Body Mass Index and Thromboembolic Stroke in Nonsmoking Men in Older Middle Age

The Honolulu Heart Program

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Background and Purpose While evidence suggests that obesity has an independent relation to coronary artery disease, similar findings for stroke have not been established. The purpose of this study was to examine the relation between body mass index and the risk of thromboembolic stroke independently of other risk factors.

Methods Since 1965, the Honolulu Heart Program has followed a cohort of men in a prospective study of cardiovascular disease. This article examines the relationship between the baseline measurement of body mass index and the risk of thromboembolic stroke in 1163 nonsmoking men in older middle age (55 to 68 years). Men who had an elevated risk of stroke due to hypertension, diabetes, and other risk factors were excluded from the analysis.

Results After 22 years of follow-up, the rate of stroke increased significantly with increasing levels of body mass index \( (P < .01) \). In the bottom tertile of the body mass index, the rate of thromboembolic stroke was 28.7 per 1000 (11/383). In the middle tertile, the rate was increased by 40% to 40.7 per 1000 (16/393), and in the top tertile, the rate of thromboembolic stroke was 55.4 per 1000 (21/387), a twofold excess compared with the bottom tertile. After adjustment for age and the residual effects of confounding risk factors, including systolic blood pressure and serum glucose, the estimated relative risk of stroke for the average body mass index in the top tertile \( (26.6 \text{ kg/m}^2) \) compared with that in the bottom tertile \( (20.3 \text{ kg/m}^2) \) was 2.1 (95% confidence interval, 1.1 to 4.1). These findings were not affected by coronary events that occurred in the course of follow-up, nor did they appear to be influenced by deaths from other causes.

Conclusions We conclude that elevated body mass is associated with an increased risk of thromboembolic stroke in nonsmoking men in older middle age who are free of commonly observed conditions related to cardiovascular disease.

Key Words: • body mass index • cerebrovascular disorders • risk factors • thrombosis

Obesity has an important role in atherogenesis, although its position in the causal pathway to overt cardiovascular disease is unknown. Obesity is directly related to hypertension, hyperlipidemia, glucose intolerance, and hyperinsulinemia. Because these relations, however, it has been difficult to demonstrate that obesity has an independent effect on specific forms of cardiovascular disease. Although evidence suggests that obesity is independently related to coronary artery disease, similar findings for stroke have not been established.

In this study, the association between body mass and the risk of thromboembolic stroke was examined with control for hypertension, diabetes, and other risk factors for stroke. Findings are presented based on 22 years of follow-up of the cohort of older middle-aged men originally enrolled in the Honolulu Heart Program.

Subjects and Methods

From 1965 to 1968, the Honolulu Heart Program began following a cohort of Japanese-American men living on the island of Oahu, Hawaii, for the development of coronary artery disease and stroke. At the time of study enrollment, subjects were aged 45 to 68 years. Each participant was given a baseline physical examination including a complete cardiovascular evaluation. Initial screening included documentation of cardiac and neurological conditions to identify prevalent cases of coronary artery disease and stroke. Procedures followed were in accordance with institutional guidelines and approved by an institutional review committee. Informed consent was obtained from the study participants.

For this study, analysis was limited to older middle-aged men who were 55 to 68 years old at the time of study inception and free of preexisting and coexisting morbidity due to coronary artery disease and stroke. Subjects with hypertension, diabetes, or left ventricular hypertrophy as determined by electrocardiogram were excluded. Because cigarette smoking is a known confounder of the relationship between body mass and cardiovascular disease, only nonsmoking men were examined in this study. Younger men were not considered for follow-up, since thromboembolic stroke was uncommon in the absence of the exclusion criteria. There were 3382 men aged 55 to 68 years at the time of study enrollment. After the

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Heart studies. Among these diverse samples, low levels of the physical activity index were identified. The current survival status is unknown in only four men.

In this study, subjects were followed for the first occurrence of a fatal or nonfatal thromboembolic stroke. For a definite diagnosis of stroke, symptoms included neurological deficits that occurred suddenly and remained present for at least 2 weeks or until death. Definite stroke could usually be classified as thromboembolic or hemorrhagic on the basis of clinical findings at hospitalization, surgery, through computed tomography, or autopsy. Subjects who had possible strokes or experienced neurological episodes attributed to other conditions such as dysdrasias, neoplastic disease, head injury, surgical accident, meningocerebralitis, fat embolism, epilepsy, or cardiac arrest were not included among the stroke victims.

