Role of Body Mass Index History in Predicting Risk of the Development of Hypertension in Japanese Individuals

Toranomon Hospital Health Management Center Study 18 (TOPICS 18)

Yoriko Heianza, Satoru Kodama, Yasuji Arase, Shiun Dong Hsieh, Sakiko Yoshizawa, Hiroshi Tsuji, Kazumi Saito, Shiro Tanaka, Shigeko Hara, Hirohito Sone

Abstract—It has not been clarified whether overall adiposity in early adulthood or at the lifetime maximum weight would confer a residual risk of hypertension after considering the risk associated with current adiposity. Studied were 6121 Japanese without hypertension. The risk of developing hypertension 4 years after a baseline examination was investigated using body mass index in the early 20s, at the lifetime maximum, or at the baseline examination. An elevated body mass index at baseline or at the maximum rather than in the early 20s was strongly associated with future hypertension. Compared with individuals with low body mass index both at baseline and in the early 20s, those with an elevated body mass index at the baseline alone had an odds ratio of 1.89 (95% confidence interval, 1.58–2.27) and those with an elevated body mass index both at baseline and in the early 20s had the highest odds ratio of 2.26 (1.76–2.89). Individuals with an elevated body mass index both at baseline and at the maximum had a 2.26-fold (1.87–2.72) increased risk of hypertension compared with those without the 2 factors. An elevated body mass index at the baseline examination weakened the favorable influence of a low body mass index in early adulthood on developing hypertension. Adding information on body mass index in early adulthood or at the maximum in addition to that at the baseline examination contributed to differentiating the risk of hypertension among Japanese, particularly among those with an elevated overall adiposity at present. (Hypertension. 2014;64:247-252.) • Online Data Supplement

Key Words: blood pressure • body mass index • obesity • risk

It is generally accepted that being overweight or obese increases the risk of the development of hypertension.1–10 Also, it has been noted that weight gain increases the risk of hypertension,1,2,7,11–17 whereas weight loss contributes to a reduction in risk.18–20 However, it has not been fully clarified whether overall adiposity in early adulthood such as around age 20 years or at the lifetime maximum would irreversibly confer a residual risk of hypertension after considering the risk associated with current adiposity. In a study of a British birth cohort that analyzed the association of body mass index (BMI) across childhood and adulthood with blood pressure in midadulthood, it was suggested that high BMI and BMI gain at any life stage, especially when recent, were associated with elevated blood pressure levels.21 A strong positive association between being overweight in early adulthood was reported to be associated with elevated midlife blood pressure levels.22 To date, only a few large longitudinal studies investigated the role of BMI in early adulthood in the prediction of future hypertension,7,8,23 and these studies did not clarify the effect of the lifetime maximum BMI (BMImax) on the development of hypertension. In addition, although a few studies suggested that the association between weight gain and incident hypertension tended to be greater in younger than in older participants,7,24 it remains undetermined whether histories of elevated BMI in early adulthood such as the 20s (BMIage20y) or the BMImax would be positively associated with an increased risk of hypertension even in later life.

Therefore, we aimed to investigate the role of BMI histories, including BMI in the early 20s, at its maximum and at the time of baseline examination, as predictors of future hypertension in Japanese individuals.

Study Participants
The Toranomon Hospital Health Management Center Study included a cohort consisting mainly of apparently healthy government employees who underwent annual examinations for health screening in Tokyo, Japan. This study included 8142 individuals who underwent a baseline examination during the period from 2003 to 2007 and provided data on blood pressure measurements, a self-reported history of medical treatment for hypertension, or the use of antihypertensive

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medication at a re-examination 4 years after the baseline examination at the Toranomon Hospital Health Management Center. Among the 8142 individuals, we had available data on self-reported weight in early adulthood (20s) and on the maximum weight by the day before the examination for 7855 participants. We excluded individuals with hypertension at the baseline examination (indicated by systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg; a self-reported history of medical treatment for hypertension, or the use of antihypertensive medication [n=1691]) or with missing data on baseline characteristics (n=45). Participants with contradictory data on self-reported weight (weight in their early 20s > lifetime maximum weight) were also excluded (n=6). Subsequently, 6121 individuals (4387 men and 1734 women) aged 22 to 82 years were included in the current analysis. Informed consent was obtained from the participants. The study protocol followed the Japanese Government’s Ethical Guidelines Regarding Epidemiological Studies in accordance with the Declaration of Helsinki and was reviewed and approved by the Institutional Review Board at Toranomon Hospital.

