Sagittal Abdominal Diameter and Risk of Sudden Death in Asymptomatic Middle-Aged Men
The Paris Prospective Study I

J.P. Empana, MD, MPH; P. Ducimetiere, PhD; M.A. Charles, MD, MPH; X. Jouven, MD, PhD

Background—Abdominal (visceral) and overall obesity are both related to coronary heart disease mortality risk; however, the relative contribution of these 2 components of fat deposit in the etiology of sudden death is unknown.

Methods and Results—We used the data from 7079 asymptomatic men of the Paris Prospective Study I who were free of ischemic heart disease and who were 43 to 52 years of age at first clinical examination between 1967 and 1972. Body mass index (BMI) and sagittal abdominal diameter (SAD) were measured at baseline and used as markers of overall and abdominal obesity. During a follow-up of 23 years, there were 118 sudden deaths and 192 fatal myocardial infarctions. After adjustment for baseline level of cardiovascular risk factors, trunk subcutaneous fat, and thoracic diameter, the ratio of the fifth over the first quintile of SAD was 2.6 (95% CI 1.0 to 6.7) and 2.6 (95% CI 1.3 to 5.1) for sudden death and fatal myocardial infarction, respectively, and the risk of sudden death increased proportionally with SAD level. The corresponding ratios for BMI were 2.0 (95% CI 1.1 to 3.8) and 1.0 (95% CI 0.6 to 1.7), respectively. Compared with men with low SAD (first tertile) and normal BMI (<25 kg/m²), men with elevated SAD (third tertile) were at increased risk of sudden death but not of fatal myocardial infarction, whether they were of normal weight (multivariate adjusted relative risk 3.0 [95% CI 1.3 to 6.9]) or overweight (BMI ≥25 kg/m²; 1.9 [95% CI 1.0 to 3.9]).

Conclusions—In asymptomatic French middle-aged men, larger SAD was associated with a particularly increased risk of sudden death, independent of BMI level and known cardiovascular risk factors. (Circulation. 2004;110:2781-2785.)

Key Words: epidemiology ■ risk factors ■ death, sudden ■ obesity

Identification of asymptomatic individuals from the general population who are at particular elevated risk of sudden death and who could benefit from primary prevention is a critical challenge in Western industrialized countries.1,2 In a previous work based on data from the Paris Prospective Study I, we reported that body mass index (BMI) was positively associated with the risk of sudden death but not of fatal myocardial infarction (MI), after adjustment for cardiovascular risk factors.3 BMI combines estimates of fat mass and fat-free mass and therefore has questionable clinical relevance, although it is widely used. Several observational studies have shown that the sagittal abdominal diameter (SAD), or waist circumference, or waist-to-hip ratio, was associated with an increased risk of coronary heart disease (CHD) morbidity4–7 and mortality,8–11 which suggests that regional fat adiposity and abdominal fat in particular might contribute to CHD outcome. However, the extent to which abdominal fat deposit is related to sudden death and is a better risk marker of sudden death than overall fat localization is unknown. Using data from the Paris Prospective Study I, a study of asymptomatic middle-aged men whose mortality was prospectively checked for 23 years, we investigated whether SAD was associated with an increased risk of sudden death and if so, whether it was a better risk marker than the widely used BMI.

Methods

Subjects

Details of the Paris Prospective Study I recruitment, design, and procedures have been described elsewhere.12 Briefly, it concerned 7746 native Frenchmen employed by the Paris Civil Service who were 43 to 52 years of age at first examination, which was performed between 1967 and 1972. Subjects underwent ECG and physical examination conducted by a physician, provided blood samples for laboratory tests, and answered questionnaires administered by trained interviewers with regard to sociodemographic factors, family and personal medical histories, and smoking habits. Subjects were asked about parental myocardial infarction, parental age at death, and whether the death was sudden or not. Type 2 diabetes was defined as past or present reported diabetes whether treated or not and a fasting blood glycemia level ≥126 mg/dL at first examination.