In this study, body mass was quantified through the use of the body mass index (BMI), a weight-for-height index, defined as weight divided by height squared (kilograms divided by meters squared). Consideration of other confounding information or effect modifiers included age, systolic blood pressure, and levels of physical activity, serum glucose, serum uric acid, hematocrit, and total cholesterol. As one of the exclusion criteria, hypertension was defined as present when a subject was receiving antihypertensive medication, a systolic blood pressure was 160 mm Hg or higher, or a diastolic blood pressure was 95 mm Hg or higher. Serum glucose level was measured in a nonfasting state 1 hour after a 50-g load. Because fasting glucose levels were not observed at study entry, participants were classified as diabetic on the basis of medical history, either as diagnosed by physician or based on the reported use of insulin or administration of oral hypoglycemic therapy. Further description of the above characteristics is provided elsewhere. 

Physical activity was measured by use of an index to quantify overall metabolic output that occurred during a typical 24-hour period. The physical activity index was derived by summing the average number of hours per day spent in five different activity levels (basal, sedentary, slight, moderate, and heavy) after each was multiplied by a weighting factor based on the level of exertion required to undertake the activity. High levels of the physical activity index indicated active lifestyles, and low levels indicated inactive lifestyles. In addition to the Honolulu Heart Program, the physical activity index has been used in the Framingham and Puerto Rico heart studies. Among these diverse samples, low levels of the physical activity index have been shown to be related to an increased risk of coronary artery disease and stroke. Refer-
Table 1. Incidence of Thromboembolic Stroke in 22 Years of Follow-up by Tertile of Body Mass Index in Nonsmoking Men Aged 55 to 68 Years

| Tertile of BMI | Range in BMI, kg/m² | Average Age (SD) | Unadjusted* (No. Events/Subjects or Risk) | Adjusted for Age*
<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Bottom</td>
<td>15.33-22.31</td>
<td>60.5 (3.6)</td>
<td>28.7 (11/383)</td>
<td>27.2</td>
</tr>
<tr>
<td>Middle</td>
<td>22.32-24.71</td>
<td>59.5 (3.5)</td>
<td>40.7 (16/393)</td>
<td>42.2</td>
</tr>
<tr>
<td>Top</td>
<td>24.75-35.96</td>
<td>59.6 (3.6)</td>
<td>54.3 (21/387)</td>
<td>55.4</td>
</tr>
<tr>
<td>Total</td>
<td>15.33-35.96</td>
<td>59.8 (3.6)</td>
<td>41.3 (48/1163)</td>
<td></td>
</tr>
</tbody>
</table>

BMI indicates body mass index. Follow-up began at the time of study enrollment (1965-1968). All men were free of known coronary artery disease, stroke, hypertension, diabetes, and left ventricular hypertrophy at the time of study enrollment. Subjects being treated for hypertension or diabetes were also excluded.

*Rate of thromboembolic stroke increased significantly with increasing levels of BMI (P<.01).

The cumulative incidence of thromboembolic stroke that was observed during the course of follow-up in the Honolulu Heart Program is shown in the Figure for each tertile of the BMI. Age-adjusted curves are also given. Early into follow-up, for approximately the first 8 years, little or no difference in the incidence of stroke was observed across the tertiles of the BMI, although the incidence of stroke was consistently lower in the bottom tertile at all years of follow-up. Beyond 8 years (more than half the follow-up period), clear differences emerged in the ordering of the incidence curves, including differences in the risk between the middle and top tertiles of the BMI. Men in the top tertile had the highest incidence, followed by men in the middle tertile, with the lowest incidence occurring in those in the bottom tertile. Differences between the curves for the top and bottom tertiles of the BMI were statistically significant (P<.01).

Table 2 describes the relation of BMI to the other cardiovascular risk factors that are considered in this study. As expected, higher BMIs were associated with several suspected risk factors for cardiovascular disease. In Table 2, age-adjusted mean levels for the additional risk factors are given across the three tertiles of the BMI. For systolic blood pressure, there is a consistent increase in blood pressure levels with each increase in tertile of the BMI. Men in the middle and top tertiles had significantly higher levels of systolic blood pressure compared with men in the bottom tertile (P<.001). Although a modest inverse relation was observed between the physical activity index and body mass, men in the bottom tertile were significantly more active than men who were in either the middle (P<.01) or top tertiles (P<.001).

Glucose levels increased with increasing tertile of the BMI, but the increase was not statistically significant because of the baseline exclusion of men with diabetes. In contrast, a significant increase in serum uric acid levels was observed from the bottom to top tertiles of the BMI. Serum uric acid level in the middle and top tertiles was significantly higher than in the bottom tertile (P<.001). Hematocrit levels also increased significantly with each increase in tertile of BMI. When compared with men in the bottom tertile of the BMI, significant elevations in concentrations of total cholesterol were observed in men who were in the middle and top tertiles (P<.001).

