Abstract—The objective of this study was to examine the effect of age on the relationship between alcohol consumption and incident hypertension in a general Japanese population. A cohort of Japanese men (n=37,310) and women (n=78,426) aged 40 to 79 years who underwent community-based health checkups from 1993 to 2004 and were free of hypertension were followed up with annual examinations, including the measurement of blood pressure, until the end of 2010. Incident hypertension was defined as systolic blood pressure of ≥140 mmHg, diastolic blood pressure of ≥90 mmHg, or the initiation of treatment for hypertension. Hazard ratios for incident hypertension according to alcohol consumption were estimated using a Cox proportional hazards model adjusted for possible confounding variables. A total of 45,428 participants (39.3%) developed hypertension (16,155 men and 29,273 women) for a mean follow-up time of 3.9 (1–18) years. Significant associations between alcohol consumption and incident hypertension were found in both sexes and age groups (P for trend was <0.001 for men aged 40–59 years and aged 60–79 years; 0.004 for women aged 40–59 years and 0.026 for women aged 60–79 years). No significant interaction with age on the association of alcohol consumption with incident hypertension was found in either sex (P for interaction, >0.05). Our results suggest that alcohol consumption is a similar risk factor for incident hypertension in both the middle-aged and the older populations. (Hypertension. 2014;63:41-47.)

Key Words: aging ▪ alcohol drinking ▪ cohort studies ▪ hypertension ▪ proportional hazards models

High blood pressure is well-established as a leading cause of stroke,1 ischemic heart disease,2 and cardiovascular disease.3 Worldwide prevalence estimates for hypertension include as many as 1 billion individuals, and ≈7.1 million deaths per year may be attributable to hypertension.4 Many studies have shown that alcohol consumption is associated with increased blood pressure or the development of hypertension.5–10 It has been estimated that 5% to 30% of hypertension cases in the general population can be attributed to alcohol.11,12 The putative beneficial effect of light to moderate alcohol consumption on hypertension, implied by the J-shaped association, has been questioned and needs further investigation.13 Aging induces various physiological changes in the human body, including muscular atrophy and the loss of renal and liver tissue and function,14 implying that the effect of alcohol on blood pressure might vary with age. Several studies have demonstrated that the relationship between alcohol consumption and elevated blood pressure is more apparent in an elderly population.15–18 These studies suggest a plausible hypothesis that a health policy for alcohol intake to prevent hypertension is more important in the older population than in the younger population. However, this age-dependent relationship has been drawn from cross-sectional studies, so there is a lack of evidence to determine whether a health policy to lower alcohol intake for primary prevention of hypertension differs by life stage. Furthermore, the relationship has not been extensively studied in a large cohort of the general population. Examination of the age-specific relationship may yield important knowledge for the primary prevention of hypertension suitable for each life stage. The purpose of this study was to investigate whether aging affects the relationship between the degree of alcohol consumption and incident hypertension in a large Japanese cohort.
Methods

Study Population
In 1993, the Ibaraki prefectural government initiated a large community-based cohort study, known as the Ibaraki Prefectural Health Study (IPHS), to obtain information on health status for the purpose of health education and policy making. The cohort included 458,353 individuals (169,801 men and 288,552 women) 40 to 79 years of age living in Ibaraki prefecture who completed an annual health checkup between 1993 and 2004.

We excluded 77,158 individuals (29,372 men and 47,786 women) from the analysis because of uncompleted follow-up health checkups from 1994 to 2005. We also excluded 5902 individuals (2586 men and 3316 women) with a history of heart disease or stroke, 52938 individuals (12,470 men and 40,468 women) who had ceased consuming alcohol, and 20,010 individuals (86,494 men and 114,516 women) with hypertension at baseline. Furthermore, we excluded 5609 individuals (1569 men and 4040 women) because of incomplete data. A total of 115,736 individuals (37,310 men and 78,426 women) were enrolled in the present study. Informed consent was obtained from community representatives to conduct an epidemiological study on the basis of the guidelines of the Council for International Organizations of Medical Science. The Ethics Committee of Ibaraki Prefecture approved this study.