Anthropometric Measures

The description of the anthropometric variables has been detailed previously.3 Briefly, a set of anthropometric measurements, includ-
The baseline characteristics of men were further explored by baseline among these 3 predefined groups. On average (SD), sudden death and fatal MI occurred 11.3 (5.3) and 16.2 (6.0) years, respectively, after initial clinical examination that included SADx and BMI assessment. Except for current sports practice, the level of all parameters was significantly different among the 3 groups. The mean level of established cardiovascular risk factors increased from the control group to the group with fatal MI and finally to the group with sudden death. The mean level of SADx and BMI was consistently higher in the sudden death group than in the other 2 groups.

### Baseline Characteristics by Level of SADx

The baseline characteristics of men were further explored by level of SADx in the entire cohort and were reported in Table 2. Men with a larger SADx had a higher amount of trunk subcutaneous fat and thoracic diameter and higher mean level of BMI. Moreover, they had a higher cardiovascular risk (RRs) of sudden death and fatal MI for each upper quintile against the first quintile of SADx and BMI using a Cox proportional hazards regression model. To this end, we included 4 indicator variables of SADx and BMI in separate Cox regression models. Adjustments for the sum of axillary, subumbilical, and subscapular skinfold thicknesses and for the sagittal thoracic diameter were performed to account for trunk subcutaneous fat and thoracic diameter, respectively. Other confounding variables were tobacco consumption (mean number of cigarettes smoked per day in the 5 preceding years), systolic blood pressure, diabetes mellitus, and plasma level of total cholesterol measured at first examination. All analyses were performed on SAS software version 8.2 (SAS Institute Inc).

### Results

**Population Characteristics**

Among the 7079 men retained for analysis, 2083 died, including 603 who died of cardiovascular causes. Of these, 118 and 192 were due to sudden death and MI, respectively. Among the remaining 6769 men, taken as controls, 1773 died of causes other than sudden death and MI, and 4996 remained alive until January 1, 1994.

Table 1 compares the level of cardiovascular risk factors at baseline among these 3 predefined groups. On average (SD), sudden death and fatal MI occurred 11.3 (5.3) and 16.2 (6.0) years, respectively, after initial clinical examination that included SADx and BMI assessment. Except for current sports practice, the level of all parameters was significantly different among the 3 groups. The mean level of established cardiovascular risk factors increased from the control group to the group with fatal MI and finally to the group with sudden death. The mean level of SADx and BMI was consistently higher in the sudden death group than in the other 2 groups.

### Statistical Analysis

ANOVA and Pearson χ² tests, respectively, were used to compare continuous and categorical baseline variables (first examination) among the groups. We computed the age-adjusted relative risks (RRs) of sudden death and fatal MI for each upper quintile against the first quintile of SADx and BMI using a Cox proportional hazards regression model. To this end, we included 4 indicator variables of SADx and BMI in separate Cox regression models. Adjustments for the sum of axillary, subumbilical, and subscapular skinfold thicknesses and for the sagittal thoracic diameter were performed to account for trunk subcutaneous fat and thoracic diameter, respectively.

Other confounding variables were tobacco consumption (mean number of cigarettes smoked per day in the 5 preceding years), systolic blood pressure, diabetes mellitus, and plasma level of total cholesterol measured at first examination. All analyses were performed on SAS software version 8.2 (SAS Institute Inc).

### Follow-Up

The administrative department in charge of the population provided a list of deceased subjects annually until participant retirement. All available data relevant to the causes of death were collected from specific inquiries, ie, medical records from hospital departments or general practitioners identified by relatives of the deceased. After retirement, causes of death were obtained from death certificates. The data were then reviewed by an independent medical committee, specifically inquiries, ie, medical records from hospital departments or general practitioners identified by relatives of the deceased. After retirement, causes of death were obtained from death certificates. The data were then reviewed by an independent medical committee, assigned a principal cause of death for each case. The ninth edition of the International Classification of Diseases was used for coding. Sudden death (code 798.1) was defined as a natural death that occurred within 1 hour of onset of acute symptoms. Fatal MI was diagnosed only if the death was found to be strictly related to an MI.