Assessment of BMIs
Weight in the early 20s and the maximum weight were obtained by a standard questionnaire. As to the maximum weight, participants were asked to report their maximum known weight at any point in time prior to the baseline examination. After assessment of weight history, anthropometric measurements such as those of body weight and height with the subject in light clothes without shoes were done at the time of the baseline examination by trained staff. If the participant’s weight at the time of the baseline examination was greater than the self-reported maximum weight, lifetime maximum weight was indicated by the weight at the time of the baseline examination. Among the 6121 individuals enrolled in this study, 403 had achieved their maximum weight at the time of the baseline examination, BMI at the baseline examination (BMIbaseline), BMIage20y, and BMImax were calculated as weight in kilograms divided by the square of height in meters (kg/m²). For calculation of BMIage20y and BMImax, we used the height measured at the baseline examination.

Clinical and Other Measurements
After a brief period of rest, systolic blood pressure and diastolic blood pressure were measured in either arm using a sphygmomanometer (BP-203RVII [OMRON Corporation] in 2003–2008 or BP-203RVIII [OMRON Corporation] in 2008–2011) using an appropriate size cuff with the participant in a sitting position. Blood pressure was measured once in most participants, but up to 3 measurements at 1- to 2-minute intervals were made in participants who had hypertensive episodes indicated by clinical hypertension. The lowest reading was used in the analysis that assessed the incidence of hypertension. The protocol was fundamentally the same throughout the observational period. Blood samples were collected after an overnight fast (12 hours) and measurements made using an automatic clinical chemistry analyzer (Hitachi, LABOSPECT 008, Tokyo, Japan). Smoking habits and parental history of hypertension were assessed by a questionnaire, as was the self-reported history of hypertension or diabetes mellitus.

Statistical Analysis
We performed analysis among all participants on the basis of our finding that there was no significant effect of interaction between BMI (BMIbaseline, BMIage20y, or BMImax) and sex on the development of hypertension. To explore potential nonlinear relationships, we estimated the spline function and 95% CI (confidence interval) of the association between the 3 BMIs and risk of hypertension at 4 years using multivariate-adjusted generalized additive models including age, sex, parental hypertension, smoking habit, high-density lipoprotein cholesterol concentrations, triglyceride concentrations, and type 2 diabetes mellitus. In this analysis, the degree of freedom was determined by the generalized cross-validation method. Based on previous findings and differences in variables at the baseline examination between individuals who developed or did not develop hypertension observed in this study, we included metabolic and demographic variables in a multivariate-adjusted model. Logistic regression analysis was performed to calculate the odds ratio (OR) and 95% CI for the development of hypertension by a 1-SD (1-standard deviation) increment in BMIbaseline, BMIage20y, or BMImax as a continuous variable in the regression model. The effect of interaction between BMI history (BMIage20y or BMImax) and BMIbaseline on the development of hypertension was examined using an interaction term in the logistic regression model. We then investigated the combined effect of BMIbaseline and BMIage20y or BMIbaseline and BMImax by categorizing participants into 4 groups. We used cutoff points of 23.0 kg/m² for BMIbaseline or BMIage20y or 25.0 kg/m² for BMImax to conduct meaningful analysis considering the number of cases for each category. To investigate the association of BMIs and hypertension in younger and older groups, we first examined whether there was a significant interaction effect between BMIs and age on the development of hypertension. We then categorized individuals according to tertiles of age at the time of the baseline examination and evaluated the OR according to a 1-U (1 kg/m²) increment in BMIbaseline, BMIage20y, or BMImax. Analysis was performed with IBM SPSS Statistics ver. 19 or SAS ver. 9.2. Statistical significance was considered for P<0.05.