Baseline Measurements

At baseline, an interview was conducted to ascertain the usual weekly intake of alcohol in go units (a traditional Japanese unit of rice wine that can be converted to grams of ethanol per day as 23 g of ethanol per go unit), smoking status (never, ex-smoker, or current smoker <20 or ≥20 cigarettes/d), histories of stroke, heart disease, hypertension, dyslipidemia, diabetes mellitus, and fasting status (<8 or ≥28 hours after meals). Systolic and diastolic blood pressures were measured once on the right arms of seated participants who had rested for ≥5 minutes. Measurements were made by trained nurses using standard mercury sphygmomanometers N-300 (Nihon Rinsho Kikai Kogyo, Tokyo, Japan) or U-300 (Sanden Ika Kogyo, Tokyo, Japan). When the systolic or diastolic blood pressure was ≥150 or 90 mm Hg, respectively, in 1993–1999, or ≥140 or 90 mm Hg, respectively, in 1999–2004, the blood pressure was measured again after several deep breaths. After this second measurement, lower blood pressure values were almost always observed for the values that we used. At baseline and follow-up, the participants were asked whether they received treatment for hypertension from their physician.

Height in feet with stockings and weight in light clothing were measured, and body mass index was calculated as weight (kg) divided by the square of the height (m²).

The blood samples were drawn from seated subjects. Fasting was not required. Serum total cholesterol and triglycerides were measured by enzymatic methods with an RX-30 device (Nihon Denshi, Tokyo, Japan), and high-density lipoprotein-cholesterol levels were measured by phosphotungstic acid magnesium methods with an MTP-32 (Corona Electric, Ibaraki, Japan). These measurements were performed on the premises of the Osaka Medical Centre for Health Science and Promotion under the aegis of the US National Cholesterol Reference Method Laboratory Network. The laboratory of the Osaka Medical Centre for Health Science and Promotion has been standardized since 1975 by the CDC-NHLBI (Centers for Disease Control and Prevention- The National Heart, Lung, and Blood Institute) Lipid Standardized Program provided by the Centers for Disease Control and Prevention (Atlanta, GA) and has met all criteria for both precision and accuracy of lipid measurements. The plasma glucose level was measured by means of a glucose oxidase electrode method using a GA1140 device (Kyoto Daiichi Kagaku, Kyoto, Japan).

Follow-Up Surveillance and End Point Determination

The participants were followed up with annual examinations until hypertension was diagnosed, until the participant consecutively missed the annual examinations, or until the end of 2010. Blood pressure was measured at the annual follow-up examinations. Systolic and diastolic blood pressures were measured once on the right arms of seated participants who had rested for ≥5 minutes. These measurements were made by trained nurses who used the standard mercury sphygmomanometers N-300 (Nihon Rinsho Kikai Kogyo) or U-300 (Sanden Ika Kogyo) until 2004 and an automated sphygmomanometer Q9920 or Q106 (A&D Company, Tokyo, Japan) between 2005 and 2010. The blood pressure measurement was repeated as described in the baseline, with the values of systolic or diastolic blood pressures that prompted a second measurement being 150 or 90 mm Hg, respectively, until 1999; 140 or 90 mm Hg, respectively, from 2000 to 2007; and 130 or 85 mm Hg, respectively, from 2008 to 2010. Incident hypertension was defined as a systolic blood pressure of ≥140 mm Hg, diastolic blood pressure of ≥90 mm Hg, or initiation of treatment for hypertension.

Statistical Analyses

Participants were classified with regard to alcohol (ethanol) consumption as none, 1.0 to 19.9, 20.0 to 39.9, 40.0 to 59.9, or ≥60.0 g/d.

Blood glucose level was divided into the following 3 categories: normal, <6.1 mmol/L in fasting or <7.8 mmol/L in nonfasting; prediabetes mellitus, 6.1 to 6.9 mmol/L in fasting or 7.8 to 11.0 mmol/L in nonfasting; and diabetes mellitus, ≥7.0 mmol/L in fasting or ≥11.1 mmol/L in nonfasting. Smoking status was divided into the following 4 categories: never smoker, ex-smoker, or current smoker <20 or ≥20 cigarettes/d.

Hazard ratios (HRs) with the corresponding 95% confidence interval for hypertension according to alcohol consumption were calculated, with reference to no alcohol consumption, using a Cox proportional hazards regression model. Covariates included baseline age, body mass index, systolic blood pressure, fasting status, serum total cholesterol level, serum high-density lipoprotein-cholesterol level, log-transformed serum triglyceride level, antisyldiopidemic medication use (yes/no), blood glucose level (normal, prediabetes mellitus, or diabetes mellitus), anti–diabetes mellitus medication use (yes/no), and smoking status (never, ex-smoker, or current smoker <20 or ≥20 cigarettes/d). Groups were stratified by sex and age group (40–59 or 60–79 years). The P values for trend were calculated using a Cox proportional hazard regression model with continuous variables describing alcohol consumption and adjusted for the variables described above. P<0.05 was regarded as statistically significant. All statistical analyses were conducted using SAS, version 9.1 (SAS Institute, Cary, NC).

