

Sagittal Abdominal Diameter as a New Predictor for Incident Diabetes

PIA PAJUNEN, MD, PHD¹
 HARRI RISSANEN, MSC²
 MAARIT A. LAAKSONEN, PHD²

MARKKU HELIÖVAARA, MD, PHD²
 ANTTI REUNANEN, MD, PHD²
 PAUL KNEKT, PHD²

OBJECTIVE—Obesity, particularly visceral adiposity, is a major risk factor for type 2 diabetes. The commonly used obesity indicators, BMI, waist girth, and waist-to-hip ratio (WHR), have limited ability to measure the visceral adipose tissue. Sagittal abdominal diameter (SAD) has been shown to predict the amount of visceral fat. So far no study has been published on its ability to predict diabetes occurrence.

RESEARCH DESIGN AND METHODS—We assessed and compared the prediction of the four obesity indicators for diabetes incidence in a prospective study based on 5,168 participants from the nationally representative Health 2000 study.

RESULTS—During a mean follow-up lasting 8.1 years, 222 incident diabetes cases occurred. In multivariate models adjusted for lifestyle factors, BMI, waist girth, WHR, and SAD were significant predictors of diabetes incidence. The relative risks (95% CI) between high and low levels were 15.0 (6.94–32.6), 11.4 (5.39–23.8), 12.5 (6.47–24.2), and 14.7 (6.89–31.2), respectively. Pairwise interaction analysis showed that the co-occurrence of high BMI and high SAD was associated with the highest diabetes incidence, with a relative risk of 37.0 (11.2–122). After adjustment for waist girth and the components of the metabolic syndrome, the relative risk was 9.88 (2.81–34.7). The corresponding population-attributable fraction estimate was 84% (49–95).

CONCLUSIONS—The combination of SAD and BMI measurements yields a new predictor of diabetes incidence.

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Worldwide, at least 300 million individuals are clinically obese (1). Obesity, particularly visceral adiposity (2–4), is a well-established major modifiable risk factor of type 2 diabetes. Indeed, about 80% of the cases of type 2 diabetes are attributable to obesity (5).

Obesity can be measured with several accurate methods such as magnetic resonance imaging, computed tomography, and dual-energy X-ray absorptiometry (6–8). These methods, however, are not feasible for the needs of large-scale population-based surveys or for clinical work. Anthropometric methods, such as BMI, waist girth, and waist-to-hip ratio (WHR), are commonly used in evaluating obesity (9). Waist girth, with different cutoff

points, is the anthropometric measure incorporated in the most recent definitions of the metabolic syndrome (10–12).

One major shortcoming in using BMI and waist girth is their inability to distinguish between abdominal subcutaneous and visceral adipose tissue (13). Sagittal abdominal diameter (SAD) (i.e., the height of the abdomen when lying supine) is a less commonly used anthropometric measure for assessing the amount of fatty tissue in the abdominal region (14). SAD (14), which predicts the amount of visceral fat measured by computed tomography or magnetic resonance imaging (15–18), has been associated with components of the metabolic syndrome (17,19–21), insulin resistance

(19,22,23), inflammation (23), risk of incident cardiovascular disease (24,25), and diabetes (26).

None of the currently used anthropometric methods—BMI, WHR, or waist girth—is superior for risk prediction of type 2 diabetes (27). To our knowledge, no previous longitudinal study has investigated how SAD compares with other anthropometric measures in predicting incident diabetes. In the current study, we evaluate how well SAD predicts incident diabetes and what its relative importance is compared with BMI and waist girth by presenting population-attributable fraction (PAF) estimates in a general population setting.

RESEARCH DESIGN AND METHODS

Study population and study design

This study is based on data from Health 2000, a health examination survey with a nationally representative sample of 8,028 people aged 30 years or older. The sample was drawn from 80 health service districts throughout Finland using a two-stage cluster sampling (28,29). Of the 6,771 people who participated in the health examination, those 5,590 who were aged 30 to 79 years and did not have diabetes at baseline were included in the current study. After further exclusion of pregnant women and individuals with missing information for the variables used in the analysis, the final dataset comprised 5,168 individuals (2,399 men and 2,769 women). The field phase was conducted in 2000–2001, and the individuals were followed up in a cohort study design for 8.1 years (41,689 person-years of follow-up). During the follow-up, type 2 diabetes developed in 222 individuals.

