




A risk score predicting new incidence of hypertension in Japan

Yuka Kadomatsu^{1,2} · Mineko Tsukamoto¹ · Tae Sasakabe^{1,3} · Sayo Kawai^{1,3} · Mariko Naito^{1,4} · Yoko Kubo¹ · Rieko Okada¹  · Takashi Tamura¹ · Asahi Hishida¹ · Atsuyoshi Mori⁵ · Nobuyuki Hamajima⁶ · Kohei Yokoi² · Kenji Wakai¹

Received: 29 March 2019 / Revised: 16 July 2019 / Accepted: 6 August 2019 / Published online: 20 August 2019
© The Author(s), under exclusive licence to Springer Nature Limited 2019

Abstract

The prevention of hypertension starts with the awareness of risk. Our aim was to construct a simple and well-validated risk model for nonhypertensive people in Japan consisting of basic clinical variables, using a dataset for two areas derived from the Japan Multi-Institutional Collaborative Cohort Study. We constructed a continuous-value model using data on 5105 subjects participating in both the baseline survey and a second survey conducted after 5 years. The area under the receiver operating characteristic curve (AUC) and the Hosmer–Lemeshow χ^2 statistic for the entire cohort were 0.826 and 7.06, respectively. For validation, the entire cohort was randomly divided 100 times into derivation and validation sets at a ratio of 6:4. The summarized median AUC and the Hosmer–Lemeshow χ^2 statistic were 0.83 and 12.2, respectively. The AUC of a point-based model consisting of integer scores assigned to each variable was 0.826 and showed no difference, compared with the continuous-value model. This simple risk model may help the general population to assess their risks of new-onset hypertension.

Introduction

Hypertension is a major risk factor for cardiovascular, cerebrovascular, and kidney disease [1–4]. In Japan, in 2013, the Ministry of Health, Labour and Welfare launched a health promotion initiative aiming to decrease blood pressure in the population to certain levels by 2022, as part of

Health Japan 21 [5]. Despite this governmental policy, over 30% of adults in Japan still had hypertension in 2016, based on the results from the National Health and Nutrition Survey [6]. From the viewpoint of preventive medicine, it is important to identify individuals at higher risk of hypertension, who should be targeted by interventions.

Several risk models have been developed for various populations and validated through comparison with the well-established Framingham risk score model [7–10]. Variables included in these risk models vary widely, ranging from simple clinical measurements to blood chemistry data and genetic information [11]. However, some risk models that comprised only clinical, easily measurable variables, such as weight, have been demonstrated to have enough power to predict hypertension [7, 12, 13], and the most frequently included variables are age, sex, body mass index (BMI) or weight and height, cigarette smoking status, parental history of hypertension, and blood pressure at baseline [11]. Such simple models have the potential to be useful in clinical practice.

The Framingham risk score model has been found to be a valid tool in multiple populations [9]. However, other investigations have reported that ethnicity is statistically associated with hypertension incidence [10], and the Framingham model might lead to underestimation compared with a risk model developed for the specific population

Supplementary information The online version of this article (<https://doi.org/10.1038/s41371-019-0226-7>) contains supplementary material, which is available to authorized users.

✉ Yuka Kadomatsu
yuka.kadomatsu@gmail.com

¹ Department of Preventive Medicine, Nagoya University Graduate School of Medicine, Nagoya, Japan

² Department of Thoracic Surgery, Nagoya University Graduate School of Medicine, Nagoya, Japan

³ Department of Public Health, Aichi Medical University, Nagakute, Japan

⁴ Department of Oral Epidemiology, Hiroshima University Graduate School of Biomedical and Health Sciences, Hiroshima, Japan

⁵ Seirei Preventive Health Care Center, Shizuoka, Japan

⁶ Department of Healthcare Administration, Nagoya University Graduate School of Medicine, Nagoya, Japan

[14]. To the best of our knowledge, no risk model for new-onset hypertension for both sexes has previously been reported for populations in Japan.

The aim of this study, therefore, was to develop a simple risk model for the prediction of new-onset hypertension among men and women in Japan and to confirm this model's accuracy by cross-validation.

Subjects and methods

Population and study design

The study subjects were drawn from participants in the Japan Multi-Institutional Collaborative Cohort (J-MICC) Study in the Shizuoka and Daiko areas [15]. The J-MICC is a prospective cohort study, whose target population is people who underwent medical health checkups (Shizuoka area) or voluntarily participated in the study (Daiko area). Subjects provided blood samples and information on their lifestyle, parental history, and medical history via a self-administered questionnaire. The baseline surveys in the Shizuoka and Daiko areas were conducted in 2006–2007 and 2008–2010, respectively. The details of the two study areas have been described elsewhere [16, 17]. A second survey was carried out about 5 years after the initial data collection period in each area (Shizuoka: 2012–2013, Daiko: 2013–2014). All of the participants provided written informed consent. The study protocols were approved by the ethics committee of Nagoya University Graduate School of Medicine (numbers 2011–1248 and 2008–0618).

