

A Risk Score for Predicting Near-Term Incidence of Hypertension: The Framingham Heart Study

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Background: Studies suggest that targeting high-risk, nonhypertensive individuals for treatment may delay hypertension onset, thereby possibly mitigating vascular complications. Risk stratification may facilitate cost-effective approaches to management.

Objective: To develop a simple risk score for predicting hypertension incidence by using measures readily obtained in the physician's office.

Design: Longitudinal cohort study.

Setting: Framingham Heart Study, Framingham, Massachusetts.

Patients: 1717 nonhypertensive white individuals 20 to 69 years of age (mean age, 42 years; 54% women), without diabetes and with both parents in the original cohort of the Framingham Heart Study, contributed 5814 person-examinations.

Measurements: Scores were developed for predicting the 1-, 2-, and 4-year risk for new-onset hypertension, and performance characteristics of the prediction algorithm were assessed by using calibration and discrimination measures. Parental hypertension was ascertained from examinations of the original cohort of the Framingham Heart Study.

Results: During follow-up (median time over all person-examinations, 3.8 years), 796 persons (52% women) developed new-onset hypertension. In multivariable analyses, age, sex, systolic and diastolic blood pressure, body mass index, parental hypertension, and cigarette smoking were significant predictors of hypertension. According to the risk score based on these factors, the 4-year risk for incident hypertension was classified as low (<5%) in 34% of participants, medium (5% to 10%) in 19%, and high (>10%) in 47%. The c-statistic for the prediction model was 0.788, and calibration was very good.

Limitations: The risk score findings may not be generalizable to persons of nonwhite race or ethnicity or to persons with diabetes. The risk score algorithm has not been validated in an independent cohort and is based on single measurements of risk factors and blood pressure.

Conclusion: The hypertension risk prediction score can be used to estimate an individual's absolute risk for hypertension on short-term follow-up, and it represents a simple, office-based tool that may facilitate management of high-risk individuals with prehypertension.

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In 2003, the Seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure created a prehypertension category for persons with blood pressures ranging from 120 to 139 mm Hg (systolic) or from 80 to 89 mm Hg (diastolic). The committee strongly advocated lifestyle and behavioral modification for individuals with prehypertension (1). This new recommendation was based largely on epidemiologic evidence that individuals with nonoptimal blood pressure (>120/80 mm Hg) are at increased risk for progression to overt hypertension (1, 2) and that cardiovascular disease risk increases in a graded fashion beginning at a blood pressure of 115/75 mm Hg, well within the "normal" range (1, 3). However, this new categorization scheme resulted in 70 million people being considered prehyperten-

sive, a situation that poses a challenge for both physicians and patients.

Recent clinical trials have focused on the treatment of persons with prehypertension to determine whether hypertension onset can be delayed. A recent clinical trial demonstrated that individuals with blood pressures of 130 to 139 mm Hg (systolic) and 80 to 85 mm Hg (diastolic) who were treated for 2 years with candesartan, an angiotensin II receptor blocker, had a 15% reduction in the incidence of hypertension over 4 years compared with patients randomly assigned to placebo (4). A clinical trial of lifestyle modification among overweight individuals without hypertension demonstrated that a low-salt diet and regular physical exercise reduced hypertension incidence in the short term and several years after the clinical trial (5, 6).

Although these investigations have demonstrated the feasibility and efficacy of preventing hypertension, a strategy that targets all individuals with prehypertension is likely to be associated with substantial medical and economic resources. Evidence suggests that the risk for progression to hypertension depends on clinical factors, such as baseline blood pressure, age, and body mass index (2). An individualized approach of risk stratification and targeted treatment of the nonhypertensive persons who are at greatest risk for progression may be more desirable.

We developed a simple risk score (available at www.annals.org) that can be used in a physician's office to esti-

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Appendix

Appendix Table

Conversion of graphics into slides

Risk calculator

mate the probability that an individual will develop hypertension over 1 to 4 years and to identify the persons who are at the highest risk for hypertension. Such high-risk individuals will probably derive the maximal benefit from nonpharmacologic (lifestyle-related) and pharmacologic interventions aimed at preventing hypertension. Thus, we believe that knowledge of the risk for hypertension will aid patient education and counseling, and it will assist clinical decision making and design of future interventional studies.

METHODS

Sample

The Framingham Heart Study is a community-based, prospective cohort study that began in 1948 with the enrollment of 5209 men and women (whom we refer to as the “original cohort”) (7). In 1971, 5124 men and women were enrolled into the Framingham Offspring Study cohort, which included the children of the original cohort and the spouses of the children. Participant examinations for the offspring cohort occurred approximately every 4 years. The design and methods of the Framingham Offspring Study are described elsewhere (8). For our investigation, offspring cohort participants were eligible if they attended 2 consecutive examinations between the second (1979 to 1983) and seventh examinations (1998 to 2001) and if both of their parents were in the original cohort (8813 person-examinations) (Figure 1). Participants were eligible for inclusion at more than 1 heart study examination cycle if they reached the next examination without

Figure 1. Number of participants available for evaluation of hypertension incidence at baseline examination and number of participants with incident hypertension at follow-up examination.