Baseline measurements

The field phase included a health examination, a home interview, and questionnaires. The study was approved by the ethics committee of the Hospital District of Helsinki and Uusimaa, and all participants gave a written informed consent (28,29). The methods used have been described earlier in detail and are briefly summarized here (29).

From the ¹Diabetes Prevention Unit, Department of Chronic Disease Prevention, Division of Welfare and Health Promotion, National Institute for Health and Welfare, Helsinki, Finland; and the ²Population Health Research Unit, Department of Health, Functional Capacity and Welfare, Division of Welfare and Health Policies National Institute for Health and Welfare, Helsinki, Finland.

Corresponding author: Harri Rissanen, harri.rissanen@thl.fi.

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Weight was measured in lightweight clothes to the nearest 0.1 kg and height to the nearest 0.5 cm. BMI was calculated as body weight (kg)/height squared (m^2). Waist girth was measured midway between the lower rib margin and the iliac crest, and the top layer of the lower body was undressed. SAD was measured using an instrument developed by the technical unit of the National Health Institute. The subject was asked to lie down, with the upper body naked, on the examination table. One of the wings of the measuring device was placed under the subject's back, at the high point of the iliac crest. The subject was asked to relax and breathe regularly. The other wing of the measuring device was lowered gently onto the subject's stomach. The measurement was performed after normal expiration. The intraclass correlation coefficient for the raters' repeatability of the SAD measurements was 0.88, and the agreement between raters was of the same order.

Data on education, smoking, leisure time physical exercise, alcohol consumption, previous diseases, and medication were self-reported in a health interview or a self-administered questionnaire at baseline. Casual blood pressure was measured after a 5-min rest twice at a 1.5-min interval using a standard mercury sphygmomanometer, and the mean of the two measurements was used in the analyses. Participants were asked to fast for at least 4 h before the examination. Blood samples were drawn and stored at $-70^{\circ}C$. Serum HDL cholesterol, triglycerides, and glucose levels were determined as soon as technically possible (usually some weeks) after the samples were taken. Glucose was measured using a hexokinase method (Olympus System Reagent, Hamburg, Germany), and HDL cholesterol (HDL-C Plus, Roche Diagnostics GmbH, Mannheim, Germany) and triglycerides (GPO PAP, Olympus System Reagent) were measured using a photometric enzymatic method.

The harmonization definition (12) was used for defining the metabolic syndrome. A positive definition requires three or more of the following five criteria to be satisfied: large waist girth (≥ 94 cm in men and ≥ 80 cm in women), hypertriglyceridemia (≥ 1.7 mmol/L), low HDL-cholesterol level (< 1.0 mmol/L in men and < 1.3 mmol/L in women), elevated blood pressure (systolic ≥ 130 and/or diastolic ≥ 85 mmHg) and/or antihypertensive drug treatment or history of hypertension, and elevated fasting serum

glucose (≥ 5.6 mmol/L) or antidiabetic drug treatment.

Follow-up measurements

Nationwide administrative registers, which reliably cover every death, hospitalization, and medication use, were used to find cases of incident diabetes by linking the participants of the current study to these registers by a unique personal identification code assigned to each Finnish citizen (5). The identification was based on the presence of any of the ICD-10 codes E10 to E14 in at least one of three registers: the Drug Reimbursement Register (30) and covering all patients with diabetes receiving free hypoglycemic drugs under the Sickness Insurance Act; the Finnish Hospital Discharge Register (31); and the National Causes-of-Deaths Register (32). These data were augmented with information on the purchase of antidiabetic drugs (Anatomical Therapeutic Chemical codes beginning with A10) by individuals who had been diagnosed with diabetes but had not yet received a reimbursement decision according to a nationwide Prescription Register maintained by the Social Insurance Institution.

Statistical analyses

The Cox proportional hazards model was used to calculate the relative risk (RR) for developing incident diabetes during the follow-up. The follow-up time was defined as days from the baseline examination to the date of type 2 diabetes occurrence, death, or end of follow-up, whichever came first. Because of the two-stage cluster sampling design, the analyses were conducted using the program package SUDAAN, release 10, using the Taylor series linearization method and poststratification weights (33). A piecewise constant hazards model was used to assess the PAF for the three obesity indicators (34). The PAF estimates the proportion of cases in a given population that would theoretically not have occurred if all the individuals had had low-risk target values of the risk factors of interest instead of their true values. This is performed by combining information about the prevalence of the risk factor in the population with estimates of the strength of the association between the risk factor and the outcome. The PAF analyses were performed using an SAS macro (35,36).