Of the 10,160 participants in the baseline survey, we identified 5105 subjects who completed the second onsite survey. We excluded participants with hypertension ($n = 1094$), cardiovascular disease ($n = 94$), or cerebrovascular disease ($n = 65$) at baseline.

Definition of the variables

Hypertension was defined as any of the following: systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or use of antihypertensive medication. In Daiko, we measured blood pressure twice, with the participant in the sitting position, and used the mean of the two measures for the analysis. In Shizuoka, we measured blood pressure once. If the value was higher than 130/85 mmHg, a second measurement was taken, and the lower of the two values was used for the analysis. BMI was calculated as weight (kg)/height (m^2). Self-reported data were collected on current smoking status, drinking habits, parental history of hypertension, and medicine use. Participants with diabetes mellitus (DM) were defined as those who took any hypoglycemic drug and/or reported a history of diabetes. Ethanol

intake (g/week) was estimated for current drinkers based on the reported frequency and amount consumed per time for six alcoholic beverages (Japanese sake, shochu, shochu-based cocktails, beer, whiskey, and wine) [18].

Statistical analysis

Before analyzing the data, missing data on two variables (smoking status: $n = 3$, BMI: $n = 2$) were replaced with the most common category for the subject's sex.

First, a logistic regression analysis was performed using the entire cohort to analyze the concurrent effects of the selected factors at baseline on new-onset hypertension. To assess the newly developed risk score (continuous-value model), we used the goodness-of-fit test based on the Hosmer–Lemeshow χ^2 statistic [19]. The subjects were divided into deciles by estimated probability of new-onset hypertension; the estimated and observed probabilities were then compared for each group. To examine how the number of factors included in the risk model affects the area under the receiver operating characteristic curve (AUC) and the Hosmer–Lemeshow χ^2 statistic, we tentatively developed models with only age and systolic blood pressure as predictors. Other factors were then included and removed one by one to investigate changes in the AUC. Factors were then added sequentially from the factor with the largest change in the AUC to the smallest change, and the values of the AUC and the Hosmer–Lemeshow χ^2 statistics were computed.

Second, as cross-validation, we randomly split the subjects into two sets: 60% for a derivation set and 40% for a validation set. We developed a risk prediction model from the derivation data using the same variables as in the whole-cohort analysis and assessed the goodness of fit using the validation group's data. We repeated this procedure 100 times. The validity of each model was assessed by the AUC and the goodness-of-fit test based on the Hosmer–Lemeshow χ^2 statistic. We summarized the 100 AUC and Hosmer–Lemeshow χ^2 statistic values by computing a median and its 95% confidence interval (CI).

Third, we constructed a risk prediction model consisting of integer points by rounding the coefficients obtained from the continuous-value model based on data from the entire cohort (point-based model) [20]. We compared the performance of the continuous-value model with that of the point-based model in the entire cohort.

Finally, we developed another risk model with additional biochemical variables. We used the following four measurements that are considered to be related to hypertension: triglycerides, uric acid, low-density, and high-density lipoprotein cholesterol (HDL-C). We used the Friedewald equation to calculate the value of low-density lipoprotein cholesterol [21]. Each additional biochemical variable was

categorized into three to four groups, and each variable was assessed by adding the groups to the main continuous-value model. In this risk model, the study population was limited to subjects with a fasting time of 6 h or longer.

Statistical analyses were conducted using Stata (Version 15.1; Stata Corporation, College Station, TX, USA) and SPSS (Version 24.0; IBM Corporation, Armonk, NY, USA). All *P* values were two-sided, and *P* values < 0.05 were considered to indicate statistical significance.

Results

Hypertension incidence and the multivariate model

Table 1 shows the baseline characteristics of the 3936 participants in the present study. The mean (SD) age was 51.3 (9.2) years, and 58% were women. During the median 5-year follow-up (interquartile range: 4.8–5.3 years), 324 (8.9%) participants newly developed hypertension. In the age-sex-adjusted logistic model, participants with new-onset hypertension were likely to be smokers and to have higher BMI, higher systolic and diastolic blood pressure, higher alcohol consumption, DM, and a parental history of hypertension. In the multivariate-adjusted model (continuous-value model), age ≥ 50 years, current smoking status, ethanol consumption of 100–200 g/week, presence of DM, a history of hypertension among both parents, unknown parental history of hypertension, measured systolic blood pressure ≥ 120 mmHg, and diastolic blood pressure ≥ 80 mmHg were significantly associated with hypertension incidence, while a BMI < 18.5 kg/m² had a significant negative effect on new-onset hypertension.