Results

At baseline, the prevalence of no alcohol consumption was 55.6% in men and 89.6% in women. The prevalence of consumption, 1.0 to 19.9, 20.0 to 39.9, 40.0 to 59.9, and ≥60.0 g/d, were 10.0%, 18.0%, 12.6%, and 3.8% in men and 6.5%, 3.4%, 0.4%, and 0.1% in women, respectively.

Table 1 presents sex-specific comparisons of baseline characteristics between the participants who were and who were not followed up. The participants who were not followed up were significantly younger and had lower systolic and diastolic blood pressure. Among women, alcohol consumption was significantly higher in the participants who were not followed up, but this was not the case among men.

During the follow-up through 2010, for a mean period of 3.9 (1–18) years (3.4 years in men and 4.1 years in women), 45,428 hypertension cases (16,155 in men and 29,273 in women) among the 115,736 adults (37,310 men and 78,426 women) were observed.

Table 2 presents sex-specific baseline characteristics according to the alcohol consumption categories. Statistically significant differences between these study participants
according to alcohol consumption were found for all covari-
ables at baseline in both sexes.

Figure 1 presents sex-stratified HRs for hypertension
according to the alcohol consumption categories. Among
men, compared with participants who consumed no alco-
hol, the multivariable HR for hypertension was significantly
increased in each of the higher alcohol consumption-groups
(\( P \) for trend, \(<0.001\)). Among women, compared with par-
ticipants who consumed no alcohol, the multivariable HR for
hypertension was significantly increased among participants
who consumed 20.0 to 39.9 g/d and insignificantly increased
among participants who consumed 1.0 to 19.9, 40.0 to 59.9,
and \( \geq 60.0 \) g/d (\( P \) for trend, \(<0.001\)).

Figure 2 presents sex- and age-stratified HRs for hyper-
tension according to the alcohol consumption categories.
Significant associations between alcohol consumption and the
risk of incident hypertension were found in both sexes and in
both age groups. Among men aged 40 to 59 years, the mul-
tivariable HR for hypertension was significantly higher for
higher alcohol consumption (\( P \) for trend, \(<0.001\)). Among
men aged 60 to 79 years, the multivariable HR for hyperten-
sion was significantly higher for higher alcohol consumption
(\( P \) for trend, \(<0.001\)). The effect of alcohol consumption on
the risk of hypertension did not differ significantly between
men aged 40 to 59 and 60 to 79 years (\( P \) for interaction, 0.574).

The present large cohort study examined the effect of age
on the association of alcohol consumption with incident hyper-
tension. To the best of our knowledge, this is the first large pro-
spective cohort study showing that the relationships between
alcohol consumption and the risk of incident hypertension are
the same among the older population as they are among the
middle-aged population.

A significant linear association between alcohol consump-
tion and the risk of incident hypertension was consistently
observed in middle-aged and older men and women in our
cohort (\( P \) for trend, \(<0.05\)). MacMahon\(^21\) reviewed 30 cross-
sectional studies and reported that the J-shaped association,
with a higher blood pressure among nondrinkers than among
drinkers of 1 to 2 drinks/d, was observed in \( \approx 40\% \) of the stud-
ies. However, meta-analyses of high-quality cohort studies of
alcohol consumption and incident hypertension\(^22,23\) showed a
significant linear association. The ARIC (Atherosclerosis Risk
in Communities) study, which is the largest US cohort, consist-
ing of 8334 middle-aged individuals, indicated that the associ-
ation between alcohol consumption and incident hyperten-

discussion

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alcohol consumption and incident hypertension\(^22,23\) showed a
significant linear association. The ARIC (Atherosclerosis Risk
in Communities) study, which is the largest US cohort, consist-
ing of 8334 middle-aged individuals, indicated that the associ-
ation between alcohol consumption and incident hyperten-
was J-shaped in white men and women, without excluding former drinkers.24 If former drinkers, who have a high prevalence25 and incidence of hypertension,26 were classified as nondrinkers, it may be the cause of the J-shaped association.27,28 However, further investigations are needed to determine whether light to moderate alcohol consumption has a beneficial effect on hypertension.