Four main effect models were used. The first model included age, sex,

education, smoking, alcohol consumption, physical exercise, and one obesity indicator at a time. The second model was otherwise identical to the first model but included all three obesity indicators. In the third and fourth models, serum triglycerides, HDL-cholesterol, glucose, and blood pressure were entered into the first and second models.

Possible interactions between the obesity indicators were also studied in four models. The first model included age, sex, education, smoking, alcohol consumption, physical exercise, and interaction terms of two obesity indicators at a time. The second model was otherwise identical to the first model but also included the third obesity indicator. The two remaining models were formed by entering serum triglycerides, HDL cholesterol, glucose, and blood pressure in the former two models. These eight models were used for estimation of the RR and the PAF.

RESULTS—The mean value of the SAD was 21.5 cm (SD 3.19). Individuals in the highest quartile were more likely to be men, to have a lower educational attainment, to smoke less frequently, to consume more alcohol, and to exercise less than those belonging to the lower quartiles of SAD (Table 1). The likelihood of harmonization metabolic syndrome increased with increasing SAD ($P < 0.001$ for trend, Table 1)

Table 2 displays the adjusted RRs of diabetes in quartiles of SAD, BMI, WHR, and waist girth, and the corresponding PAF. All indicators predicted a more than 10-fold risk of diabetes after adjustment for age, sex, education, smoking, alcohol consumption, and physical exercise (model 1). The RR (95% CI) of diabetes between the highest and lowest quartile was 14.7 (6.89–31.2) for SAD, 15.0 (6.94–32.6) for BMI, 11.4 (5.39–23.8) for waist girth, and 12.5 (6.47–24.2) for WHR. The corresponding PAF estimates (95% CI) were 83% (67–92), 85% (69–93), 79% (61–88), and 78% (63–87), indicating that approximately four of five diabetes cases could have been avoided if the obesity indicators had been in their lowest quartile. After simultaneous inclusion of all three obesity indicators in the model, the RRs were considerably attenuated (model 2). Adjustment of models 1 and 2 with components of the harmonization metabolic syndrome further attenuated the RRs (models 3 and 4). The same was true for the PAF estimates. A total of 61%

Table 1—Age- and sex-adjusted characteristics of subjects in quartiles of SAD

Characteristic	Quartiles of SAD (cm)				P
	13.5–19	19.5–21	21.5–23	23.5–43	
n	1,309	1,423	1,134	1,302	
Age (years) ¹	44.6 (11.5)	48.8 (12.2)	52.4 (12.3)	54.3 (12.3)	<0.001
Males (%) ²	30.1	49.3	57.7	57.4	<0.001
Higher education (%)	35.1	30.9	30.0	27.1	<0.001
Current smokers (%)	30.1	26.1	24.4	23.5	0.007
Alcohol consumption (g/week)	66.8 (109)	77.8 (138)	87.0 (157)	93.5 (165)	<0.001
Exercise at least 4 times/week (%)	29.9	28.1	24.6	22.7	<0.001
Triglycerides (mmol/L)	1.14 (0.45)	1.38 (0.73)	1.69 (1.08)	1.92 (1.11)	<0.001
HDL-cholesterol (mmol/L)	1.51 (0.38)	1.38 (0.36)	1.29 (0.35)	1.18 (0.31)	<0.001
Glucose (mmol/L)	5.27 (0.44)	5.32 (0.46)	5.36 (0.46)	5.43 (0.51)	<0.001
BMI (kg/m ²)	22.1 (2.1)	25.1 (2.1)	27.8 (2.3)	31.8 (4.1)	<0.001
Waist girth (cm)	79.4 (6.9)	87.8 (6.7)	95.2 (6.3)	106.1 (9.8)	<0.001
WHR	0.86 (0.06)	0.90 (0.07)	0.93 (0.07)	0.96 (0.08)	<0.001
Metabolic syndrome harmonization (%) ³	9.4	27.3	52.2	70.1	<0.001
Metabolic syndrome components					
Large waist girth (%) ⁴	16.8	56.6	91.1	98.9	<0.001
Hypertriglyceridemia (%) ⁵	12.2	23.1	35.6	50.1	<0.001
Low HDL (%) ⁶	16.5	26.7	36.9	48.7	<0.001
Elevated blood pressure (%) ⁷	43.1	53.1	64.8	76.0	<0.001
Elevated fasting glucose (%) ⁸	24.7	27.6	30.5	35.5	<0.001