Men with a diagnosis of ischemic heart disease (MI or angina) established at entry from personal medical history, clinical examination, and ECG were excluded from analysis (n=312), and no men had diagnosed cancer at baseline. On January 1, 1994, the vital status could not be obtained for 355 subjects (4.6%). The analysis was thus conducted on the remaining 7079 subjects.

### Table 1. Baseline Characteristics of Men Who Died of Sudden Death or MI and of Men Who Died of Other Causes or Who Remained Alive During Follow-Up: Paris Prospective Study I

<table>
<thead>
<tr>
<th>Variables Measured at Examination</th>
<th>Sudden Death (n=118)</th>
<th>MI (n=192)</th>
<th>Other Causes of Death or Alive at Follow-Up (n=6789)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delay between examination and outcome, y</td>
<td>11.3 (5.3)</td>
<td>16.2 (6.0)</td>
<td>21.5 (5.3)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, y</td>
<td>47.9 (1.9)</td>
<td>48.1 (1.9)</td>
<td>47.6 (1.9)</td>
<td>0.018</td>
</tr>
<tr>
<td>SADx, cm</td>
<td>23.7 (3.0)</td>
<td>23.3 (2.8)</td>
<td>22.3 (3.1)</td>
<td>0.0001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.2 (3.5)</td>
<td>26.4 (3.3)</td>
<td>25.9 (3.3)</td>
<td>0.0001</td>
</tr>
<tr>
<td>No. of cigarettes/day in past 5 years</td>
<td>15.3 (9.5)</td>
<td>13.9 (10.8)</td>
<td>11.7 (10.6)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Current sports activity</td>
<td>11.1 (13)</td>
<td>13.5 (26)</td>
<td>14.1 (946)</td>
<td>0.64</td>
</tr>
<tr>
<td>Diabetes</td>
<td>12.7 (15)</td>
<td>6.3 (12)</td>
<td>5.4 (368)</td>
<td>0.003</td>
</tr>
<tr>
<td>Parental history of sudden death</td>
<td>20.9 (22)</td>
<td>10.2 (19)</td>
<td>11.1 (718)</td>
<td>0.006</td>
</tr>
<tr>
<td>Parental history of MI</td>
<td>9.4 (10)</td>
<td>12.8 (24)</td>
<td>6.8 (442)</td>
<td>0.004</td>
</tr>
<tr>
<td>Resting heart rate, bpm</td>
<td>72.6 (11.7)</td>
<td>71.2 (11.9)</td>
<td>68.5 (10.3)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>246.0 (45.5)</td>
<td>236.2 (50.9)</td>
<td>221.6 (42.4)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>150.8 (28.7)</td>
<td>150.3 (25.4)</td>
<td>140.7 (21.1)</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Unadjusted mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. *P* values for global test of comparison: ANOVA and χ² tests for continuous and categorical variables, respectively.
profile, as indicated by their increased mean age, daily consumption of cigarettes in the past 5 years, systolic and diastolic blood pressure, resting heart rate, total cholesterol and triglyceride levels, higher prevalence of diabetes, and lower prevalence of an active lifestyle.

Age-Adjusted and Multivariate Analysis of SADx

As shown in Figure 1, the age-adjusted risk of sudden death increased linearly across quintiles of SADx (P for trend = 0.0003), and the RR (95% CI) of the fifth over the first quintile of SADx was 4.1 (2.0 to 8.3). In contrast, the risk of fatal MI did not increase proportionally within the first 4 quintiles of SADx, and only men in the fifth quintile were at significant increased risk (age-adjusted RR of 2.3; 95% CI 1.3 to 3.8). The pattern of increased risk of sudden death and fatal MI by quintiles of SADx remained similar after further adjustment for baseline parameters, including trunk subcutaneous fat, sagittal thoracic diameter, tobacco consumption, systolic blood pressure, diabetes, and plasma level of cholesterol. The RRs of sudden death and fatal MI for the fifth over the first quintile of SADx were 2.6 (1.0 to 6.7) and 2.6 (1.3 to 5.1), respectively.