Effect of the number of factors included in the risk models

The change in the AUC value and the Hosmer–Lemeshow χ^2 statistic with the number of factors included in the continuous-value model are shown in Fig. 1. The risk model with only age and systolic blood pressure achieved a relatively high AUC value (0.799, 95% CI: 0.775–0.824). When we focused only on the value of the AUC, the risk model with six factors (Model 5) reached an AUC value almost equivalent to the continuous-value model. The Hosmer–Lemeshow χ^2 statistic increased until Model 5, and then consistently declined with increasing predictors.

Model validation

For the continuous-value model, the AUC and Hosmer–Lemeshow χ^2 statistic in the entire cohort were 0.826 (95% CI: 0.804–0.848) and 7.06 (*P* value for lack of

fit = 0.53, degrees of freedom = 8), respectively. A comparison of the predicted risk of new-onset hypertension and the observed cumulative incidence by risk score decile is shown in Fig. 2. In the cross-validation sample by 6:4 split, the median AUC was 0.830 (95% CI: 0.828–0.832), and the median Hosmer–Lemeshow χ^2 statistic was 12.2 (95% CI: 6.6–33.3).

Point-based model

Points assigned to each variable and the association between the total score and risk of hypertension are summarized in Tables 2 and 3, respectively. Figure 3 compares the receiver operating characteristic curve of the continuous-value model with that of the point-based model. The AUC for the prediction of new-onset hypertension was 0.826 (95% CI: 0.804–0.848) for the continuous-value model and 0.826 (95% CI: 0.804–0.848) for the point-based model.

Model with biochemical factors

The number of subjects with a fasting time of 6 h or longer was 3 812. All other variables, except for HDL-C, were not significant. Therefore, only HDL-C was added to the final model. Supplementary 2 shows the numbers of and odds ratios for each category. In the risk model with HDL-C, the AUC value was 0.830 (95% CI: 0.809–0.852) and the Hosmer–Lemeshow χ^2 statistic was 7.85 (*P* value for lack of fit = 0.45).

Discussion

We developed a risk score to predict hypertension over a 5-year follow-up among men and women in Japan with a wide age range. This risk prediction model, consisting of basic clinical variables, was evaluated by 100-time cross-validation and was shown to have enough accuracy to predict new-onset hypertension. In particular, the point-based model, in which each variable is weighted by integers, is easy to administer by health care providers and the general population and can help individuals reconsider their lifestyles. The AUCs of the newly developed risk models were almost the same for the continuous and simplified point-based models and the model with a biochemical factor. The model was well validated by cross-validation (AUC = 0.830), considering the AUC value of 0.767 calculated from 35 pooled models in a meta-analysis [11].

Consistent with previously reported risk models, age, smoking history, DM, parental history of hypertension, and blood pressure at baseline were reliable predictors of new-onset hypertension in the present study. Although this finding was not statistically significant, women were found

Table 1 Means and proportions of clinical variables, and odds ratios for risk of new hypertension onset in five years

