Supplementary Table A1: Framingham Offspring characteristics at baseline for each of four time periods in the pooled analysis.

	Period 1		Period 2		Period 3		Period 4			Total	
Offspring examination	Exam 1-2		Exam 2-4		Exam 4-6		Exam 6-8			Exam 1-8	
Calendar year	1971 - 1983		1979 - 1991		1987 - 1998		1995 - 2007			1971 - 2007	
Period interval length, years (SD)*	7.9	0.6	7.8	0.6	7.7	0.7	9.5	1.0		8.2	1.0
Number of subjects	3,213		2,740		2,783		2,622			11,358	
Age, mean years (SD)	36	9.4	43	9.8	51	9.8	58	9.6		46	12.7
Female (%)	53.3		53.0		52.8		54.8			53.5	
Self-reported family history of diabetes (%)	17.3		17.8		18.0		17.8			17.7	
BMI, mean kg/m2 (SD) †	25.0	4.1	25.3	4.2	26.5	4.6	27.5	4.8		26.0	4.5
Systolic blood pressure, mean mm Hg (SD)	121	15.4	122	17.9	128	21.6	132	22.9		126	20.1
HDL-C, mean mg/dL (SD) ‡	51.9	14.7	49.1	13.4	50.5	14.7	52.2	16.1		50.8	14.8
Triglyceride, mean mg/dL (SD)	89	68.2	99	80.3	118	88.2	133	82.1		109	81.4
Fasting plasma glucose, mean mg/dL (SD)	91	8.0	91	8.9	91	9.0	97	9.8		92	9.2
Number of diabetes cases at end of interval	54		66		163		163			446	

*SD: standard deviation. †BMI: body mass index, weight in kg/square of height in m. ‡HDL: high density lipoproteins.

Supplementary Table A2: Genetic loci in descending order position, genotype frequencies and individual associations with diabetes in Framingham for the 40 autosomal SNPs associated with type 2 diabetes in prior studies (4-10).

SNP*	Locus†	Chr‡	Risk allele	Risk allele frequency in Framingham§	Odds Ratio per risk allele for diabetes in Framingham	95% Confidence Interval	*P* Value
rs10923931	*NOTCH2*	1	T	9.6%	1.05	0.86 - 1.35	0.51
rs340874	*PROX1*	1	C	52.1%	1.16	0.98 - 1.36	0.08
rs780094	*GCKR*	2	C	55.1%	1.13	0.98 - 1.29	0.09
rs7578597	*THADA*	2	T	89.9%	1.30	1.01 - 1.68	0.04
rs243021	*BCL11A*	2	A	44.9%	1.11	0.96 - 1.27	0.15
rs7593730	*RBMS1/ITGB6*	2	C	23.6%	0.92	0.78 - 1.08	0.29
rs7578326	*KIAA1486*	2	A	65.7%	0.99	0.85 - 1.15	0.87
rs1801282	*PPARg*	3	C	88.8%	1.09	0.87 - 1.36	0.46
rs4607103	*ADAMTS9*	3	C	73.4%	1.06	0.91 - 1.24	0.46
rs11708067	*ADCY5*	3	A	79.1%	1.13	0.95 - 1.36	0.17
rs1470579	*IGF2BP2*	3	C	33.3%	1.06	0.93 - 1.23	0.35
rs10010131	*WFS1*	4	G	60.3%	1.02	0.89 - 1.18	0.75
rs4457053	*ZBED3*	5	G	32.9%	0.99	0.85 - 1.16	0.92
rs7754840	*CDKAL1*	6	C	31.4%	1.13	0.98 - 1.31	0.10
rs9472138	*VEGFA*	6	T	28.5%	1.13	0.98 - 1.31	0.10
rs2191349	*DGKB/TME195*	7	T	55.4%	1.14	0.99 - 1.30	0.07
rs864745	*JAZF1*	7	T	49.2%	1.05	0.93 - 1.21	0.41
rs4607517	*GCK*	7	A	18.1%	1.15	0.97 - 1.37	0.11
rs972283	*KLF14*	7	G	52.4%	1.08	0.94 - 1.24	0.26
rs896854	*TP53INP1*	8	T	47.8%	0.99	0.86 - 1.14	0.88
rs13266634	*SLC30A8*	8	C	74.1%	1.01	0.77 - 1.33	0.92
rs10811661	*CDKNA/2B*	9	T	81.7%	1.49	1.21 - 1.80	0.0001
rs13292136	*TLE4*	9	C	94.3%	1.15	0.83 - 1.59	0.41
rs12779790	*CDC123,CAMK1D*	10	G	17.9%	1.01	0.83 - 1.24	0.89
rs1111875	*HHEX*	10	C	59.8%	1.06	0.92 - 1.21	0.45
rs7903146	*TCF7L2*	10	T	31.7%	1.30	1.13 - 1.49	0.0003
rs2334499	*HCCA2*	11	T	38.7%	1.13	0.97 - 1.33	0.13
rs231362	*KCNQ1*	11	G	50.6%	1.01	0.85 - 1.19	0.95
rs2237892	*KCNQ1*	11	C	7.8%	0.97	0.69 - 1.36	0.86
rs5215	*KCNJ11*	11	C	35.8%	1.05	0.91 - 1.20	0.56
rs1552224	*CENTD2*	11	A	86.3%	1.05	0.86 - 1.29	0.63
rs10830963	*MTNR1B*	11	G	28.1%	1.21	1.01 - 1.44	0.04
rs1153188	*DCD*	12	A	74.0%	0.94	0.80 - 1.09	0.39
rs1531343	*HMGA2*	12	C	11.7%	1.08	0.89 - 1.32	0.45
rs7961581	*TSPAN8,LGR5*	12	C	30.2%	1.12	0.96 - 1.29	0.17
rs7957197	*OASL/TCF1*	12	T	80.3%	1.06	0.89 - 1.26	0.52
rs11634397	*ZFAND6*	15	G	65.5%	1.09	0.93 - 1.27	0.28
rs8042680	*PRC1*	15	A	32.9%	0.98	0.85 - 1.13	0.79
rs9939609	*FTO*	16	A	39.7%	1.10	0.95 - 1.26	0.21
rs757210	*HNF1B*	17	T	39.4%	0.88	0.64 - 1.19	0.40