Age-Adjusted and Multivariate Analysis of BMI

As shown in Figure 2, no clear relationship was observed between increasing levels of BMI across the first 4 quintiles and the risk of sudden death, and only men in the last quintile of BMI had a significant 3-fold age-adjusted increased risk of sudden death (1.7 to 5.7) compared with men from the lowest quintile. Moreover, there was no increased risk of fatal MI across the quintiles of BMI, and the age-adjusted RR of the fifth over the first quintile of BMI was 1.5 (1.0 to 2.5).

After additional adjustment for tobacco consumption, systolic blood pressure, diabetes, and plasma level of cholesterol, higher BMI remained significantly associated with an increased risk of sudden death but not of fatal MI. The multivariate adjusted RR of the fifth over the first quintile of BMI was 2.0 (1.1 to 3.8) and 1.0 (0.6 to 1.7), respectively.

Relative Contribution of SADx and BMI Level

When SADx and BMI were simultaneously introduced in the same multivariate regression Cox model (with 4 indicator variables of SADx and BMI) that included previous cardio-

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**TABLE 2. Baseline Characteristics by Quintile of SADx in Total Sample: Paris Prospective Study I**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Quintiles of SADx, cm (Minimum–Maximum)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Q1 (12–19) (n=1194)</td>
</tr>
<tr>
<td>Age, y</td>
<td>46.8 (1.8)</td>
</tr>
<tr>
<td>No. of cigarettes/day in past 5 years</td>
<td>11.2 (10.5)</td>
</tr>
<tr>
<td>Current sports activity</td>
<td>11.6 (138)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3.4 (41)</td>
</tr>
<tr>
<td>Resting heart rate, bpm</td>
<td>68.1 (10.2)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>130.7 (15.9)</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>73.9 (10.1)</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>211.7 (41.2)</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>116.6 (78.8)</td>
</tr>
<tr>
<td>Trunk subcutaneous fat,* mm</td>
<td>593.8 (242.1)</td>
</tr>
<tr>
<td>Thoracic diameter, cm</td>
<td>15.3 (2.1)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23.5 (2.8)</td>
</tr>
</tbody>
</table>

*Unadjusted mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. ANOVA and χ² tests for trend for continuous and categorical variables, respectively; comparisons between groups were all statistically significant (all P<0.001).

*Sum of axillary, subumbilical, and subscapular skinfold thicknesses.

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Figure 1. Age-adjusted RRs of sudden death and fatal MI by quintile of SAD, Paris Prospective Study I. RRs were estimated by Cox proportional hazards model, with first quintile of SAD as reference category.

Figure 2. Age-adjusted RRs of sudden death and fatal MI by quintile of BMI, Paris Prospective Study I. RRs were estimated by Cox proportional hazards model, with first quintile of BMI as reference category.
vascular risk factors, trunk subcutaneous fat, and sagittal thoracic diameter, SADx but not BMI was associated with a significantly increased risk of both sudden death and fatal MI. The RR (95% CI) of the fifth over the first quintile of SADx was 2.3 (1.0 to 5.3) and 1.9 (1.0 to 3.5) for sudden death and fatal MI, respectively. In this multivariate regression model, neither trunk subcutaneous fat nor sagittal thoracic diameter was significantly predictive of sudden death and fatal MI (data not shown).

We further investigated the predictive value of SADx for sudden death and fatal MI by level of BMI and considered BMI and SADx respectively in 2 (<25 and ≥25 kg/m²) and 3 (tertiles of SADx) categories. Table 3 shows that compared with men with normal BMI (<25 kg/m²) and low SADx (first tertile), men with elevated SADx (third tertile) had a consistently increased risk of sudden death but not fatal MI, whether they were of normal weight (BMI <25 kg/m²; multivariate adjusted RR 3.0; 95% CI 1.3 to 6.9) or were overweight (BMI ≥25 kg/m²; multivariate adjusted RR 1.9; 95% CI 1.0 to 3.9).