Results
Mean (SD) age at the time of the baseline examination was 49.9 (9.2) years, and mean (SD) BMIbaseline, BMIage20y, and BMImax were 22.7 (2.9) kg/m², 20.7 (2.2) kg/m², and 24.3 (2.9) kg/m², respectively (Table S1 in the online-only Data Supplement). Results of Pearson correlation showed that BMIbaseline was positively associated with BMIage20y (r=0.586, P<0.001) and BMImax (r=0.885, P<0.001). During the 4-year follow-up, we documented 751 incident cases of hypertension. Even within low ranges of BMIbaseline (Figure A) and BMImax (Figure C), there was an increasingly higher risk of hypertension when those values were elevated. The risk of hypertension was not increased within low ranges of BMIage20y (Figure B). In the multivariate model, the ORs for the development of hypertension for a 1-SD increment in BMIbaseline and BMImax were similarly higher (OR, 1.56 [95% CI, 1.43–1.70] and OR, 1.52 [95% CI, 1.40–1.64], respectively) than for a 1-SD increment in BMIage20y (OR, 1.24 [95% CI, 1.15–1.34]) (Table 1). When we calculated multivariate-adjusted ORs (95% CIs) for hypertension indicated by only medical treatment of hypertension or use of antihypertensive agents (incident cases 284/total 6121 individuals), a 1-SD increment in BMIbaseline, BMIage20y, or BMImax was associated with an OR (95% CI) of 1.46 (1.28–1.66), 1.26 (1.12–1.42), or 1.43 (1.26–1.62), respectively.

We observed a significant effect of interaction between BMIbaseline and BMIage20y (P=0.046) and between BMIbaseline by BMImax (P=0.001) on the development of hypertension. The results of the separate or combined effect of elevated values of BMIbaseline with BMIage20y or elevated BMImax on the development of hypertension are shown in Table 2. For the combination of BMIbaseline and BMIage20y (Table 2), compared with group I (both low BMIbaseline [<23.0 kg/m²] and low BMIage20y [<23.0 kg/m²]), group II (low BMIbaseline [<23.0 kg/m²] and elevated BMIage20y [≥23.0 kg/m²]) did not have an increased risk of the development of hypertension (OR, 1.47 [95% CI, 0.83–2.59]; P=0.188); however, there were only a few participants in group II. We found that group III (elevated BMIbaseline [≥23.0 kg/m²] and low BMIage20y [<23.0 kg/m²]) had an OR of 1.89 (95% CI, 1.58–2.27) for
hypertension and that group IV, which included individuals with both elevated BMIbaseline and elevated BMIage20y, had the highest OR of 2.26 (95% CI, 1.76–2.89). When we calculated ORs for hypertension among only those with an elevated BMIbaseline (groups III and IV), the multivariate-adjusted OR was 1.17 (95% CI, 0.92–1.47) for those who also had BMIage20y ≥23.0 kg/m² and 1.60 (95% CI, 1.11–2.31; P=0.013) for those who also had BMIage20y ≥25.0 kg/m². In examining results of the combination of BMIbaseline and BMImax (Table 2), we observed that most participants with an elevated BMIbaseline also had an elevated BMImax. Compared with group I (both low BMIbaseline [<23.0 kg/m²] and BMImax [<25.0 kg/m²]), group II (low BMIbaseline [<23.0 kg/m²] and elevated BMImax [≥25.0 kg/m²]) was not associated with a significantly increased OR for the development of hypertension (OR, 1.29 [0.86–1.94]). On the other hand, group IV (both elevated BMIbaseline and elevated BMImax) was associated with an OR of 2.26 (1.87–2.72) for hypertension. Among individuals with an elevated BMIbaseline (group III and group IV), an elevated BMImax (≥25.0 kg/m²) was associated with a 1.61 (1.25–2.07)-fold increased risk of the development of hypertension. When we reanalyzed data after excluding the 403 individuals who achieved their maximum weight at the time of baseline examination, we observed fundamentally the same results as those in Tables 1 and 2 (data not shown).

In the results of our interaction analysis of BMI history and age, a significant interaction effect was observed between BMIage20y and age (P=0.003) on the development of hypertension, but not between BMIbaseline and age (P=0.38) or between BMImax and age (P=0.17). When we investigated an effect of the 3 BMIs on the risk of hypertension across tertile categories of ages, BMIbaseline, BMIage20y, or BMImax was significantly predictive of the development of hypertension among individuals in the lowest and middle tertile categories of age (<54 years). However, BMIage20y was not significantly predictive of the development of hypertension among individuals in the highest tertile of age (≥54 years) (Table S2). BMIbaseline and BMImax were similarly predictive even in older adults.

