CCR2, CCR5*, or *CX3CR1* captured in the MIGen and CARDIoGRAM Exome array meta-analysis were significantly associated with CAD. Of the 20 polymorphic SNPs with MAF $\geq$ 0.1% in *CCR2, CCR5*, and *CX3CR1* captured in the MIGen and CARDIoGRAM Exome array dataset, none were significantly associated with CAD after correction for multiple testing. Key: CAD= Coronary artery disease; AA = Amino acid; MAF = Minor Allele Frequency; SNP = Single nucleotide polymorphism.

rs#	Location	Minor allele	CARDIoGRAMplusC4D (n=184,305)			PROMIS (n=17,437)			LD with V249I (R^2)		LD with T280M (R^2)	
			MAF (%)*	Beta	P-Value	MAF (%)*	Beta	P-Value	CEU	SAS	EUR	SAS
rs1050592†	3' UTR	G	28.63	-0.003	0.79	12.78	-0.12	1.59×10^{-4}	1.00	1.00	0.58	0.82
rs11129819†	Downstream	T	28.53	-0.003	0.77	12.78	-0.11	2.49×10^{-4}	1.00	1.00	0.58	0.82
rs11713282	Downstream	C	28.63	-0.002	0.84	12.88	-0.11	2.56×10^{-4}	1.00	0.99	0.58	0.81
rs3732379†	Exon	T	28.53	-0.002	0.88	12.78	-0.11	2.64×10^{-4}	-	-	0.58	0.82
rs11710546†	3' UTR	A	28.53	-0.002	0.84	12.78	-0.11	2.67×10^{-4}	1.00	1.00	0.58	0.82
rs73060524	Downstream	A	28.63	-0.002	0.83	12.88	-0.11	2.69×10^{-4}	1.00	0.98	0.58	0.80

* MAF per the 1000 genomes phase 3 EUR and SAS reference panels respectively.
† Variant genotyped in PROMIS.

Supplemental Table 3. *CX3CR1* variants significantly associated with MI in PROMIS: Values significant at a Bonferroni correction threshold of 2.76×10^{-4} (n=181). Key: MI = Myocardial infarction; MAF = Minor allele frequency; LD = Linkage disequilibrium; UTR= Untranslated region.

rs#	Location	Minor allele	PROMIS			LD with V249I (R^2)		LD with T280M (R^2)	
			MAF (%)*	Beta	P-Value	CEU	SAS	CEU	SAS
rs17038647	Downstream	C	4.70	0.22	1.61×10^{-6}	0.00	0.02	0.00	0.02
rs75540383	Downstream	C	4.70	0.22	3.15×10^{-6}	0.00	0.02	0.00	0.02
rs17038663†	3' UTR	T	4.60	0.22	3.29×10^{-6}	0.00	0.02	0.00	0.02

* MAF per 1000 genomes phase 3, version 5 SAS reference panel.
† Variant genotyped in PROMIS.

Supplemental Table 4. *CX3CR1* variants significantly associated with type II DM in PROMIS: Values significant at a Bonferroni correction threshold of 2.76×10^{-4} (n=181). The three variants are in near perfect LD with one another (r^2 >0.97) though not with the *CX3CR1* variants V249I and T280M. Key: DM = Diabetes mellitus; MAF = Minor allele frequency; LD = Linkage disequilibrium; UTR= Untranslated region.