To help determine whether the association of BMI with the risk of stroke could be attributed to the association between body mass and the other risk factors in Table 2, Cox regression models were estimated in an attempt to control for any cross-sectional confounding. Table 3 gives the estimated relative risk of stroke comparing the expected risk associated with the average BMI in the top tertile (26.6 kg/m²) with the average BMI in the bottom tertile (20.3 kg/m²), a...
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TABLE 2. Age-Adjusted Mean Levels of Selected Risk Factors by Tertile of Body Mass Index in Nonsmoking Men Aged 55 to 68 Years

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Bottom Tertile</th>
<th>Middle Tertile</th>
<th>Top Tertile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>125.0 (15.1)</td>
<td>129.7* (14.1)</td>
<td>132.4* (14.1)</td>
</tr>
<tr>
<td>Physical activity index</td>
<td>33.5 (37.5)</td>
<td>32.7 (41.4)</td>
<td>32.4 (41.1)</td>
</tr>
<tr>
<td>Serum glucose, mmol/L</td>
<td>8.5 (2.6)</td>
<td>8.6 (2.4)</td>
<td>8.8 (2.7)</td>
</tr>
<tr>
<td>Serum uric acid, μmol/L</td>
<td>315.2 (83.3)</td>
<td>345.0* (77.3)</td>
<td>368.8* (83.3)</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>43.0 (2.7)</td>
<td>44.2* (3.0)</td>
<td>44.5* (2.8)</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.4 (0.9)</td>
<td>5.6* (0.9)</td>
<td>5.7* (0.9)</td>
</tr>
</tbody>
</table>

Risk factors were measured at baseline examination (1965-1968) in the Honolulu Heart Program. All men were free of known coronary artery disease, stroke, hypertension, diabetes, and left ventricular hypertrophy at the time of study enrollment. Subjects being treated for hypertension or diabetes were also excluded. Numbers in parentheses indicate standard deviation.

*Significant difference from the bottom tertile (P<.001).
\*Significant difference from the bottom tertile (P<.01).

Difference of approximately 16 to 17 kg for men of average height in the Honolulu Heart Program. After risk-factor adjustment, a twofold excess of thromboembolic stroke persisted for the average BMI in the top tertile compared with the bottom tertile. The observed association between an increase in the risk of stroke with an increase in levels of BMI was statistically significant (P<.05).

For comparison purposes, similar relative risks are provided for the other risk factors in Table 2. Here, estimated relative risks compare the risk of stroke for the average value in the top tertile of the risk factor with the average value in the bottom tertile of the risk factor.

After adjustment for age, the association between systolic blood pressure and thromboembolic stroke is slightly weaker than the association for BMI. This latter finding is largely due to the exclusion of men who were enrolled in the Honolulu Heart Program with hypertension. For men with an average systolic blood pressure in the top tertile of that risk factor (145.4 mm Hg), the risk of stroke is double (P<.05) compared with that of men with average systolic blood pressure in the bottom tertile.

TABLE 3. Relative Risk of Thromboembolic Stroke in Nonsmoking Men Aged 55 to 68 Years Comparing the Risk of Stroke Associated With the Average Value of a Risk Factor Within the Top Tertile of That Risk Factor to the Average Value Within the Bottom Tertile

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Average Risk-Factor Level*</th>
<th>Adjusted for Age (95% Cl)</th>
<th>Adjusted for Risk Factor† (95% Cl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index, kg/m²</td>
<td>20.3 (Bottom) 26.6 (Top)</td>
<td>2.6§ (1.4-4.7)</td>
<td>2.1† (1.1-4.1)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>113.0 (Bottom) 145.4 (Top)</td>
<td>2.2‡ (1.2-4.2)</td>
<td>2.0 (1.0-4.0)</td>
</tr>
<tr>
<td>Physical activity index</td>
<td>28.9 (Bottom) 37.4 (Top)</td>
<td>0.4‡ (0.2-0.8)</td>
<td>0.4‡ (0.2-0.9)</td>
</tr>
<tr>
<td>Serum glucose, mmol/L</td>
<td>6.1 (Bottom) 11.5 (Top)</td>
<td>1.1 (0.6-2.0)</td>
<td>0.9 (0.5-1.6)</td>
</tr>
<tr>
<td>Serum uric acid, μmol/L</td>
<td>255.8 (Bottom) 434.2 (Top)</td>
<td>1.8 (1.0-3.3)</td>
<td>1.3 (0.7-2.6)</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>40.7 (Bottom) 47.3 (Top)</td>
<td>0.9 (0.5-1.7)</td>
<td>0.7 (0.4-1.5)</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>4.7 (Bottom) 6.6 (Top)</td>
<td>1.8 (1.0-3.3)</td>
<td>1.6 (0.9-2.8)</td>
</tr>
</tbody>
</table>

Cl indicates confidence intervals. All men were free of known coronary artery disease, stroke, hypertension, diabetes, and left ventricular hypertrophy at the time of study enrollment. Subjects being treated for hypertension or diabetes were also excluded.

