Supplementary material

Haberka M, Machnik G, Kowalówka A, et al. Epicardial, paracardial, and perivascular fat quantity, gene expressions, and serum cytokines in patients with coronary artery disease and diabetes. Pol Arch Intern Med. 2019; 129: 738-746. doi:10.20452/pamw.14961

Table S1 Single nucleotide polymorphisms used in constructing genetic risk scores

Gene	rs number	Odds ratio	Risk allele frequency	Weight	Effect allele	Other allele	Source reference
PROX1	rs340874	1.09	0.477	0.09	C	T	[18]
GCKR	rs780094	1.07	0.363	0.07	C	T	[18]
ADCY5	rs11708067	0.88	0.211	–0.12	G	A	[18]
CDKAL1	rs10946398	1.13	0.336	0.13	C	A	[18]
DGKB / TMEM195	rs2191349	1.09	0.485	0.09	T	G	[18]
SLC30A8	rs11558471	0.89	0.332	–0.11	G	A	[18]
CDKN2B	rs10811661	0.84	0.154	–0.16	C	T	[20]
HHEX	rs7923837	0.90	0.376	–0.10	A	G	[18]
TCF7L2	rs7903146	1.37	0.269	0.37	T	C	[18]
MC4R	rs17782313	1.08	0.265	0.08	C	T	[20]
HHEX	rs5015480	0.89	0.425	–0.11	T	C	[18]
KCNJ11	rs5215	1.07	0.402	0.07	C	T	[18]
MTNR1B	rs10830963	1.09	0.295	0.09	G	C	[18]
FTO	rs8050136	1.15	0.412	0.15	A	C	[18]
FTO	rs8044769	0.90	0.480	–0.10	T	C	[20]
PPARG	rs1801282	0.88	0.136	–0.12	G	C	[20]
IGF2BP2	rs4402960	1.12	0.316	0.12	T	G	[18]
COBLL1	rs13389219	0.92	0.391	–0.08	T	C	[18]
COBLL2	rs7607980	0.88	0.117	–0.12	C	T	[18]

Table describes the single nucleotide polymorphisms (rs number) from which type 2 diabetes genetic risk scores were calculated. These single nucleotide polymorphisms were selected from two large-scale genome-wide association studies [18,20] that estimated the effect size of each single nucleotide polymorphism is explaining the variation in type 2 diabetes prevalence in very large patient populations.

Table S2 Characteristics of the participants by unweighted Genetic Risk Score					
	Low uGRS		**High uGRS**		***P*-value**
	Median	**IQR**	**Median**	**IQR**	
Age, years	37.67	26.12–52.64	37.85	27.30–52.91	0.97
BMI, kg/m^2	26.74	23.78–31.84	27.16	24.45–31.87	0.33
BFM, kg	21.91	14.80–34.24	24.00	16.72–32.18	0.23
PBF, %	28.75	21.04–37.15	30.00	22.71–38.04	0.23
SMM, kg	32.60	25.08–38.32	31.00	25.11–38.64	0.77
SM%, %	39.43	34.33–44.09	38.93	34.04–43.22	0.29
FPG, mg/dl	91.00	83.00–99.00	94.00	89.00–105.00	0.002
2-h PG, mg/dl	86.20	73.75–106.00	91.00	74.50–111.00	0.09
Fasting insulin, μU/ml	9.49	6.25–15.56	9.81	7.18–16.02	0.58
HOMA-β, %	125.29	81.23–214.25	115.45	80.06–173.06	0.09
HOMA-IR	2.15	1.46–3.77	2.30	1.60–3.69	0.18
HbA1c, %	5.40	5.10–5.60	5.40	5.10–5.70	0.24
CHOL, mg/dl	187.00	156.0–217.0	186.00	164.0–211.5	0.91
LDL, mg/dl	104.60	80.75–134.50	106.50	82.56–129.05	0.73
HDL, mg/dl	58.00	50.00–68.00	58.00	49.00–67.00	0.78
TG, mg/dl	91.00	65.75–137.18	94.50	67.48–134.85	0.50
DEI, kcal/day	1728.61	1385–2412	1607.09	1324–2223	0.17
Protein intake, g/day	81.39	62.12–109.77	81.88	60.34–96.87	0.58
Fat intake, g/day	57.84	44.29–84.12	58.23	43.87–81.99	0.45
Carbohydrates intake, g/day	220.80	171.3–286.3	218.72	170.8–283.4	0.72
Physical activity, MET min/week	7464	3412–14340	7587	3588–12980	0.88

Table shows median values and interquartile ranges (IQR). The *P*-value describes significance of difference between participants with high and low unweighted genetic risk score (high uGRS and low uGRS, respectively) using Wilcoxon rank-sum test.
Abbreviations: BFM, body fat mass; BMI, body mass index; CHOL, total cholesterol concentration; DEI, average daily energy intake; FPG, fasting plasma glucose; HbA1c, haemoglobin A1c concentration; HDL, HDL-cholesterol concentration; HOMA-β, homeostatic model assessment of beta cell function; HOMA-IR, homeostatic model assessment of insulin resistance; IQR, interquartile ranges; LDL, LDL-cholesterol concentration; n, sample size; PBF, percent body fat; SMM, skeletal muscle mass; SM%, skeletal muscle percentage; TG, triglycerides concentration; uGRS, unweighted genetic risk score; 2-h PG, 2-hour plasma glucose
SI conversion factors: to convert glucose to mmol/l, multiply by 0.0555; insulin to pmol/l, multiply by 6.0; CHOL to mmol/l, multiply by 0.0259; HDL-cholesterol to mmol/l, multiply by 0.0259; LDL-cholesterol to mmol/l, multiply by 0.0259; TG to mmol/l, multiply by 0.0113.

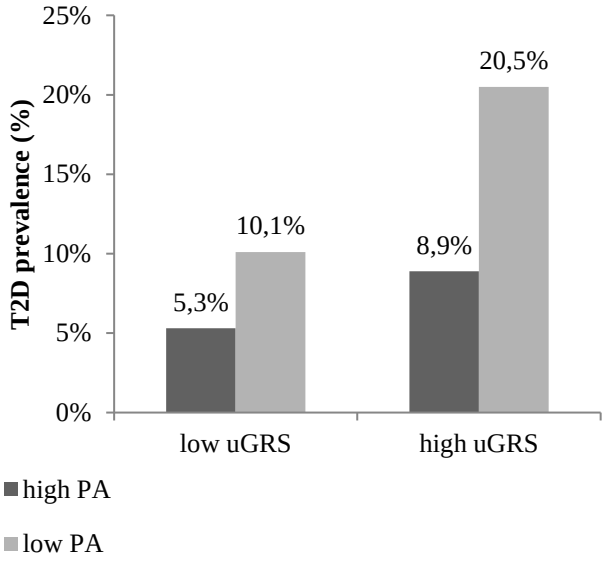

Figure S1 Type 2 diabetes prevalence (%) in unweighted genetic risk score and physical activity groups

References

1 Fuchsberger C, Flannick J, Teslovich TM, et al. The genetic architecture of type 2 diabetes. Nature. 2016; 536: 41–47.

2 Morris AP, Voight BF, Teslovich TM, et al. Large-scale association analysis provides insights into the genetic architecture and pathophysiology of type 2 diabetes. Nat Genet. 2012; 44: 981–990.