**Discussion**

In asymptomatic French middle-aged men whose mortality was assessed prospectively over 23 years, larger SAD measured below the xiphoid level at baseline was specifically predictive of sudden death independent of the level of BMI and of cardiovascular risk factors. To the best of our knowledge, the association between SAD and sudden death has never been reported. In a recent report based on the first 15 years of follow-up of the Paris Prospective Study I, larger SAD was associated with cardiac death (MI, sudden death and heart failure) in smokers and nonsmokers; however, no distinction was made between sudden death and fatal MI. In a case-control study that included 35 cases subjects free of clinical coronary artery disease and 81 aged-matched control subjects (25 deceased and 56 living control subjects, all free of clinical coronary artery disease), the abdominal diameter index as measured by the ratio of abdominal diameter over midthigh circumference was positively associated with the odds of sudden death. This study had the benefit of autopsy confirmation, but the analysis did not take into account the confounding effect of cardiovascular risk factors such as tobacco consumption, systolic blood pressure, diabetes, or plasma cholesterol level.

The positive association between SAD and sudden death was robust in the present analysis. Because this was a prospective study, it is highly unlikely that SADx and BMI measurement were influenced by outcome, and the fact that all anthropometric assessment was made on average more than 10 years before the outcome emphasizes this point. The association was observed beyond the effect of trunk subcutaneous fat and sagittal thoracic diameter. Although diabetes was a strong risk factor for sudden death, the association of SADx with sudden death persisted after adjustment for this variable. Similarly, after exclusion of men with diabetes at entry, men with larger SADx remained at greater risk of sudden death, with a 2-fold increased risk in multivariate analysis (not shown). In multivariate analysis that included classic cardiovascular risk factors, additional control for other more specific markers of sudden death risk, such as parental history of sudden death or resting heart rate, did not modify the results (data not shown). Ultimately, when iliac circumference was used as a surrogate of abdominal (visceral) fat instead of SADx, similar results were observed, although with a lesser magnitude: the multivariate adjusted sudden death risk of the fifth over the first quintile of iliac circumference (95% CI) was 2.0 (1.0 to 3.9; data not shown).

Our data also indicate that SADx is specifically associated with an increased risk of sudden death. This is supported by the fact that a larger SADx was associated with an increased risk of sudden death but not of fatal MI in normal-weight (BMI <25 kg/m²) or overweight (BMI ≥25 kg/m²) men. This confirms that sudden death and fatal MI do not entirely share the same set of risk factors and thus the same underlying causes. Although coronary atherosclerosis is a major common cause of these types of deaths, other mechanisms might lead specifically to sudden death rather than fatal MI, as has been shown in the Paris Prospective Study I for parental history of sudden death, resting heart rate, and circulating free fatty acid levels. In that study, men with larger SADx but normal BMI (<25 kg/m²), who represented 4% of the cohort, were at particularly increased risk of sudden death. Among men with normal BMI (<25 kg/m²), the mean level of established cardiovascular risk factors increased moderately with the level of SADx; however, diabetes was substantially more prevalent in men in the highest tertile of SADx than in men in the lower tertiles (8.4% versus 2.7% and 2.4%, respectively; data not shown). The higher prevalence of

**TABLE 3. Multivariate Adjusted RRs and 95% CIs of Sudden Death and Fatal MI According to Joint Level of BMI and SAD: Paris Prospective Study I**

<table>
<thead>
<tr>
<th>Levels of BMI (kg/m²) and SAD (cm)</th>
<th>n</th>
<th>No. of Cases</th>
<th>Adjusted RR* (95% CI)</th>
<th>No. of Cases</th>
<th>Adjusted RR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &lt;25 and SAD x≥20</td>
<td>1782</td>
<td>14</td>
<td>1</td>
<td>37</td>
<td>1</td>
</tr>
<tr>
<td>and 21≤SAD≤23</td>
<td>754</td>
<td>10</td>
<td>1.4 (0.6–3.2)</td>
<td>25</td>
<td>1.3 (0.8–2.1)</td>
</tr>
<tr>
<td>and SAD &gt;23</td>
<td>311</td>
<td>10</td>
<td>3.0 (1.3–6.9)</td>
<td>8</td>
<td>1.0 (0.5–2.1)</td>
</tr>
<tr>
<td>BMI ≥25 and SAD ≤20</td>
<td>873</td>
<td>13</td>
<td>1.6 (0.7–3.5)</td>
<td>16</td>
<td>0.9 (0.5–1.7)</td>
</tr>
<tr>
<td>and 21≤SAD≤23</td>
<td>1111</td>
<td>15</td>
<td>1.3 (0.6–2.8)</td>
<td>26</td>
<td>0.9 (0.5–1.6)</td>
</tr>
<tr>
<td>and SAD &gt;23</td>
<td>2237</td>
<td>56</td>
<td>1.9 (1.0–3.9)</td>
<td>79</td>
<td>1.3 (0.8–2.1)</td>
</tr>
</tbody>
</table>