Previous cross-sectional studies investigating the age-specific relationship between alcohol consumption and blood pressure were inconsistent with our results.15–18 Barboriak et al17 studied 2307 patients with heart disease and reported that higher alcohol consumption was significantly related to higher systolic and diastolic blood pressure only among patients aged ≥50 years. Klatsky et al16 studied 66510

### Table 2. Sex-Specific Baseline Characteristics According to Level of Daily Alcohol (Ethanol) Consumption Among 37,310 Men and 78,426 Women in Ibaraki, Japan, 1993–2004

<table>
<thead>
<tr>
<th>Variables</th>
<th>None</th>
<th>1.0–19.9</th>
<th>20.0–39.9</th>
<th>40.0–59.9</th>
<th>≥60.0</th>
<th>P for Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of participants</td>
<td>20,740</td>
<td>3729</td>
<td>6721</td>
<td>4706</td>
<td>1414</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>57.4±10.3</td>
<td>56.2±10.3</td>
<td>57.2±10.4</td>
<td>55.9±9.8</td>
<td>53.1±9.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23±2.9</td>
<td>23.1±2.8</td>
<td>22.7±2.7</td>
<td>23.0±2.7</td>
<td>23.2±2.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>122.3±10.6</td>
<td>122.2±10.4</td>
<td>123.2±10.2</td>
<td>124.5±9.8</td>
<td>125.3±9.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>74.5±7.9</td>
<td>74.6±7.8</td>
<td>75.0±7.8</td>
<td>76.0±7.5</td>
<td>76.4±7.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.2±0.9</td>
<td>5.1±0.9</td>
<td>5.0±0.9</td>
<td>5.0±0.9</td>
<td>5.0±0.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL-cholesterol, mmol/L</td>
<td>1.3±0.4</td>
<td>1.3±0.3</td>
<td>1.4±0.4</td>
<td>1.5±0.4</td>
<td>1.6±0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglyceride, mmol/L</td>
<td>1.7±1.1</td>
<td>1.7±1.1</td>
<td>1.5±1.1</td>
<td>1.6±1.1</td>
<td>1.8±1.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Antidyslipidemic medication use, %</td>
<td>1.3</td>
<td>1.0</td>
<td>0.9</td>
<td>0.7</td>
<td>0.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smoking status, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>24.9</td>
<td>27.2</td>
<td>23.6</td>
<td>13.4</td>
<td>8.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>24.8</td>
<td>27.2</td>
<td>27.8</td>
<td>25.6</td>
<td>19.9</td>
<td></td>
</tr>
<tr>
<td>Current &lt;20 cigarettes/d</td>
<td>13.8</td>
<td>14.7</td>
<td>16.1</td>
<td>14.7</td>
<td>9.3</td>
<td></td>
</tr>
<tr>
<td>Current ≥20 cigarettes/d</td>
<td>36.4</td>
<td>30.9</td>
<td>32.6</td>
<td>46.4</td>
<td>61.8</td>
<td></td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of participants</td>
<td>70,266</td>
<td>5120</td>
<td>2682</td>
<td>278</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>53.7±9.7</td>
<td>50.6±8.8</td>
<td>52.6±9.7</td>
<td>49.5±7.2</td>
<td>48.8±6.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.8±2.9</td>
<td>22.8±2.7</td>
<td>22.5±2.9</td>
<td>22.8±3.0</td>
<td>23.7±3.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>120.1±11.3</td>
<td>118.8±11.2</td>
<td>120.2±11.1</td>
<td>119.5±12.3</td>
<td>122.3±11.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>72.5±8.3</td>
<td>72.7±8.2</td>
<td>73.2±8.2</td>
<td>72.7±9</td>
<td>75.4±7.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.3±0.9</td>
<td>5.3±0.9</td>
<td>5.3±0.9</td>
<td>5.2±0.9</td>
<td>5.1±0.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL-cholesterol, mmol/L</td>
<td>1.5±0.4</td>
<td>1.6±0.4</td>
<td>1.7±0.4</td>
<td>1.8±0.4</td>
<td>1.7±0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglyceride, mmol/L</td>
<td>1.4±0.8</td>
<td>1.3±0.8</td>
<td>1.3±0.8</td>
<td>1.3±0.8</td>
<td>1.5±1.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Antidyslipidemic medication use, %</td>
<td>2.2</td>
<td>1.4</td>
<td>1.9</td>
<td>2.9</td>
<td>1.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Smoking status, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>93.1</td>
<td>86.5</td>
<td>77.1</td>
<td>45.3</td>
<td>33.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>0.8</td>
<td>1.8</td>
<td>2.3</td>
<td>3.6</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Current &lt;20 cigarettes/d</td>
<td>4.2</td>
<td>8.7</td>
<td>13.0</td>
<td>19.1</td>
<td>18.8</td>
<td></td>
</tr>
<tr>
<td>Current ≥20 cigarettes/d</td>
<td>2.0</td>
<td>3.0</td>
<td>7.6</td>
<td>32.0</td>
<td>42.5</td>
<td></td>
</tr>
</tbody>
</table>