Continuous data are presented as mean (SD). ¹Adjusted for sex. ²Adjusted for age. ³The harmonization definition of metabolic syndrome. ⁴Large waist girth defined as ≥ 94 cm in men and ≥ 80 cm in women. ⁵Hypertriglyceridemia (≥ 1.7 mmol/L). ⁶Low HDL-cholesterol level (< 1.0 in men or < 1.3 mmol/L in women). ⁷Elevated blood pressure (systolic ≥ 130 or diastolic ≥ 85 mmHg) or antihypertensive drug treatment or history of hypertension. ⁸Elevated fasting serum glucose (≥ 5.6 mmol/L).

(19–81) of all diabetes cases could have been avoided if the SAD of all subjects had been in the lowest quartile (model 4). The corresponding values were 60% (15–82) for BMI, 46% (–2 to 72) for waist girth, and 46% (7–68) and for WHR.

RRs of diabetes generated by pairwise interaction terms of the three obesity indicators are reported in Table 3. Of the combinations, the co-occurrence of high BMI and high SAD was associated with the highest diabetes risk. The RR (95% CI) between the highest and lowest quartiles was 37.0 (11.2–122) in model 1, and further adjustment for waist girth and the components of the harmonization metabolic syndrome (model 4) attenuated the RR to 9.88 (2.81–34.7). Adjustment affected the PAF estimates (95% CI) of the BMI and SAD combination only marginally, being 93% (78–98) in model 1 and 84% (49–95) in model 4. The predictive power of the interaction between waist girth and WHR and SAD was moderate, and the interaction between BMI and waist girth proved the least predictive of the combinations (PAF 50% [11–71]). Thus, 50% of the diabetes cases could have been avoided if BMI was < 25 kg/m² and waist girth was < 94 cm in men and < 80 cm in women, whereas 84% could

have been avoided if BMI was < 25 kg/m² and SAD was < 20 cm after adjustment for the other factors of the metabolic syndrome.

CONCLUSIONS—We found that SAD, BMI, WHR, and waist girth all are powerful predictors of incident diabetes during an 8.1-year follow-up period. Our data further revealed that combining information on SAD and BMI outperforms information gained by measuring SAD, BMI, or waist girth alone, or any other combination thereof. Indeed, even after adjustment for age, sex, and lifestyle factors, the combination of high SAD and high BMI showed a nearly 37-fold increased risk of diabetes incidence compared with the risk of individuals who had normal BMI (< 25 kg/m²) and belonged to the lowest SAD quartile (13.5–19 cm). Even when the components of the harmonization metabolic syndrome were taken into account, the risk associated with co-occurring high SAD and high BMI remained nearly 10 times higher. The corresponding PAF was 84% compared with 61% and 60% for the individual components of the combination, respectively. The population-level importance of observing and attempting to influence both of these aspects of obesity in

clinical work would thus be expected to be significant.

To our knowledge, this is the first longitudinal study comparing SAD and other anthropometric measures in predicting diabetes incidence. The results support earlier findings (4) indicating that not only the degree of obesity but also the location of fat is a risk factor for diabetes. SAD correlated strongly with visceral obesity in normal-weight and obese individuals (15–18). When SAD is measured in a supine patient, the loose subcutaneous fat falls toward the sides due to gravity, leaving harder visceral fat to contribute to SAD measurement. Our findings are in line with results showing that SAD is associated with diabetes in a cross-sectional setting (26) and is a predictor of incident cardiovascular disease (24,25), even when SAD is measured with the subject standing (24).

BMI is an overall measure that does not differentiate lean mass from adipose tissue, and waist girth does not differentiate between subcutaneous fat and visceral fat. Thus, it is not surprising that a combination of SAD and BMI, both of which take into account overall fatness and indirectly estimate the amount of visceral fat, seems to be the most powerful risk indicator for diabetes.