* Cox proportional hazards model adjusted for age, trunk subcutaneous fat, sagittal thoracic diameter, mean 5-year number of cigarettes smoked per day, systolic blood pressure, diabetes, and plasma level of cholesterol measured at first examination; trunk subcutaneous fat is the sum of axillary, subumbilical, and subscapular skinfold thicknesses.
diabetes in these men with high SADx and normal BMI is in line with the hypothesis that visceral fat is associated with metabolic consequences that might trigger sudden death, as stated below. Moreover, the present data suggest that in these men with normal BMI, who are usually considered at low cardiovascular risk, the estimation of visceral fat may help to identify subjects who are at substantially increased risk of sudden death. In contrast, men with higher BMI (≥25 kg/m²) are already considered at increased cardiovascular risk, and although we observed that at such BMI levels, higher SADx also conferred an increased risk of sudden death, the clinical additive value of visceral fat estimate appears more limited.

Mechanisms
An increased level of abdominal fat has systemic metabolic consequences that might trigger sudden death. Visceral fat is a major source of release of circulating fatty acids in the portal circulation,16 and their proarrhythmic properties and association with sudden death have been reported in experimental17 and observational studies,18 respectively. Recent data also suggest that a chronic increased release of free fatty acids might facilitate the development of type 2 diabetes by increasing insulin resistance and by deteriorating the insulin-secretion ability of islet B cells.18 This is of critical importance, because type 2 diabetes recently has been proposed as a risk factor for sudden death in asymptomatic men.19 Ultimately, the release of free fatty acids is largely mediated by adrenergic stimulation, a state that is thought to be a major “cause” of sudden death. Levels of resting heart rate and blood pressure are highly correlated with adrenergic tone, and in the present study, men in the top tertile of the SADx distribution had the highest mean level of resting heart rate and systolic and diastolic blood pressure.

Study Limitations
The assessment of SAD in the Paris Prospective Study I study was unusual. Recent studies measured the SAD at the level of the iliac crests and with subjects in a supine position, contrary to the present study. However, in the Paris Prospective Study I, SAD was measured between 1967 and 1972, before the existence of more common measurement methods of SAD.8,11 Because SAD was assessed below the xiphoid level in the present study, it might also reflect to some extent the thoracic diameter. However, the results showed that SADx remained highly predictive of sudden death after adjustment for sagittal thoracic diameter. To investigate this point further, we repeated previous analyses after adjustment for estimates of baseline pulmonary function (forced expiratory volume in 1 second and vital capacity) and found similar results. Taken together, the possible residual confounding effect of the thoracic diameter seems rather small. In most epidemiological studies of abdominal obesity, waist-to-hip ratio circumference has been used, but waist and hip circumferences were not measured in the Paris Prospective Study I. However, the waist-to-hip ratio is difficult to interpret biologically, and SAD has repeatedly been shown to be a better predictor of intra-abdominal fat than any other anthropometric measurement.9,11 Computed tomography or MRI offers a more precise quantification of intra-abdominal fat than anthropometric markers but are rarely available in routine practice. SADx and BMI measurements were measured once at baseline, which was, on average, more than 10 years before outcome (sudden death and fatal MI). It is highly likely that their respective value has varied with this delay, but the effect of their variation on risk of sudden death and fatal MI could not be assessed.

In conclusion, this large prospective study of asymptomatic French middle-aged men suggests that intra-abdominal fat localization as estimated by the SAD is a better risk marker of sudden death than the overall amount of fat as widely estimated by the BMI.

References
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