*SNPs: single nucleotide polymorphisms. †rs689 in *INS*, on chromosome 11, previously included in our 18-SNP genetic risk score (1), was not replicated in posterior meta-analyses and is therefore not included in the current analysis. rs5945326, in *DUSP9*, on chromosome X (10), is not included in the analysis, since it was not genotyped or imputed in the Framingham Offspring Study. ‡Chr: chromosome.

§ Risk allele frequency in Framingham: subjects without diabetes

P values and odds ratios for type 2 diabetes associated with individual risk SNPs are estimated from additive pooled logistic regression models with generalized estimating equations adjusted for age, age^2, and sex.

We had 80% statistical power to detect ORs for risk of type 2 diabetes of 1.28 for MAF = 20%, ORs of 1.25 for MAF = 30% and ORs of 1.23 for MAF = 50%, for an uncorrected for multiple testing two-sided α of 0.05.

Supplementary Table A3: Odds ratios (ORs) and risk for incident type 2 diabetes associated with 40 individual SNPs*, a weighted 40-SNP genetic risk score and a weighted 17-SNP genetic risk score in the Framingham Offspring Study, stratified by age (<50 years and ≥50 years old), in the sex-adjusted model.

	Model without genetic information		**Model using 40 individual SNPs**		**Model using 40-SNP weighted risk score**		**Model using prior 17-SNP weighted risk score**	
Subjects < 50 years old (N = 144 diabetes cases)								
Model: Sex-adjusted								
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Men (vs. women)	1.46	(1.05-2.03)	1.48	(1.05-2.07)	1.43	(1.03-2.00)	1.44	(1.03-2.00)
Genetic risk score	-	-	-	-	1.29	(1.19-1.40)	1.40	(1.24-1.57)
C-statistic (95% CI)†	0.547	(0.505-0.588)	0.712	(0.670-0.753)	0.657	(0.611-0.703)	0.645	(0.600-0.690)
P value for difference in C-statistic			3.5×10^{-11}		8.8×10^{-6}		0.00006	
Calibration Chi Square (*P* value)				6.38 (0.6)		9.41 (0.3)		9.64 (0.29)
Net reclassification improvement				24.8%		13.8%		11.0%
P value				5.1×10^{-6}		0.005		0.03
	Model without genetic information		**Model using 40 individual SNPs**		**Model using 40-SNP weighted risk score**		**Model using prior 17-SNP weighted risk score**	
Subjects ≥ 50 years old (N = 302 diabetes cases)								
Model: Sex-adjusted								
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Men (vs. women)	1.57	(1.24-1.99)	1.59	(1.25-2.03)	1.59	(1.25-2.01)	1.59	(1.26-2.01)
Genetic risk score	-	-	-	-	1.12	(1.05-1.19)	1.14	(1.05-1.24)
C-statistic (95% CI)	0.557	(0.528-0.586)	0.630	(0.597-0.663)	0.590	(0.557-0.623)	0.581	(0.548-0.614)
P value for difference in C-statistic			3.2×10^{-6}		0.003		0.01	
Calibration Chi Square (*P* value)				4.27 (0.8)		3.32 (0.9)		2.37 (0.97)
Net reclassification improvement				8.5%		0.5%		-3.1%
P value				0.01		0.9		0.3