Values are the mean±SD for continuous variables and the prevalence (%) for categorical variables. BMI indicates body mass index; and HDL, high-density lipoprotein.
individuals aged <40, 40 to 59, and ≥60 years and reported that the relationship between alcohol consumption and higher blood pressure was slightly stronger among individuals aged ≥60 years. Wakabayashi et al. studied 153,546 patients aged 20 to 29, 30 to 39, 40 to 49, 50 to 59, and 60 to 69 years and reported that only among men aged ≥40 years the mean blood pressure was higher in light drinkers than in nondrinkers. However, Nakanishi et al. conducted a 4-year longitudinal study among 3948 Japanese men aged 23 to 35, 36 to 47, and 45 to 59 years. Consistent linear associations between alcohol consumption and the incidence of hypertension were observed among all of the age groups. Our results support the results of Nakanishi et al., suggesting that there was no age difference on the effect of alcohol consumption with regard to incident hypertension. The differing results between the previous cross-sectional studies and the longitudinal study may have been caused by the different methods of the studies. First, some of the cross-sectional studies did not examine the statistical interaction between older age and higher alcohol consumption. Second, although a cross-sectional study is insufficient to examine the causal relationship, a longitudinal study may include the survivor effect because it excludes hypertensive participants at baseline; thus, remaining older participants may have resistance against alcohol. Therefore, there is a possibility that the HR in participants aged 60 to 79 years was underestimated. However, this is often the case in cohort studies. Third, considering that older age is one of the strongest risk factors for the development of hypertension, the results of a stronger pressure effect of alcohol in older age groups may reflect the higher prevalence of hypertension at older ages.

The putative pathology of alcohol-induced hypertension is through the effects of alcohol on cardiac function, acetaldehyde, blood vessels, neural conduction, sympathetic activity, noradrenaline metabolism, renin–angiotensin system, plasma vasopressin, plasma cortisol and ACTH (adrenocorticotropic hormone), and calcium metabolism. Blood pressure generally increases with age because of increased arterial stiffness and the deterioration of the salt- and water-retaining abilities of renal dopamine and angiotensin II. However, our results suggest that the effect of alcohol on blood pressure was not substantially different among the middle-aged and

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**Figure 1.** Sex-stratified hazard ratios (adjusted for age, body mass index, systolic blood pressure, serum total cholesterol, serum total cholesterol level, serum high-density lipoprotein-cholesterol level, serum triglyceride level [log], antidiyslipidemic medication use [yes or no], blood glucose level [normal, pre–diabetes mellitus, and diabetes mellitus], anti–diabetes mellitus medication use [yes or no], and smoking status [never smoker, ex-smoker, or current smoker <20 or ≥20 cigarettes/d]) and 95% confidence intervals of hypertension according to level of daily alcohol (ethanol) consumption among 37,310 men and 78,426 women in Ibaraki, Japan, 1993–2010. **(A)** All men (40–79 years) and **(B)** all women (40–79 years). †P for the trend was calculated using a Cox proportional hazard regression model adjusted for the variables, as described above.

**Figure 2.** Sex- and age-stratified hazard ratios (adjusted for age, body mass index, systolic blood pressure, serum total cholesterol, serum total cholesterol level, serum high-density lipoprotein-cholesterol level, serum triglyceride level [log], antidiyslipidemic medication use [yes or no], blood glucose level [normal, pre–diabetes mellitus, and diabetes mellitus], anti–diabetes mellitus medication use [yes or no], and smoking status [never smoker, ex-smoker, or current smokers <20 or ≥20 cigarettes/d]) and 95% confidence intervals of hypertension according to level of daily alcohol (ethanol) consumption among 37,310 men and 78,426 women in Ibaraki, Japan, 1993–2010. **(A)** Middle aged-men (40–59 years), **(B)** older men (60–79 years), **(C)** middle aged-women (40–59 years), and **(D)** older women (60–79 years). †P for the trend was calculated using a Cox proportional hazard regression model adjusted for the variables, as described above.
older populations. Indeed, alcohol sensitivity, which can be explained by racial and genetic factors, is unlikely to change because of aging within an individual.