Discussion

Our study of Japanese individuals demonstrated that elevated values for BMIbaseline or lifetime BMImax, rather than elevated BMIage20y, even within low to normal ranges were positively associated with the development of future hypertension. An elevated BMIbaseline weakened the favorable effect of a low BMIage20y on the development of hypertension. Individuals with elevated BMIbaseline and BMIage20y and those with elevated BMIbaseline and BMImax had almost double the risk of hypertension as compared with individuals without those 2 factors. To date, there has been little clarification of the risk of hypertension based on comparisons and combinations of these BMI histories (BMIs at the time of baseline examination, at maximum, and in early adulthood), especially in Asian individuals who were not predominantly obese. Our study results suggested that overall adiposity in early adulthood, such as around 20 years of age, or at the lifetime maximum might confer a residual risk of hypertension after

Figure. Spline curves and 95% confidence interval for the association between BMIbaseline (A), BMIage20y (B), and BMImax (C) and risk of the development of hypertension by 4 years after the baseline examination. BMIage20y indicates body mass index in early adulthood such as the 20s; BMIbaseline, body mass index at the baseline examination; and BMImax, maximum body mass index.
considering BMIbaseline, but this effect might be particularly relevant among participants with an elevated current BMI.

Our study is unique in assessing the role of the lifetime BMImax in the development of future hypertension, and our results suggested that adding information on BMImax in addition to BMIbaseline might contribute to differentiating the risk of developing hypertension. In a cross-sectional study, it was reported that hypertensive individuals had higher BMIage20y and BMImax in adulthood compared with nonhypertensive individuals.29 We found that BMIbaseline and BMIage20y, but not BMImax, were similarly predictive of future hypertension in a multivariate-adjusted model and that BMIbaseline and BMImax were highly correlated. Although we did not have data on the age at which each participant experienced the maximum weight, participants in which the 2 factors overlapped might be obese individuals with a prolonged lifetime BMImax extending until the baseline examination.

Some cross-sectional studies showed a positive association of weight gain since early adulthood and the presence of elevated blood pressure in later life.30–34 To date, few prospective studies have shown a positive association of an elevated BMI in early adulthood and the development of future hypertension,7,8,23 although these studies did not provide data on the role of the lifetime BMImax in the development of hypertension. In a prospective study,4 white men who were overweight or obese (BMI ≥25.0) at age 25 years and remained overweight or who were obese at age 45 years had a doubled risk of hypertension compared with those having a normal BMI (BMI <25.0) at both age 25 and 45 years in that study.3 Importantly, their results in white men and our results also suggested that only a small number of participants who had an elevated BMI in early adulthood had lost weight by the time of the baseline examination, which implies the difficulty of managing adult obesity. However, for those participants with BMI <23.0 at the baseline examination, a history of elevated BMI in early adulthood or elevated BMI at maximum was not associated with a significantly increased risk of the development of hypertension as compared with individuals without either of the 2 factors. In a prospective study,23 an elevated BMI in adolescence (age, 17 years) was a strong determinant of future hypertension

### Table 1. Comparison of a 1-SD Increment in BMImbaseline, BMImage20y, and BMImax in Predicting the Development of Hypertension 4 Years After Baseline

<table>
<thead>
<tr>
<th>Model</th>
<th>Age- and Sex-Adjusted OR (95% CI)</th>
<th>P Value</th>
<th>Multivariate-Adjusted OR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMIbaseline</td>
<td>1.62 (1.50–1.76)</td>
<td>&lt;0.001</td>
<td>1.56 (1.43–1.70)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMImage20y</td>
<td>1.28 (1.18–1.37)</td>
<td>&lt;0.001</td>
<td>1.24 (1.15–1.34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMImax</td>
<td>1.58 (1.46–1.70)</td>
<td>&lt;0.001</td>
<td>1.52 (1.40–1.64)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Multivariate-adjusted model included age, sex, smoking habit (never, former, and current), parental history of hypertension, high-density lipoprotein cholesterol, log-transformed triglycerides, and type 2 diabetes mellitus.