*SNP: single-nucleotide polymorphism. †95% CI: 95% confidence interval. No age-adjustment was done in the age-stratified models.

To evaluate the individual contribution of each SNP, we entered one term per SNP (total 40 terms plus terms for sex or clinical variables) in the logistic regression models.

We constructed a weighted genetic risk score using 40 SNPs currently associated with type 2 diabetes, and a weighted genetic risk score using 17 SNPs that we used in our previous report (1). rs689, at *INS*, on chromosome 11, previously included in our 18-SNP genetic risk score (1), was not replicated in posterior meta-analyses and is therefore not included in the current 17-SNP or 40-SNP analyses. Moreover, rs5945326, at *DUSP9*, on chromosome X (10), is not included in the analysis, since there are no available genotyping or imputation data for this SNP in the Framingham Offspring Study.

For the construction of the weighted risk scores, we counted risk alleles (0,1,2) for each genotyped SNP -or its dosage, when imputed- (actual distribution ranging from 28 to 53) and multiplied each SNP genotype by its published beta coefficient for diabetes risk (10). We added up the product of that multiplication at each SNP, divided the sum by twice the sum of the betas and multiplied the result by the number of SNPs.

Odds ratios (ORs), 95% CI and C-statistics for the 144 cases of diabetes in 6,763 person-observations in subjects <50 years old and for the 302 cases of diabetes in 4,595 person-observations in subjects ≥50 years old were calculated using pooled logistic regression with generalized estimating equations. Mean age at diabetes onset was 49.30 years for subjects younger than 50 years at baseline and 66.07 years for subjects older than 50 years.

For NRI evaluation, we established three risk categories (low, intermediate and high). The percentages of low, medium and high risk of diabetes are based on the distribution of the cumulative incidence of diabetes across our population, in which cumulative incidence was low for a predicted risk <2%, intermediate for predicted risks ≥2% and ≤ 8%, and high when predicted risk was >8% (this assumption is an *a priori* requirement for the NRI calculation) (15). NRI is better if more people who develop diabetes are reclassified as higher-risk when the genotype score is added to the model, and more people who remain free of diabetes are classified as lower-risk when the score is added. The NRI is penalized for mis-reclassification; for instance, if many people who develop diabetes are classified as lower risk by adding the genetic risk score to the model.

Supplementary Table A4: Odds ratios (ORs) and risk for incident type 2 diabetes associated with 40 individual SNPs*, a weighted 40-SNP genetic risk score and a weighted 17-SNP genetic risk score in the Framingham Offspring Study, both in sex- and simple clinical variables- adjusted models, for the population overall.