Variable	Category	All participant <i>n</i> = 3936		New HT onset (+) <i>n</i> = 324		New HT onset (−) <i>n</i> = 3612		Age-sex adjusted model OR (95% CI)	Multivariate model OR (95% CI)
Area	Shizuoka	1799	(46)	151	(47)	1648	(46)	Reference	—
	Daiko	2137	(54)	173	(53)	1964	(54)	1.19 (0.91–1.57)	—
Sex	Men	1651	(42)	177	(55)	1471	(41)	Reference	Reference
	Women	2285	(58)	147	(45)	2138	(59)	0.58 (0.46–0.73)	1.21 (0.88–1.67)
Age (years)		51.3 ± 9.19		56.1 ± 8.01		50.9 ± 9.17		—	—
	<40	462	(12)	9	(3)	453	(13)	Reference	Reference
	40–49	1325	(34)	64	(20)	1261	(35)	2.47 (1.22–5.01)	1.97 (0.94–4.11)
	50–60	1259	(32)	131	(40)	1128	(31)	5.59 (2.82–11.09)	3.38 (1.65–6.95)
	≥60	890	(23)	120	(37)	770	(21)	7.76 (3.90–15.43)	4.37 (2.11–9.06)
BMI (kg/m ²)		21.8 ± 2.93		22.9 ± 3.05		21.7 ± 2.89		—	—
	18.5–25	2986	(76)	245	(76)	2741	(76)	Reference	Reference
	<18.5	427	(11)	13	(4)	414	(12)	0.45 (0.25–0.79)	0.48 (0.26–0.87)
	25–30	476	(12)	56	(17)	420	(12)	1.50 (1.09–2.06)	1.07 (0.76–1.50)
	<30	47	(1)	10	(3)	37	(1)	4.30 (2.05–9.02)	1.87 (0.84–4.19)
Smoking habit	Never	2536	(65)	172	(53)	2364	(66)	Reference	Reference
	Former	899	(23)	90	(28)	809	(22)	1.15 (0.84–1.56)	1.07 (0.77–1.51)
	Current	498	(13)	62	(19)	436	(12)	1.69 (1.20–2.39)	2.14 (1.45–3.16)
Drinking habit ^a (g/week)	Never	1664	(42)	115	(36)	1549	(43)	Reference	Reference
	Former	49	(1)	6	(2)	43	(1)	1.72 (0.70–4.21)	2.19 (0.83–5.76)
	<100	1351	(34)	90	(28)	1261	(35)	0.97 (0.73–1.31)	1.07 (0.79–1.47)
	100–200	427	(11)	53	(16)	374	(10)	1.66 (1.15–2.40)	1.54 (1.04–2.29)
	<200	445	(11)	60	(19)	385	(11)	1.71 (1.18–2.47)	1.30 (0.86–1.95)
DM	No	3845	(98)	302	(93)	3543	(98)	Reference	Reference
	Yes	91	(2)	22	(7)	69	(2)	2.36 (1.43–3.92)	2.68 (1.52–4.74)
Parental history	None	1634	(42)	106	(33)	1528	(42)	Reference	Reference
	One	1445	(37)	123	(38)	1322	(37)	1.42 (1.08–1.87)	1.26 (0.94–1.69)
	Both	294	(8)	31	(10)	263	(7)	1.86 (1.21–2.86)	1.73 (1.09–2.75)
	Unknown	563	(14)	64	(11)	499	(14)	1.69 (1.21–2.36)	1.49 (1.04–2.14)
SBP (mmHg)		112.5 ± 12.5		125.3 ± 9.3		111.4 ± 12.2		—	—
	<120	2715	(69)	77	(24)	2638	(73)	Reference	Reference
	120–129	767	(20)	115	(36)	652	(18)	5.08 (3.73–6.92)	4.17 (2.98–5.84)
	130–139	454	(12)	132	(41)	322	(9)	11.12 (8.12–15.2)	8.15 (5.62–11.82)
DBP (mmHg)		69.9 ± 9.2		77.8 ± 7.4		69.2 ± 9.1		—	—
	<80	3237	(82)	170	(53)	3067	(85)	Reference	Reference
	80–84	471	(12)	87	(27)	384	(11)	3.55 (2.66–4.74)	1.41 (1.01–1.96)
	85–89	228	(6)	67	(21)	161	(5)	6.88 (4.90–9.66)	2.19 (1.49–3.23)

Values are expressed as mean ± standard deviation, or number (proportion)

BMI body mass index, *CI* confidence interval, *DBP* diastolic blood pressure, *DM* diabetes mellitus, *HT* hypertension, *OR* odds ratio, *SBP* systolic blood pressure

^aEthanol consumption (g/week) was estimated by using conversion factors for each alcoholic beverage

to be at higher risk of hypertension in our multivariate model. A higher prevalence of hypertension has been reported among men, compared with women [21, 22]. The relative risk for men, however, is still controversial when

confounding factors (e.g., smoking and drinking) are considered. The weight of sex varies even among several risk models developed in the same Asian countries [12, 14]. One possible explanation for these inconsistent findings may be

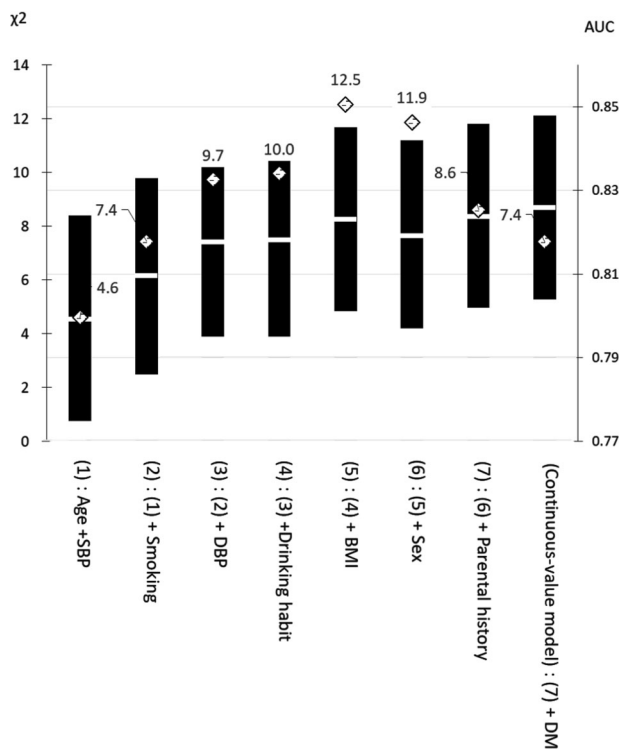


Fig. 1 Effect of the number of factors included in the risk models on AUC values (white horizontal lines indicate point estimates and black bars indicate the 95% CI) and the Hosmer-Lemeshow χ^2 statistics (diamonds)