*Average levels of a risk factor are calculated within the bottom and top tertiles of that risk factor.
†Adjusted for age and the other risk factors.
‡Significant effect on thromboembolic stroke (P<.05).
§Significant effect on thromboembolic stroke (P<.01).
tertile (113 mm Hg). After adjustment for the other risk factors, the excess risk is no longer statistically significant (P=0.06).

The effect of physical activity on the risk of stroke was similar to the effect of body mass in that significant effects persisted after control of the other risk factors. Magnitudes of association were also similar to those of the BMI, although the associations were reversed. Here, the average physical activity index in the top tertile of that index is associated with half the risk of thromboembolic stroke compared with the average physical activity index in the bottom tertile (P<0.05).

There was no association between glucose and thromboembolic stroke. Once again, this is due to the exclusion in this study of men who entered the Honolulu Heart Program with diabetes. Elevations in serum uric acid and total cholesterol levels also appeared to place men at an increased risk of stroke, and hematocrit seemed to be protective. None of these latter associations were statistically significant.

Discussion

Although the incidence of stroke has declined in recent decades,22-23 stroke remains a leading cause of death and disability in the United States and in other developed countries.24 Unfortunately, as the risk of stroke rises with advancing age and as older populations expand,22,23,25 the impact of stroke on healthcare resources is likely to increase. Early prevention is especially important because stroke often occurs without warning, and for nonfatal events, long periods of convalescence and permanent dysfunction can follow.

For elderly populations, in whom prevention, delay, and minimization of the consequences of stroke are in greatest need, it becomes critical to identify risk factors that can have a major role in altering the risk of stroke. While hypertension is one such risk factor,26,27 obesity may also be related to stroke susceptibility before hypertension has had a chance to evolve, although stroke may be a consequence of hypertension that develops later in life. In the Honolulu Heart Program, elevated BMI was associated with an increased risk of thromboembolic stroke in men without hypertension at the time body mass was measured. Whether hypertension developed in the course of follow-up could not be determined.

BMI was also associated with thromboembolic stroke in older middle-aged men who appeared to be clinically healthy with regard to other conditions related to cardiovascular disease (ie, nonsmoking men without diabetes, left ventricular hypertrophy, or known coronary artery disease and stroke). Even after adjustment for the residual effects of confounding risk factors, including systolic blood pressure and serum glucose levels, the association between body mass and thromboembolic stroke remained.

Possible relations of body mass to stroke have also been put forth by other investigators, but the strength of the association has been equivocal.9,12,26-37 In the Framingham study,32 there is an indication that relative weight is inversely associated with the incidence of stroke in the elderly (ages 65 to 74). In light of such findings, the generalizability of data collected in the unique and uncontrolled setting in Hawaii cannot be ensured, although data reported here are consistent with trends or suggestions that have been observed in other population-based samples with regard to total mortality9-10 and to stroke.28-31 The uniqueness of this study is based on the length of follow-up and the ability to make important baseline exclusions so that body mass could exhibit an independent effect on stroke that would not be compromised by important confounders. Attempts to explain the variability among earlier reports can be based partly on differences in study methods, limited follow-up, or large differences in the age range of the samples being studied.12,23-36 Cigarette smoking, however, is the most important factor that is most likely to contribute to the differences between findings in earlier reports and those given here. Cigarette smoking has a strong inverse association with weight, and it is well established that lean cigarette smokers experience an excess in mortality.8-10 In a study of college alumni, lean smokers had a higher mortality rate than the heaviest of former smokers and nonsmokers.10 In the Honolulu Heart Program, the 22-year incidence of thromboembolic stroke in smoking men in the bottom tertile of the BMI was 76.1 per 1000 (36/473) compared with 54.3 per 1000 (21/387) in nonsmoking men in the top tertile. After adjustment for systolic blood pressure, the excess of stroke in the leaner smoking men was statistically significant (P<0.05). In conjunction with the experiences of others,8-10 such data suggest that body mass represents a different phenomenon in smokers and nonsmokers in terms of the role that body mass has in the development of cardiovascular disease. As a result, to study the effects of body mass on cardiovascular disease, it seems reasonable to consider smokers and nonsmokers separately.