	Model without genetic information		**Model using 40 individual SNPs**		**Model using 40-SNP weighted risk score**		**Model using prior 17-SNP weighted risk score**	
Population overall (N = 446 diabetes cases)								
Model: Sex-adjusted								
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Men (vs. women)	1.53	(1.26-1.85)	1.53	(1.26-1.86)	1.53	(1.26-1.85)	1.53	(1.26-1.85)
Genetic risk score	-	-	-	-	1.17	(1.11-1.22)	1.22	(1.14-1.30)
C-statistic (95% CI)†	0.553	(0.530-0.577)	0.631	(0.605-0.657)	0.606	(0.579-0.632)	0.596	(0.570-0.623)
P value for difference in C-statistic			$3.1x10^{-9}$		$4.9x10^{-6}$		0.00005	
Calibration Chi Square (*P* value)				13.69 (0.09)		3.38 (0.91)		6.94 (0.54)
Net reclassification improvement				13.9%		4.1%		3.1%
P value				$2.9x10^{-14}$		0.0009		0.002
Model: Simple clinical variables -adjusted‡								
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Age (per year)	1.01	(1.00-1.02)	1.01	(1.00-1.02)	1.01	(1.00-1.02)	1.01	(1.00-1.02)
Men (vs. women)	0.78	(0.61-0.99)	0.79	(0.62-1.01)	0.79	(0.62-1.01)	0.80	(0.63-1.02)
Family history of diabetes vs. not	2.19	(1.72-2.77)	2.23	(1.75-2.85)	2.19	(1.72-2.78)	2.19	(1.73-2.78)
Body mass index, per kg/m^2	1.10	(1.07-1.12)	1.11	(1.08-1.15)	1.10	(1.08-1.13)	1.10	(1.08-1.13)
Fasting plasma glucose per mg/dl	1.14	(1.13-1.15)	1.14	(1.12-1.13)	1.14	(1.12-1.15)	1.14	(1.12-1.15)
Systolic blood pressure per mmHg	1.01	(1.01-1.02)	1.01	(1.01-1.02)	1.01	(1.01-1.02)	1.01	(1.01-1.02)
HDL cholesterol, per mg/dL	0.98	(0.97-0.99)	0.98	(0.97-0.99)	0.98	(0.97-0.99)	0.98	(0.97-0.99)
Fasting triglycerides, per mg/dL	1.00	(1.00-1.00)	1.00	(1.00-1.00)	1.00	(1.00-1.00)	1.00	(1.00-1.00)
Genetic risk score	-	-	-	-	1.15	(1.09-1.22)	1.22	(1.12-1.32)
C-statistic (95% CI)	**0.903**	(0.889-0.917)	**0.907**	(0.893-0.921)	**0.906**	(0.892-0.920)	**0.905**	(0.891-0.919)
P value for difference in C-statistic			0.01		0.04		0.11	
Calibration Chi Square (*P* value)				7.59 (0.47)		8.26 (0.41)		11.89 (0.16)
Net reclassification improvement				**4.3%**		**1.8%**		**1.2%**
P value				0.004		0.2		0.15

* SNP: single-nucleotide polymorphism. †95% CI: 95% confidence interval. ‡"Simple clinical variables- adjusted" model included: sex, family history of diabetes (self-report that one or both parents had diabetes), body mass index, fasting glucose level, systolic blood pressure, HDL (high density lipoprotein) and fasting triglycerides levels (3). No age-adjustment was done in the age-stratified models.

To evaluate the individual contribution of each SNP, we entered one term per SNP (total 40 terms plus terms for sex or clinical variables) in the logistic regression models.

We constructed a weighted genetic risk score using 40 SNPs currently associated with type 2 diabetes, and a weighted genetic risk score using 17 SNPs that we used in our previous report (1). rs689, at *INS*, on chromosome 11, previously included in our 18-SNP genetic risk score (1), was not replicated in posterior meta-analyses and is therefore not included in the current 17-SNP or 40-SNP analyses. Moreover, rs5945326, at *DUSP9*, on chromosome X (10), is not included in the analysis, since there are no available genotyping or imputation data for this SNP in the Framingham Offspring Study.

For the construction of the weighted risk scores, we counted risk alleles (0,1,2) for each genotyped SNP -or its dosage, when imputed- (actual distribution ranging from 28 to 53) and multiplied each SNP genotype by its published beta coefficient for diabetes risk (10). We added up the product of that multiplication at each SNP, divided the sum by twice the sum of the betas and multiplied the result by the number of SNPs.

Odds ratios (ORs), 95% CI and C-statistics for the 446 cases of diabetes in 11,358 person-observations in the population overall were calculated using pooled logistic regression with generalized estimating equations.

For NRI evaluation, we established three risk categories (low, intermediate and high). The percentages of low, medium and high risk of diabetes are based on the distribution of the cumulative incidence of diabetes across our population, in which cumulative incidence was low for a predicted risk <2%, intermediate for predicted risks ≥2% and ≤ 8%, and high when predicted risk was >8% (this assumption is an *a priori* requirement for the NRI calculation) (15). NRI is better if more people who develop diabetes are reclassified as higher-risk when the genotype score is added to the model, and more people who remain free of diabetes are classified as lower-risk when the score is added. The NRI is penalized for mis-reclassification; for instance, if many people who develop diabetes are classified as lower risk by adding the genetic risk score to the model.

Supplementary Table A5: List of MAGIC and DIAGRAM+ Consortia investigators.

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