### Table 2. Combination of BMImbaseline With BMImage20y or BMImax in Predicting the Development of Hypertension

<table>
<thead>
<tr>
<th>Baseline Group</th>
<th>Cases/Total n</th>
<th>Age- and Sex-Adjusted OR (95% CI)</th>
<th>P Value</th>
<th>Multivariate-Adjusted OR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combination of BMImbaseline and BMImage20y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group I (BMIbaseline &lt;23.0 and BMImage20y &lt;23.0)</td>
<td>278/3315</td>
<td>1.00 (Reference)</td>
<td>...</td>
<td>1.00 (Reference)</td>
<td>...</td>
</tr>
<tr>
<td>Group II (BMIbaseline &lt;23.0 and BMImage20y ≥23.0)</td>
<td>15/116</td>
<td>1.43 (0.81–2.52)</td>
<td>0.215</td>
<td>1.47 (0.83–2.59)</td>
<td>0.188</td>
</tr>
<tr>
<td>Group III (BMIbaseline ≥23.0 and BMImage20y &lt;23.0)</td>
<td>338/2018</td>
<td>2.11 (1.77–2.51)</td>
<td>&lt;0.001</td>
<td>1.89 (1.58–2.27)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Group IV (BMIbaseline ≥23.0 and BMImage20y ≥23.0)</td>
<td>120/672</td>
<td>2.52 (1.98–3.20)</td>
<td>&lt;0.001</td>
<td>2.26 (1.76–2.89)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Combination of BMImbaseline and BMImax</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (BMIbaseline &lt;23.0 and BMImax &lt;25.0)</td>
<td>262/3166</td>
<td>1.00 (Reference)</td>
<td>...</td>
<td>1.00 (Reference)</td>
<td>...</td>
</tr>
<tr>
<td>Group II (BMIbaseline &lt;23.0 and BMImax ≥25.0)</td>
<td>31/265</td>
<td>1.29 (0.86–1.92)</td>
<td>0.220</td>
<td>1.29 (0.86–1.94)</td>
<td>0.219</td>
</tr>
<tr>
<td>Group III (BMIbaseline ≥23.0 and BMImax &lt;25.0)</td>
<td>91/739</td>
<td>1.50 (1.16–1.94)</td>
<td>0.002</td>
<td>1.40 (1.07–1.81)</td>
<td>0.013</td>
</tr>
<tr>
<td>Group IV (BMIbaseline ≥23.0 and BMImax ≥25.0)</td>
<td>367/1951</td>
<td>2.53 (2.12–3.02)</td>
<td>&lt;0.001</td>
<td>2.26 (1.87–2.72)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Multivariate-adjusted model included age, sex, smoking habit (never, former, and current), parental history of hypertension, high-density lipoprotein cholesterol, log-transformed triglycerides, and type 2 diabetes mellitus.

BMImage20y indicates body mass index in early adulthood such as the 20s; BMImbaseline, body mass index at the baseline examination; BMImax, maximum body mass index; CI, confidence interval; and OR, odds ratio.
independently of BMI in adulthood (age, 30 years); it was also reported that much of the risk of hypertension associated with BMI at age 17 years might be mediated by its prediction of BMI in adulthood more evidently in women than in men.\textsuperscript{23} It was also suggested that the current BMI rather than the past BMI trajectory may be associated with hypertension.\textsuperscript{35,36} Our findings add to the existing literature and provide further evidence that managing current overweight/obesity might weaken the unfavorable effect of BMImax on the increased risk for future hypertension. Furthermore, the residual risk of hypertension associated with elevated BMImax might persist under the influence of an elevated BMIbaseline.

It would be important to consider the influence of age on the association of BMI with hypertension. Among our study participants, high values for BMImax were more strongly predictive of hypertension for younger participants, and BMImax did not confer a significantly increased risk of hypertension in later life. Because we assessed weight in early adulthood by self-report, we cannot deny the possibility of recall bias in the older participants. Nonetheless, BMImax was predictive of an increased risk of hypertension even in later life, and BMImax might not reflect an increased risk of hypertension among elderly Japanese individuals.

Our study investigated the unanswered issue of the association of weight histories (including the lifetime maximum body weight) and future hypertension in a relatively large number of individuals. Several limitations should be considered in this study. Blood pressure data were based on results of measurements at a single visit, and these measurements can vary according to various external factors, so whether these measurements reflected the actual blood pressure levels in each participant is unknown. Information on weight in early adulthood and maximum weight were assessed by a self-reported questionnaire so that data on past body weight might not reflect actual values. This could have led to a misclassification of participants. Although it would be difficult to acquire data on measured lifetime maximum weight in a person over the course of one’s lifetime, further studies should be conducted to confirm our findings with actual measured body weight and height and including other data such as BMI at birth or in childhood. Nonetheless, data in the literature showed that long-term memory of weight was relatively good\textsuperscript{17} and that recalled weight for around the literature showed that long-term memory of weight was relatively good\textsuperscript{17} and that recalled weight for around the literature showed that long-term memory of weight was relatively good\textsuperscript{17} and that recalled weight for around the literature showed that long-term memory of weight was relatively good\textsuperscript{17} and that recalled weight for around the literature showed that long-term memory of weight was relatively good\textsuperscript{17} and that recalled weight for