Table 2—RR and PAF of diabetes in quartiles of different obesity indicators: SAD, BMI, waist girth, and WHR

Obesity indicator	Cases/at risk n/N	Model 1 RR (95% CI)	Model 2 RR (95% CI)	Model 3 RR (95% CI)	Model 4 RR (95% CI)
SAD (cm) quartile ²					
13.5–19	8/1,309	1	1	1	1
19.5–21	32/1,423	3.47 (1.65–7.30)	2.51 (1.18–5.33)	2.58 (1.23–5.40)	2.16 (1.03–4.51)
21.5–23	45/1,134	5.52 (2.57–11.9)	3.04 (1.37–6.75)	3.28 (1.52–7.11)	2.34 (1.08–5.09)
23.5–43	137/1,302	14.7 (6.89–31.2)	5.11 (2.17–12.0)	6.62 (3.03–14.4)	3.57 (1.54–8.25)
P		<0.001	<0.001	<0.001	0.004
PAF		0.83 (0.67–0.92)	0.70 (0.39–0.86)	0.73 (0.46–0.86)	0.61 (0.19–0.81)
BMI (kg/m ²) quartile ²					
12.2–23.4	7/1,297	1	1	1	1
23.5–26.0	28/1,286	3.67 (1.69–8.00)	2.57 (1.18–5.60)	3.11 (1.42–6.81)	2.43 (1.11–5.33)
26.1–29.1	63/1,297	7.73 (3.50–17.1)	3.95 (1.79–8.69)	4.91 (2.22–10.9)	3.12 (1.43–6.82)
29.2–52.5	124/1,288	15.0 (6.94–32.6)	4.04 (1.76–9.30)	6.89 (3.13–15.2)	2.70 (1.15–6.32)
P		<0.001	0.19	<0.001	0.60
PAF		0.85 (0.69–0.93)	0.70 (0.36–0.86)	0.77 (0.52–0.89)	0.60 (0.15–0.82)
Waist girth (cm) quartile ^{1,2}					
1 (lowest)	10/1,281	1	1	1	1
2	26/1,308	2.42 (1.17–5.00)	1.75 (0.84–3.64)	1.87 (0.91–3.83)	1.53 (0.75–3.12)
3	52/1,296	4.49 (2.16–9.35)	2.41 (1.14–5.09)	2.78 (1.32–5.83)	1.83 (0.87–3.87)
4 (highest)	134/1,283	11.4 (5.39–23.8)	3.41 (1.49–7.81)	5.05 (2.35–10.8)	2.28 (1.00–5.22)
P		<0.001	0.36	<0.001	0.53
PAF		0.79 (0.61–0.88)	0.60 (0.24–0.79)	0.66 (0.38–0.82)	0.46 (–0.02 to 0.72)
WHR quartile ³					
0.621–0.840	14/1,289	1	1	1	1
0.841–0.905	47/1,295	3.37 (1.75–6.48)	2.16 (1.13–4.14)	2.27 (1.21–4.28)	1.79 (0.94–3.36)
0.906–0.970	51/1,295	4.44 (2.30–8.56)	2.12 (1.09–4.13)	2.28 (1.20–4.34)	1.55 (0.80–3.01)
0.971–1.22	110/1,289	12.5 (6.47–24.2)	3.62 (1.79–7.32)	4.70 (2.36–9.34)	2.40 (1.16–4.97)
P		<0.001	0.003	<0.001	0.03
PAF		0.78 (0.63–0.87)	0.59 (0.30–0.76)	0.62 (0.36–0.78)	0.46 (0.07–0.68)

Model 1: Age, sex, education, smoking, alcohol consumption, physical exercise, respective obesity indicator. Model 2: Age, sex, education, smoking, alcohol consumption, physical exercise + ²SAD, BMI, waist girth; ³WHR, SAD, BMI. Model 3: Model 1 + triglycerides, HDL-cholesterol, glucose, blood pressure. Model 4: Model 2 + triglycerides, HDL-cholesterol, glucose, blood pressure. ¹Waist girth (cm) quartile cutoff points: Male, 60–89.5, 90–96, 96.5–104, and 104.5–147; female, 53–77, 77.5–85, 85.5–94.5, and 95–162.