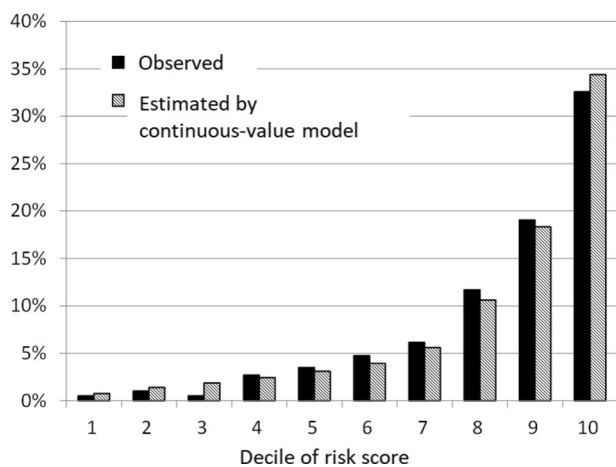


Fig. 2 Comparison of the probability of new-onset hypertension over five years by risk score decile (continuous-value model estimates vs. observed probabilities)

that the proportion of menopausal women differed among existing studies, and estrogen withdrawal is a risk factor for elevated blood pressure [23].

Because of the polymorphism of mitochondrial aldehyde dehydrogenase, tolerance to alcohol varies widely among people in Asian regions, including Japan [24], and hypertension has been shown to be associated with drinking in a

Table 2 Risk points of new onset of hypertension in five years according to risk factor categories (point-based model)

Risk factor		Points awarded
Sex	Male	0
	Female	2
Age (years)	<40	0
	40–49	7
	50–59	12
	≥60	15
BMI (kg/m ²)	18.5–25	0
	<18.5	–7
	25–30	1
	<30	6
	>30	12
Smoking habit	Never	0
	Former	1
	Current	8
Drinking habit ^a (g/week)	Never	0
	Former	8
	<100	1
	100–200	4
DM	<200	3
	No	0
	Yes	10
Parental history	None	0
	One	2
	Both	5
	Unknown	4
SBP (mmHg)	<120	0
	120–129	14
	130–139	21
DBP (mmHg)	<80	0
	80–84	3
	85–89	8

BMI body mass index, *DBP* diastolic blood pressure, *DM* diabetes mellitus, *HT* hypertension, *SBP* systolic blood pressure

^a Ethanol consumption (g/week) was estimated by using conversion factors for each alcoholic beverage

sizeable Japanese cohort [25]. We therefore added calculated ethanol consumption to the variables included in the Framingham risk score model [7]. Several studies have reported that heavy alcohol consumption is associated with increased risk of hypertension [26, 27], and two risk models (a Japanese risk model for working-age men and a Swedish model) have included a variable for drinking [28, 29]. However, these previous risk models did not consider the total amount of alcohol consumption. Using ethanol consumption calculated from responses to the questionnaire, we found that alcohol consumption of 100–200 g/week was a significant risk factor for hypertension. Unexpectedly, the

Table 3 Risk of new onset of hypertension in five years according to total points

Total points	Risk, %	Total points	Risk, %
-7	0.3	41	26.9
-6	0.3	42	28.9
-5	0.4	43	31.0
-4	0.4	44	33.2
-3	0.5	45	35.5
-2	0.5	46	37.8
-1	0.5	47	40.2
0	0.6	48	42.6
1	0.7	49	45.1
2	0.7	50	47.6
3	0.8	51	50.0
4	0.9	52	52.5
5	1.0	53	55.0
6	1.1	54	57.5
7	1.2	55	59.9
8	1.3	56	62.3
9	1.5	57	64.6
10	1.6	58	66.9
11	1.8	59	69.0
12	2.0	60	71.1
13	2.2	61	73.1
14	2.4	62	75.1
15	2.7	63	76.9
16	2.9	64	78.6
17	3.2	65	80.3
18	3.6	66	81.8
19	3.9	67	83.2
20	4.3	68	84.6
21	4.8	69	85.8
22	5.2	70	87.0
23	5.7	71	88.1
24	6.3	72	89.1
25	6.9	73	90.0
26	7.6	74	90.9
27	8.3	75	91.7
28	9.1	76	92.4
29	10.0	77	93.1
30	10.9	78	93.7
31	11.9	79	94.3
32	13.0	80	94.8
33	14.2	81	95.3
34	15.5	82	95.7
35	16.8	83	96.1
36	18.3		
37	19.8		
38	21.5		
39	23.2		
40	25.0		

odds ratio for alcohol consumption of more than 200 g/week was somewhat lower, and this effect was not statistically significant. We do not have a definitive interpretation of this result, but we cannot rule out the possibility of reverse causality.