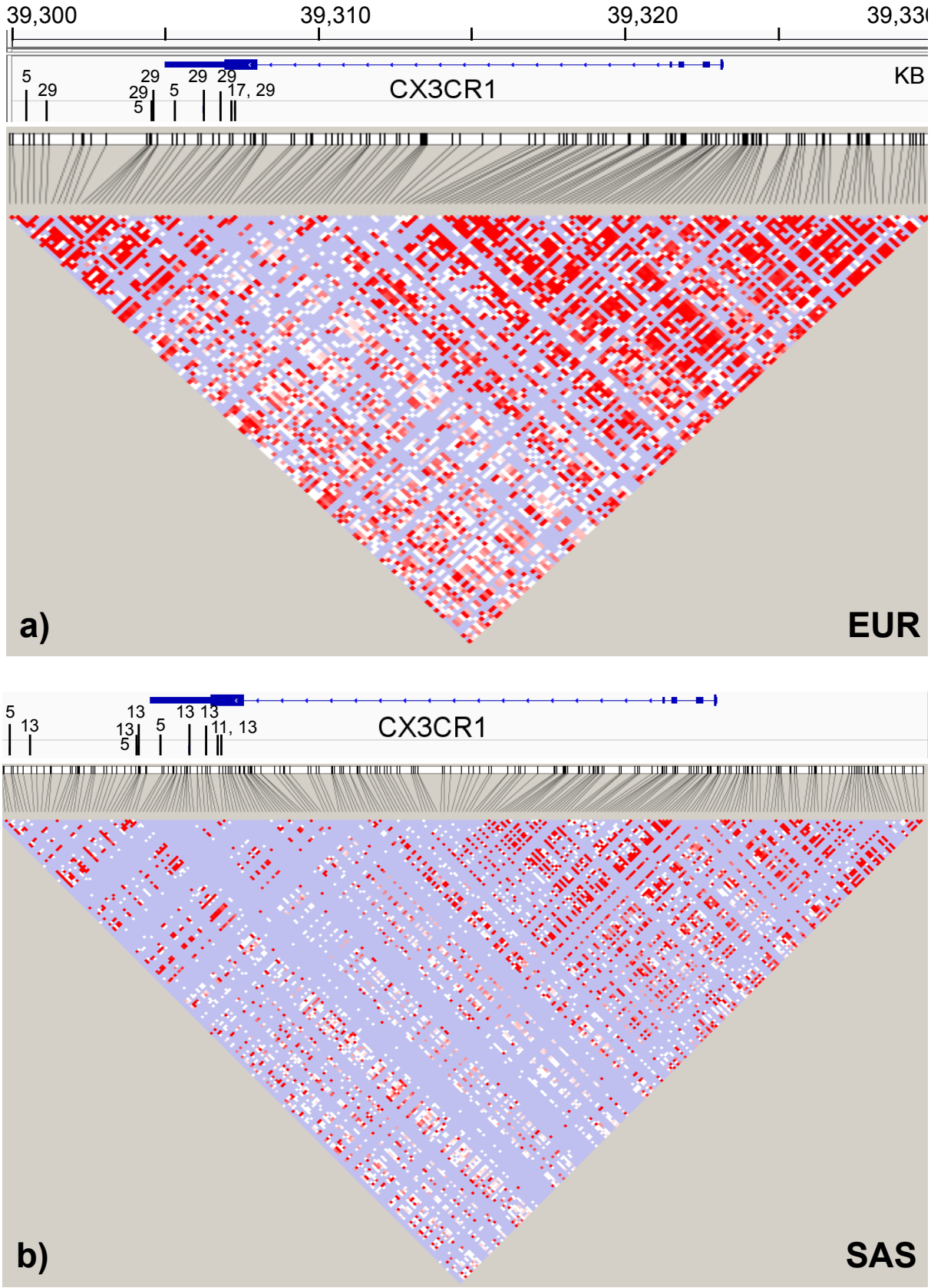
a) CARDIoGRAMplusC4D, 1000 genomes imputed (based on GWAS of 60,801 case subjects and 123,504 control subjects)

Risk AF	AF 0.05	AF 0.1	AF 0.15	AF 0.2	AF 0.25	AF 0.3	AF 0.35	AF 0.4	AF 0.45	AF 0.5
0.054	0.033	1	1	1	1	1	1	1	1	1
0.103	1	0.001	1	1	1	1	1	1	1	1
0.158	1	1	0.229	1	1	1	1	1	1	1
0.216	1	1	1	0.989	1	1	1	1	1	1
0.266	1	1	1	1	0.97	1	1	1	1	1
0.307	1	1	1	1	1	0.01	1	1	1	1
0.380	1	1	1	1	1	1	1	0.998	1	1
0.403	1	1	1	1	1	1	1	0	1	1
0.444	1	1	1	1	1	1	1	1	0.002	1
0.501	1	1	1	1	1	1	1	1	1	0

b) PROMIS (based on GWAS of 9,058 case subjects and 8,379 control subjects)

Risk AF	AF 0.05	AF 0.1	AF 0.15	AF 0.2	AF 0.25	AF 0.3	AF 0.35	AF 0.4	AF 0.45	AF 0.5
0.054	0	1	1	1	1	1	1	1	1	1
0.103	1	0	1	1	1	1	1	1	1	1
0.158	1	1	0	0.958	1	1	1	1	1	1
0.216	1	1	1	0.002	0.477	1	1	1	1	1
0.266	1	1	1	1	0.001	0.33	1	1	1	1
0.307	1	1	1	1	0.998	0	0.719	1	1	1
0.380	1	1	1	1	1	1	0.086	0.003	1	1
0.403	1	1	1	1	1	1	0.962	0	0.796	1
0.444	1	1	1	1	1	1	1	0.656	0	0.977
0.501	1	1	1	1	1	1	1	1	0.899	0

Supplemental Table 5: CARDIoGRAMplusC4D but not PROMIS has ample power to detect genetic variation at a range of allele frequencies. Displayed is the power calculated using actual risk allele frequencies taken from CARDIoGRAMplusC4D tested against a range of theoretical allele frequency differences (i.e. odds ratios) under a genome-wide significance threshold of $5x10^{-8}$. Key: AF= Allele frequency.



Supplemental Figure 1: LD plots for European and South Asian subjects. Displayed are the LD plots for European (a) and South Asian (b) subjects with respect to *CX3CR1*. Hash marks above figures correspond to the nine significant variants in PROMIS as well to the V249I and T280M variants. Approximate MAFs (%) are denoted above the hash marks. Key: LD = linkage disequilibrium; MAF = Minor allele frequency.

Appendix: Data presented on behalf of CARDIoGRAMplusCD, Myocardial Infarction Genetics (MIGen) and CARDIoGRAM Exome, Exome Sequencing Project and Early-Onset Myocardial Infarction (ESP EOMI), and the Pakistan Risk of Myocardial Infarction Study (PROMIS) consortia.

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