In the current study, waist girth was the least powerful of the obesity indicators studied. In the multivariate Cox proportional hazards regression models, waist girth lost the most of its predictiveness when the models were adjusted for variables correlating with waist girth. Furthermore, in the interaction analyses, the models that included waist girth showed the weakest prediction. Of the obesity indicators, waist girth is the indicator that is currently incorporated in the newest definition of the metabolic syndrome. Although this harmonization definition does predict diabetes incidence in line with earlier definitions (37), the current study suggests that further studies are needed to assess the most powerful obesity measure to be used in the future metabolic syndrome definitions and diabetes risk algorithms.

Before SAD can be more widely used in the clinical setting, properly validated

measurement protocols and evaluation of cutoff values are needed. Study of receiver operating characteristic curves did not, however, reveal any clear cutoff point, suggesting that the diabetes risk changes over the whole range of SAD.

In the current study, SAD identified normal-weight high-risk individuals: those with a BMI of <25 kg/m² but with a large SAD were at increased risk of diabetes (data not shown). However, the number of individuals with a low BMI and a high SAD was low, and thus, the public health value of this particular finding would appear to be marginal. A larger study population is needed to clarify the importance and meaning of this finding.

The strengths of the current study were its prospective design, valid follow-up data, and the comprehensive set of methods used to measure body composition. The anthropometric measurements

were performed by trained personnel who used a systematic protocol. SAD was measured using a noncommercial instrument developed by the technical unit of the National Health Institute, and the reliability of the SAD measurements was high. Furthermore, we used the most recent consensus definition of the metabolic syndrome (12).

Despite the advantages, some limitations should be kept in mind. Our study sample was nationally representative for Finland. However, the Finnish population mainly consists of northern European Caucasian individuals, and thus, our results should be replicated in other types of populations. The number of events was relatively low in some of the subcategories of the cohort, which might have influenced the results. Because of the small number of subjects in the reference group, it was only possibly to do the analyses with men and women combined.

Table 3—RR and PAF of diabetes by interaction between obesity indicators: SAD, BMI, waist girth (WAIST), and WHR

	Cases/at risk n/N	Model 1 RR (95% CI)	Model 2 RR (95% CI)	Model 3 RR (95% CI)	Model 4 RR (95% CI)
BMI*SAD¹					
0 normal and low	3/1,175	1	1	1	1
1 normal and middle	15/814	6.86 (1.91–24.7)	5.37 (1.48–19.5)	5.41 (1.49–19.6)	4.78 (1.32–17.3)
2 overweight or high	68/1,902	13.2 (4.00–43.3)	8.09 (2.43–26.9)	8.13 (2.43–27.2)	6.22 (1.85–20.8)
3 overweight and high	136/1,277	37.0 (11.2–122.4)	14.4 (4.14–50.3)	16.7 (4.90–56.7)	9.88 (2.81–34.7)
P		<0.001	<0.001	<0.001	0.001
PAF		0.93 (0.78–0.98)	0.88 (0.63–0.96)	0.88 (0.64–0.96)	0.84 (0.49–0.95)
WAIST*SAD²					
0 normal and low	5/1,053	1	1	1	1
1 normal and middle	14/731	3.64 (1.35–9.80)	2.81 (1.04–7.58)	2.76 (1.04–7.30)	2.42 (0.91–6.42)
2 elevated or high	66/2,097	6.04 (2.47–14.8)	3.81 (1.54–9.48)	3.72 (1.53–9.04)	2.90 (1.18–7.09)
3 elevated and high	137/1,287	18.9 (7.24–49.2)	7.21 (2.55–20.4)	8.13 (3.10–21.3)	4.72 (1.69–13.2)
P		<0.001	<0.001	<0.001	0.02
PAF		0.87 (0.69–0.94)	0.77 (0.45–0.91)	0.77 (0.46–0.91)	0.68 (0.23–0.87)
BMI*WAIST³					
0 normal and normal	12/1,486	1	1	1	1
1					
2 overweight or elevated	14/841	2.02 (0.93–4.35)	1.56 (0.72–3.38)	1.60 (0.73–3.46)	1.37 (0.63–2.97)
3 overweight and elevated	196/2,841	7.38 (4.04–13.5)	3.22 (1.73–5.97)	3.87 (2.07–7.21)	2.29 (1.21–4.33)
P		<0.001	<0.001	<0.001	0.02
PAF		0.78 (0.63–0.88)	0.62 (0.33–0.78)	0.65 (0.40–0.80)	0.50 (0.11–0.71)
WHR*SAD⁴					
0 low and low	4/1,107	1	1	1	1
1 low and middle	32/1,143	7.29 (2.48–21.5)	4.95 (1.67–14.7)	5.36 (1.82–15.8)	4.35 (1.48–12.8)
2 high or high	74/1,950	10.5 (3.61–30.4)	5.00 (1.67–15.0)	5.59 (1.92–16.2)	3.70 (1.24–11.0)
3 high and high	112/968	31.6 (11.0–91.4)	9.62 (3.02–30.7)	12.3 (4.10–36.7)	6.19 (1.91–20.1)
P		<0.001	<0.001	<0.001	0.004
PAF		0.90 (0.75–0.96)	0.81 (0.49–0.93)	0.83 (0.55–0.94)	0.74 (0.29–0.90)
BMI*WHR⁵					
0 normal and low	8/1,449	1	1	1	1
1					
2 overweight or high	64/1,700	5.76 (2.57–12.9)	3.30 (1.49–7.31)	3.69 (1.69–8.06)	2.58 (1.19–5.63)
3 overweight and high	150/2,019	12.8 (5.74–28.4)	4.63 (2.09–10.2)	5.48 (2.51–11.9)	2.94 (1.35–6.41)
P		<0.001	<0.001	<0.001	0.03
PAF		0.85 (0.71–0.93)	0.73 (0.45–0.86)	0.75 (0.49–0.87)	0.61 (0.21–0.80)