Perspectives

Elevated values of BMIfinal or the lifetime BMImax, rather than of BMImax or BMIfinal, within normal ranges were strongly associated with the development of hypertension among Japanese individuals. After considering the risk of hypertension associated with BMImax, having a history of elevated BMImax or BMIfinal might further confer a residual risk of hypertension, particularly for individuals with an elevated BMIfinal. Although the risk of hypertension associated with the past weight history is considered to be nonmodifiable, individuals with an elevated BMIfinal, particularly that which is close to the lifetime BMImax, should be offered an appropriate weight management program.

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We sincerely thank the late Professor and Director Kinori Kosaka, MD, PhD, who established the foundation and framework of this project and was always the foremost pillar of spiritual support of the Toranomon Hospital Health Management Center Study project.

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Disclosures

None.

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Online Supplement

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Table S1: Characteristics at the time of the baseline examination of the total study population and individuals who did or did not develop hypertension during the follow-up period

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Total (n=6121)</th>
<th>Incidence of hypertension</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No (n=5370)</td>
<td>Yes (n=751)</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>49.9 ± 9.2</td>
<td>52.9 ± 9.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female sex</td>
<td>1734 (28.3)</td>
<td>156 (20.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Parental history of hypertension</td>
<td>1916 (31.3)</td>
<td>280 (37.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>118 ± 12</td>
<td>127 ± 9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>74 ± 8</td>
<td>81 ± 6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI at the baseline examination, kg/m²</td>
<td>22.7 ± 2.9</td>
<td>23.9 ± 3.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI in early 20s, kg/m²</td>
<td>20.7 ± 2.2</td>
<td>21.2 ± 2.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Maximum BMI, kg/m²</td>
<td>24.3 ± 2.9</td>
<td>25.5 ± 3.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL-cholesterol, mmol/L</td>
<td>1.48 ± 0.35</td>
<td>1.40 ± 0.33</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.03 (0.73, 1.47)</td>
<td>1.21 (0.85, 1.74)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smoking habit</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Never</td>
<td>3659 (59.8)</td>
<td>394 (52.5)</td>
<td>-</td>
</tr>
<tr>
<td>Former</td>
<td>1307 (21.4)</td>
<td>202 (26.9)</td>
<td>-</td>
</tr>
<tr>
<td>Current</td>
<td>1155 (18.9)</td>
<td>155 (20.6)</td>
<td>-</td>
</tr>
<tr>
<td>Type 2 diabetes*</td>
<td>274 (4.5)</td>
<td>53 (7.1)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are mean ± SD, n (%) or median (25th, 75th). *Type 2 diabetes was indicated by fasting glucose concentrations ≥7.0 mmol/L, self-reported clinician-diagnosed diabetes, or HbA1c (NGSP) ≥6.5%. P values were tested by t-test or median test for continuous variables and categorical data were analyzed using the χ² test.
Table S2: Risk of the development of hypertension at 4 y after baseline by a 1-unit increment in body mass index (BMI) among individuals with the highest tertile of age (≥54 y) at the baseline examination* (incident cases 332 among 2069 individuals).

<table>
<thead>
<tr>
<th>Analytic Model</th>
<th>BMIbaseline</th>
<th>BMIage20y</th>
<th>BMImax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1, OR (95% CI)</td>
<td>1.12 (1.07, 1.18)</td>
<td>1.03 (0.97, 1.09)</td>
<td>1.12 (1.07, 1.17)</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>0.381</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Model 2, OR (95% CI)</td>
<td>1.14 (1.08, 1.19)</td>
<td>1.02 (0.96, 1.09)</td>
<td>1.12 (1.07, 1.17)</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>0.465</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Mean (SD) age was 60.0 (5.6) y among the participants.

Model 1 included sex, smoking habit (never, former, current), parental history of hypertension, HDL-cholesterol, log-transformed triglycerides and type 2 diabetes.

Model 2 included age, sex, smoking habit (never, former, current), parental history of hypertension, HDL-cholesterol, log-transformed triglycerides and type 2 diabetes.