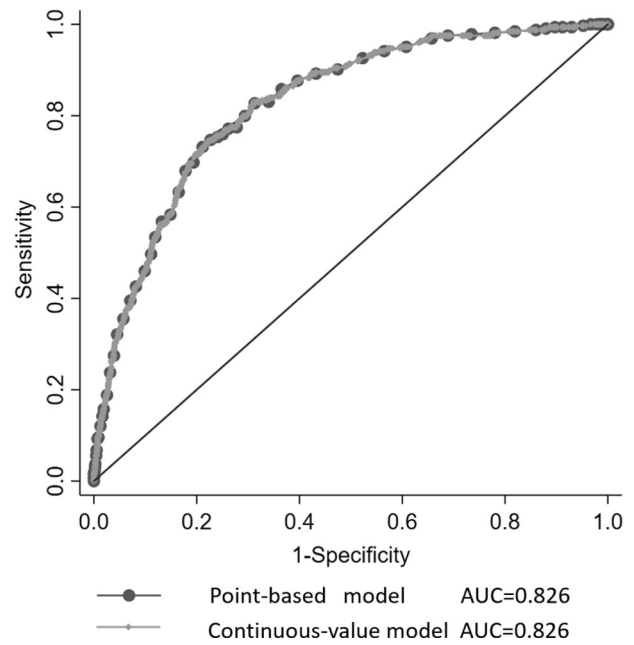


Fig. 3 Receiver operating characteristic (ROC) curves for the continuous-value and point-based models Continuous-value model: AUC = 0.826 (95% CI: 0.804–0.848) Point-based model: AUC = 0.826 (95% CI: 0.804–0.848) *P* value for difference in AUC between the two models: 0.90. AUC area under the curve, CI confidence interval

We classified parental history into four categories, including a category for “unknown.” Our findings for this variable were consistent with those of previous studies suggesting that parental hypertension is a risk factor for new-onset hypertension and that the risk is especially high when both parents have a history of hypertension [7, 14]. In the present study, having an “unknown” parental history also had a statistically meaningful impact on the incidence of hypertension, which may be explained by a lack of awareness of health issues.

The study population included those with DM. According to guideline by the Japanese Society of Hypertension guideline (JSH 2014), the blood pressure target for diabetics is 130/80 mmHg, considering their high risk of stroke in the Japanese population [30]. For this reason, some previous studies have omitted participants who had DM at baseline. However, the latest National Health and Nutrition Examination Survey in Japan [6] indicated that those with strongly or possibly suspected DM account for nearly 30% of the Japanese population. To develop a risk model applicable to as many individuals as possible, we covered subjects with DM by including a variable for the disease in the model.

We applied our study population to compare our risk model and the Framingham model. We excluded those who had an unknown category of parental history of hypertension in the latter risk model and examined its discrimination

and calibration ($n = 3373$) [7] Discrimination (AUC: 0.829, 95% CI: 0.806–0.852) and calibration (Hosmer–Lemeshow χ^2 statistic: 3.36, P value for lack of fit = 0.67) were good. However, the two models are different in that our study population included patients with DM, and the follow-up period was different (5 vs. 4 years in our model and the Framingham model, respectively). Therefore, further studies are required to conclude whether the Framingham risk model can be applied to the Japanese population.

The community-based sample of both men and women was a strength of our investigation, and the accuracy of the newly developed simple risk prediction model was confirmed by cross-validation. Our study also had several limitations. The participants were limited ethnically and geographically. Therefore, further external validation is required to adapt our model to different populations. Verification of the efficacy of this prediction model in the practical settings is expected. Second, history of DM was self-reported; thus, some misclassification might have affected the risk estimation. Third, hypertension is related to several lifestyle factors that were not included in this risk model, such as the amount of salt intake in daily life [31]. Our database, derived from the J-MICC Study, does include information regarding various lifestyle factors, but, although adding many variables to the risk prediction model would contribute to the model's prediction accuracy, it would also decrease its applicability. Finally, some of the predictive variables in our model are not etiologically related to the risk of hypertension incidence. An example of this lack of relation is that, for alcohol drinking, the highest point score as a predictor of incident hypertension was assigned to former drinkers. However, from a biological point of view, quitting drinking alcohol itself is not likely to elevate blood pressure, and the high score appears to be attributable to reverse causality. In addition, sex, age, and parental history are not modifiable, and the effects of these variables cannot be directly translated into a prevention program. Nevertheless, these variables are useful for improving the risk prediction model for hypertension incidence.