Model 1: Age, sex, education, smoking, alcohol consumption, physical exercise + interaction term. Model 2: Age, sex, education, smoking, alcohol consumption, physical exercise + interaction term + to BMI*SAD: waist girth; to WAIST*SAD: BMI; to WHR*SAD: BMI; to BMI*WHR: SAD. Model 3: Model 1 + triglycerides, HDL-cholesterol, glucose, blood pressure. Model 4: Model 2 + triglycerides, HDL-cholesterol, glucose, blood pressure. ¹BMI*SAD classification: 0 = BMI <25 kg/m² and SAD in the lowest quartile; 1 = BMI <25 kg/m² and SAD in the two middle quartiles; 2 = BMI ≥25 kg/m² or SAD in the highest quartile; 3 = BMI ≥25 kg/m² and SAD in the highest quartile. ²WAIST*SAD classification: 0 = waist girth in men <94 cm or in women <80 cm, and SAD in the lowest quartile; 1 = waist girth in men <94 cm or in women <80 cm, and SAD in the two middle quartiles; 2 = waist girth in men ≥94 cm or in women ≥80 cm, or SAD in the highest quartile; 3 = waist girth in men ≥94 cm or in women ≥80 cm, and SAD in the highest quartile. ³BMI*WAIST classification: 0 = BMI <25 kg/m² and waist girth in men <94 cm or in women <80 cm; 2 = BMI ≥25 kg/m² or waist girth in men ≥94 cm or in women ≥80 cm; 3 = BMI ≥25 kg/m² and waist girth in men ≥94 cm or in women ≥80 cm. ⁴WHR*SAD classification: 0 = WHR below median and SAD in the lowest quartile; 1 = WHR below median and SAD in the two middle quartiles; 2 = WHR above median or SAD in the highest quartile; 3 = WHR above median and SAD in the highest quartile. ⁵BMI*WHR classification: 0 = BMI <25 kg/m² and WHR below median; 2 = BMI ≥25 kg/m² or WHR above median; 3 = BMI ≥25 kg/m² and WHR above median.

Our outcome was clinically incident diabetes, and we did not obtain glucose tolerance tests at the end of the follow-up. Thus, we were only able to identify individuals who received drug treatment for diabetes or were hospitalized or died during the follow-up, but we could not identify undiagnosed individuals or diabetic persons treated with diet only. This means that the incidence of diabetes was underestimated in the follow-up, which

probably makes our estimates rather conservative.

In conclusion, we demonstrated in this longitudinal population-based study that the combination of SAD and BMI measurements may provide a new powerful predictor of incident diabetes in the general population. Because this is the first etiological study to demonstrate the ability of SAD to predict diabetes incidence, future studies have to confirm our

finding until a firm conclusion about screening can be made.

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P.P. wrote the manuscript. H.R. and M.A.L. researched data and reviewed and edited the manuscript. M.H., A.R., and P.K. reviewed the

manuscript and contributed to discussion. H.R. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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