In the Honolulu Heart Program and elsewhere, cigarette smoking, hypertension, and diabetes are potent risk factors for stroke.26,27,38-41 In Hawaii, men with any of these conditions were already at such a high risk of stroke that elevations in body mass could not affect the risk of stroke further. Without excluding subjects with these important risk factors, an effect of body mass on stroke was hidden. BMI had no effect on stroke in those who smoked cigarettes. Although not statistically significant, hypertensive men who were lean had a slightly higher incidence of stroke. Such findings may provide evidence for a difference in the pathogenesis of hypertension for subjects who become obese later in life compared with those who remain lean.42

Among men who were younger than 55, thromboembolic events were too uncommon to provide an opportunity to assess the effect of body mass on stroke in a younger age group.11 Nevertheless, body mass in those who were younger could still have an important association with strokes that occur in later life. The benefits of low body mass that were observed in the older middle-aged men could have been related to lean body mass or positive health behavior that was present during younger years. Unfortunately, information on maintaining or changing body mass over time could not be examined in this study. Establishing a baseline at a later examination so that changes in body mass could be observed results in a loss of statistical power because of a reduction in follow-up and a reduced number of stroke events. Even when follow-up is reestablished at 6 years into follow-up, men in the top tertile of BMI continued to be at a twofold risk of stroke (although no
longer statistically significant) compared with men in the bottom tertile. As before, men in the middle tertile had an intermediate risk of stroke.

Although the association between body mass and the risk of thromboembolic stroke appears to be independent of other risk factors for stroke, this can only be said of those risk factors controlled for in this study and of those risk factors that were related cross-sectionally to body mass. Data on levels of high- and low-density lipoprotein cholesterol were not available in Hawaii at the time of study entry, and triglyceride levels (measured in a nonfasting state) had no effect on the reported associations. Alcohol intake was also unrelated to thromboembolic stroke and was not an important confounder in this study. Other suspected confounders or effect modifiers such as forced expiratory volume (possibly as a surrogate for smoking history among men who had quit smoking before entry into the Honolulu Heart Program) provided no additional insight.

It is possible that elevated body mass is associated with stroke through induced changes in important risk factors over time, most notably increases in blood pressure or acute phases of hypertension that might occur with greater frequency, longer duration, or less tolerance during the aging process. Compared with body mass of leaner individuals, excesses in body mass might also be associated with a faster atherosclerotic progression due to alterations in glucose and lipid metabolism, fibrinolytic activity, clotting factors, blood viscosity, and other metabolic derangements.5,44,45

As a consequence of this progression, the possibility also exists that body mass is associated with stroke through the development of interim coronary events. Data on atrial fibrillation, congestive heart failure, and valvular disease (not among the exclusion criteria) were not available throughout the course of follow-up in the Honolulu Heart Program. Excluding subjects with any of these conditions at the time of study enrollment, however, had no effect on the findings reported here. In an attempt to account for time-dependent correlates of risk factors related to intervening cardiovascular disease, stroke events that were preceded by coronary artery disease were censored at the time the coronary event was first observed. Because most of these events occurred in individuals who were already excluded from follow-up, the relation between body mass and stroke was unaffected.

Assessment of the absolute effect of body mass on stroke or of changes in body mass over time continues to be difficult, since it is not known whether current body mass status could be the result of illness or intentional adherence to improved or learned health behavior. Lean individuals may have had a lower risk of stroke because of occult disease that might have promoted early mortality from noncardiovascular outcomes such as cancer.9 In the Honolulu Heart Program, however, only 11 cancer deaths were observed in the first 5 years of follow-up in the sample of 1163 men, with equal mortality in the top and bottom tertiles of the BMI. When follow-up is extended to 22 years, death from cancer was more frequent in the top tertile of the BMI. The exclusion of men who died within the first 2 years of follow-up or men who experienced a loss in weight of 10% or more within the first 2 years had no effect on the association between body mass and thromboembolic stroke that is reported here.

Although a specific explanation for the association between body mass and thromboembolic stroke cannot be identified in this study, control of body mass may have a role among preventive measures to reduce the risk of stroke. While this needs further study, a multifactorial approach to health promotion might include attention to the level of body mass as it affects risk factors that are known to contribute to stroke and cardiovascular disease in general. These factors include hypertension, glucose intolerance and insulin resistance, and blood lipid abnormalities, among others. Although our results indicate that body mass has an independent effect on thromboembolic stroke, a focus on independent mechanisms seems less important than a demonstration that control of body mass can affect the risk of stroke, even if it is through cross-sectional or evolutionary effects on concomitant risk factors.

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