The final goal of creating a risk model is to identify individuals at higher risk and to increase health consciousness in the general population. Further studies are required to test the effectiveness of the risk model presented here. However, nationwide screening and lifestyle interventions have been shown to have long-term effectiveness for abdominal obesity, cardiometabolic risks, and hypertension [32]. We anticipate that the point-based model proposed in this study will help the general population to identify their own risks of hypertension and motivate them to improve their lifestyles.

In conclusion, we have constructed a simple yet useful clinical prediction model for 5-year incidence of hypertension among people in Japan.

Summary table

What is known about topic

- Several risk models with clinical variables for new-onset hypertension have been established.
- The weight of each clinical variable in risk models varies across different ethnicities and has to be determined for a designated population with specific characteristics.

What this study adds

- We have constructed a well-validated risk model for both sexes across a wide age range for Japanese populations.
- Our simple point-based risk model, which allows risks to be calculated by adding integers, may help the general people to identify their own risks of hypertension and motivate them to make better lifestyle decisions.

Code availability

Stata code for 6:4 repeated cross-validation can be referenced in Supplementary 1.

Acknowledgements We thank all of the technical staff members at the Department of Preventive Medicine, Nagoya University Graduate School of Medicine; Dr Yatami Asai; and the Seirei Social Welfare Community staff for the recruitment and follow-up of participants in the Daiko and Shizuoka areas in the J-MICC study. We thank Jennifer Barrett, Ph.D., from Edanz Group (www.edanzediting.com/ac) for editing a draft of this manuscript.

Funding This study was supported by the JSPS KAKENHI Grants (No. 16H06277) and Grants-in-Aid for Scientific Research on Priority Areas (No. 17015018) and Innovative Areas (No. 221S0001) from the Japanese Ministry of Education, Culture, Sports, Science and Technology.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