The present study suggests that despite of the higher prevalence of hypertension among the older population, the need for primary prevention of hypertension by limiting alcohol consumption is the same in the middle-aged population. Considering that the adverse effect of hypertension on cardiovascular and total mortality was more apparent in the younger population, the preventive strategy for the younger population could be more greatly emphasized.

Our study has several limitations. First, this study may not be widely generalizable. This may be because (1) the follow-up rate was not high and the mean follow-up period was relatively short because participants were free to participate in the annual health checkup, and a one-time absence was sufficient to terminate follow-up; (2) small but significant differences were observed between the participants who were and were not followed up; and (3) the participants were community residents of a single prefecture in Japan. However, the prevalence of alcohol consumption was similar to the results of National Health and Nutrition Survey in Japan. Second, most of the blood pressures were determined by a single measurement. Thus, white-coat hypertension and one-time hypertension may not be excluded from this cohort. However, when the first blood pressure was elevated and classified as hypertensive, a second blood pressure was measured. Third, the instrument used to measure the blood pressure was switched from the standard mercury sphygmomanometer to an automatic sphygmomanometer in 2005. However, the quality and accuracy of automated blood pressure in relation to the awake ambulatory blood pressure were reported to be significantly better than manual blood pressure. Fourth, there is the potential for unmeasured confounding by variables that we could not assess, such as physical activity, type of alcohol, drinking pattern, nutritional status (particularly salt consumption), and family history of hypertension. Greater engagement in regular exercise by the Japanese elderly population compared with the middle-aged population may have modified the effects of alcohol consumption on the development of hypertension among middle-aged and older participants. Although we could not take the type of alcoholic beverages consumed into consideration in our analysis, Okamura et al reported that the effect of alcohol consumption on blood pressure does not depend on the type of alcoholic beverage consumed. Although dietary factors, for example, sodium intake, potassium intake, were not adjusted for in the analysis, Choudhury et al reported that there was little difference in sodium and potassium intake between Japanese male drinkers and nondrinkers.

Perspectives

This large prospective cohort study showed that the association between alcohol consumption and the risk of incident hypertension was the same among the middle-aged and older population. Furthermore, consistent linear dose–response associations between alcohol consumption and incident hypertension were observed among middle-aged and older men and women. The results suggest that limiting alcohol consumption is an important strategy for hypertension prevention for Japanese participants regardless of age.

Acknowledgments

We thank the Ibaraki Health Service Association for supplying the health data.

Sources of Funding

This research was supported by Grant-in-Aids from the Ministry of Health, Labor and Welfare, Health and Labor Sciences Research Grants, Japan (research on health services: H17-Kenkou-007; comprehensive research on cardiovascular and lifestyle-related diseases: H18-Junkankitou[Seishuu]–Ippan-012; and comprehensive research on cardiovascular and lifestyle-related diseases: H20–Junkankitou[Seishuu]–Ippan-013; intractable diseases conquest research: H21-Nanchi-Ippan-059; and comprehensive research on cardiovascular and lifestyle-related diseases: H23-Junkankitou[Seishuu]–Ippan-005).

Disclosures

None.

References

Relationship of Age, Alcohol, and Hypertension

What Is New?

- To the best of our knowledge, this is the first large prospective cohort study demonstrating that the relationships between alcohol consumption and the risk of incident hypertension among the older population are not different from those among the middle-aged population.

What Is Relevant?

- The present study suggests that despite the higher prevalence of hypertension among the older population, the need for the primary prevention of hypertension by limiting alcohol consumption is equally important in the middle-aged population.

Novelty and Significance

Summary

In this large prospective cohort study, relationships between alcohol consumption and the risk of incident hypertension among the older population were not significantly different from those among the middle-aged population. Our results provide a better understanding of the effect of age on the relationship between alcohol consumption and the risk of incident hypertension and should guide public health and clinical efforts aimed at primary prevention in all life stages.