- Fujiyoshi A, Ohkubo T, Miura K, Murakami Y, Nagasawa SY, Okamura T, et al. Blood pressure categories and long-term risk of cardiovascular disease according to age group in Japanese men and women. *Hypertens Res*. 2012;35:947–53.
- Fukuhara M, Arima H, Ninomiya T, Hata J, Yonemoto K, Doi Y, et al. Impact of lower range of prehypertension on cardiovascular events in a general population: the Hisayama Study. *J Hypertens*. 2012;30:893–900.
- Kanno A, Kikuya M, Ohkubo T, Hashimoto T, Satoh M, Hirose T, et al. Pre-hypertension as a significant predictor of chronic kidney disease in a general population: the Ohasama Study. *Nephrol Dial Transpl*. 2012;27:3218–23.
- Takashima N, Ohkubo T, Miura K, Okamura T, Murakami Y, Fujiyoshi A, et al. Long-term risk of BP values above normal for cardiovascular mortality: a 24-year observation of Japanese aged 30 to 92 years. *J Hypertens*. 2012;30:2299–306.
- Ministry of Health, Labour and Welfare. What is Health Japan 21 (the second term)? (Ministry of Health, Labour and Welfare). <http://www.nibiohn.go.jp/eiken/kenkounippon21/en/kenkounippon21/index.html>.
- Ministry of Health, Labour and Welfare. The National Health and Nutrition Survey in Japan, 2017. (Ministry of Health, Labour and Welfare, Tokyo). <https://www.mhlw.go.jp/english/>.
- Parikh NI, Pencina MJ, Wang TJ, Benjamin EJ, Lanier KJ, Levy D, et al. A risk score for predicting near-term incidence of hypertension: the Framingham Heart Study. *Ann Intern Med*. 2008;148:102–10.
- D'Agostino RBS, Grundy S, Sullivan LM, Wilson P. Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *J Am Med Assoc*. 2001;286:180–7.
- Kivimaki M, Batty GD, Singh-Manoux A, Ferrie JE, Tabak AG, Jokela M, et al. Validating the Framingham Hypertension Risk Score: results from the Whitehall II study. *Hypertension*. 2009;54:496–501.
- Muntner P, Woodward M, Mann DM, Shimbo D, Michos ED, Blumenthal RS, et al. Comparison of the Framingham Heart Study hypertension model with blood pressure alone in the prediction of risk of hypertension: the multi-ethnic study of atherosclerosis. *Hypertension*. 2010;55:1339–45.
- Sun DD, Liu JL, Xiao L, Liu Y, Wang ZG, Li C, et al. Recent development of risk-prediction models for incident hypertension: an updated systematic review. *PLoS ONE*. 2017;12:e0187240. <https://doi.org/10.1371/journal.pone.0187240>.
- Chien KL, Hsu HC, Su TC, Chang WT, Sung FC, Chen MF, et al. Prediction models for the risk of new-onset hypertension in ethnic Chinese in Taiwan. *J Hum Hypertens*. 2011;25:294–303.
- Paynter NP, Cook NR, Everett BM, Sesso HD, Buring JE, Ridker PM. Prediction of incident hypertension risk in women with currently normal blood pressure. *Am J Med*. 2009;122:464–71.
- Lim NK, Son KH, Lee KS, Park HY, Cho MC. Predicting the risk of incident hypertension in a Korean middle-aged population: Korean genome and epidemiology study. *J Clin Hypertens*. 2013;15:344–9.
- Hamajima N. The Japan Multi-Institutional Collaborative Cohort Study (J-MICC Study) to detect gene-environment interactions for cancer. *Asian Pac J Cancer Prev*. 2007;8:317–23.
- Asai Y, Naito M, Suzuki M, Tomoda A, Kuwabara M, Fukada Y, et al. Baseline data of Shizuoka area in the Japan Multi-Institutional Collaborative Cohort Study (J-MICC Study). *Nagoya J Med Sci*. 2009;71:137–44.
- Morita E, Hamajima N, Hishida A, Aoyama K, Okada R, Kawai S, et al. Study profile on baseline survey of Daiko Study in the Japan Multi-Institutional Collaborative Cohort Study (J-MICC Study). *Nagoya J Med Sci*. 2011;73:187–95.
- Sasakabe T, Wakai K, Kawai S, Hishida A, Naito M, Suzuki S, et al. Modification of the associations of alcohol intake with serum low-density lipoprotein cholesterol and triglycerides by ALDH2 and ADH1B polymorphisms in Japanese men. *J Epidemiol*. 2018;28:185–93.
- Hosmer DW, Hosmer T, Le Cessie S, Lemeshow S. A comparison of goodness-of-fit tests for the logistic regression model. *Stat Med*. 1997;16:965–80.
- Sullivan LM, Massaro JM, D'Agostino RB, Sr. Presentation of multivariate data for clinical use: the Framingham Study risk score functions. *Stat Med*. 2004;23:1631–60.
- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*. 1972;18:499–502.
- Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *J Am Med Assoc*. 2003;290:199–206.
- Rosano GM, Vitale C, Marazzi G, Volterrani M. Menopause and cardiovascular disease: the evidence. *Climacteric*. 2007;10:19–24.
- Li H, Borinskaya S, Yoshimura K, Kal'ina N, Marusin A, Stepanov VA, et al. Refined geographic distribution of the oriental ALDH2*504Lys (nee 487Lys) variant. *Ann Hum Genet*. 2009;73:335–45.
- Otani K, Haruyama R, Gilmour S. Prevalence and correlates of hypertension among Japanese adults, 1975 to 2010. *Int J Environ Res Public Health*. 2018;15:e1645. <https://doi.org/10.3390/ijerph15081645>.
- Briasoulis A, Agarwal V, Messerli FH. Alcohol consumption and the risk of hypertension in men and women: a systematic review and meta-analysis. *J Clin Hypertens*. 2012;14:792–8.
- Okubo Y, Sairenchi T, Irie F, Yamagishi K, Iso H, Watanabe H, et al. Association of alcohol consumption with incident hypertension among middle-aged and older Japanese population: the Ibarakai Prefectural Health Study (IPHS). *Hypertension*. 2014;63:41–7.
- Fava C, Sjogren M, Montagnana M, Danese E, Almgren P, Engstrom G, et al. Prediction of blood pressure changes over time and incidence of hypertension by a genetic risk score in Swedes. *Hypertension*. 2013;61:319–26.
- Otsuka T, Kachi Y, Takada H, Kato K, Kodani E, Ibuki C, et al. Development of a risk prediction model for incident hypertension in a working-age Japanese male population. *Hypertens Res*. 2015;38:419–25.
- Lindholm LH, Carlberg B. The new Japanese Society of Hypertension guidelines for the management of hypertension (JSH 2014): a giant undertaking. *Hypertens Res*. 2014;37:391–2.
- Cook NR, Cutler JA, Obarzanek E, Buring JE, Rexrode KM, Kumanyika SK, et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP). *BMJ*. 2007;334:885–8.
- Nakao YM, Miyamoto Y, Ueshima K, Nakao K, Nakai M, Nishimura K, et al. Effectiveness of nationwide screening and lifestyle intervention for abdominal obesity and cardiometabolic risks in Japan: the metabolic syndrome and comprehensive lifestyle intervention study on nationwide database in Japan (MetS ACTION-J study). *PLoS ONE*. 2018;13:e0190862.