Baseline examination		Follow-up examination
Examination 2: 1979–1983 <i>n</i> = 603 men <i>n</i> = 762 women	→	Examination 3: 1984–1987 <i>n</i> = 173
Examination 3: 1984–1987 <i>n</i> = 586 men <i>n</i> = 678 women	→	Examination 4: 1987–1991 <i>n</i> = 157
Examination 4: 1987–1991 <i>n</i> = 531 men <i>n</i> = 657 women	→	Examination 5: 1991–1995 <i>n</i> = 134
Examination 5: 1991–1995 <i>n</i> = 498 men <i>n</i> = 605 women	→	Examination 6: 1995–1998 <i>n</i> = 197
Examination 6: 1995–1998 <i>n</i> = 402 men <i>n</i> = 492 women	→	Examination 7: 1998–2001 <i>n</i> = 135

Context

Identifying adults with a high probability of developing high blood pressure could help target nonpharmacologic measures to prevent hypertension.

Contribution

Using data from the Framingham cohort study, the investigators devised a simple risk score with good performance characteristics that identified adults without diabetes who had low (<5%), medium (5% to 10%), or high (>10%) probability of developing hypertension within 4 years. The risk score included points for age, sex, systolic and diastolic blood pressure, body mass index, parental hypertension, and cigarette smoking.

Implication

If this risk score is validated in additional patient populations, it could help clinicians identify high-risk patients with prehypertension.

—The Editors

meeting any exclusion criteria. We excluded participants who had prevalent hypertension (*n* = 2542), prevalent cardiovascular disease (*n* = 233), or serum creatinine values greater than 177 $\mu\text{mol/L}$ (2 mg/dL) (*n* = 4); were younger than age 20 years or older than age 69 years (*n* = 77); had missing covariates (*n* = 46); or had prevalent diabetes mellitus (*n* = 97). We excluded persons with diabetes from our analysis because lower cut-points of systolic and diastolic pressure define the target blood pressure goal in these individuals (1). After we applied the exclusion criteria, 5814 person-examinations from 1717 unique individuals (54% women) remained eligible for analysis. All participants provided written informed consent, and the study protocol was approved by the Boston University Medical Center Institutional Review Board.

Assessment of Risk Factors

At each Framingham Heart Study examination, participants underwent a medical history and physical examination, anthropometric measurements, and laboratory assessment of vascular risk factors. Blood pressure was measured on the left arm of seated participants by a physician using a mercury-column sphygmomanometer, a cuff of the appropriate size, and a standardized protocol. Participants had rested in a chair for 5 minutes before blood pressure was measured, and the average of 2 readings obtained by a physician was considered the examination blood pressure. Systolic blood pressure of 140 mm Hg or higher, diastolic blood pressure of 90 mm Hg or higher, or use of blood pressure-lowering medications defined hypertension. Current smoking was defined as regular cigarette smoking in the year before the examination. Body mass index was calculated as body weight (in kilograms) divided by the square of height (in meters). Consumption of more

Table 1. Baseline Characteristics

Characteristic	Participants (n = 1717)
Mean age (SD), y	42.2 (9.6)
Women, %	54.1
Mean blood pressure (SD), mm Hg	
Systolic	116 (11.1)
Diastolic	75 (7.5)
Current smoker, %	35.2
Moderate to high alcohol intake, %*	23.6
History of parental hypertension, %	
Neither parent	5.7
One parent	34.0
Both parents	60.3
Mean body mass index (SD), kg/m ²	25.1 (4.1)

* Defined as >14 drinks per week in men and >7 drinks per week in women.

than 7 drinks per week in women and more than 14 drinks per week in men defined moderate alcohol intake (1). A fasting blood glucose level of 7.0 mmol/L (126 mg/dL) or more, use of hypoglycemic medications, or both defined diabetes mellitus. Parental hypertension was defined as documented maternal and paternal hypertension at or before the baseline examination at which offspring were eligible for our investigation. Parental hypertension status was ascertained on the basis of blood pressure readings of the original cohort participants by using serial data from examination cycle 3 (1952 to 1956) through examination 27 (2002 to 2004). Prevalent cardiovascular disease was defined as presence of coronary heart disease, stroke, transient ischemic attack, intermittent claudication, or heart failure at any examination. The criteria for diagnosis of these cardiovascular events are described elsewhere (9).

Statistical Analysis

We measured hypertension incidence during the interval between offspring cohort examinations (median, 3.8 years). If a person remained without hypertension at the follow-up examination, he or she remained eligible to contribute to the next 4-year period. A Weibull regression model for interval-censored data (10) was used because only the interval during which new hypertension developed was available; the exact date of onset of hypertension between 2 Framingham Heart Study examinations could not be determined. Significant predictors of hypertension incidence were identified by entering candidate risk factors (age, sex, body weight, body mass index, physical activity index, diabetes, systolic blood pressure, diastolic blood pressure, moderate alcohol intake, current smoking, and parental occurrence of hypertension [modeled as 0, 1, or 2 parents with hypertension]) into a stepwise model. These risk factors have been associated with hypertension in other studies (1). Because diastolic blood pressure decreases with age (11), we evaluated an interaction term for diastolic blood pressure by age, along with the other candidate risk factors. We also tested the following additional interaction terms in multivariable models: diastolic blood pressure by

sex, systolic blood pressure by sex, body mass index by smoking, parental hypertension by age, parental hypertension by sex, and parental hypertension by body mass index.

We developed a risk prediction score from β -coefficients for variables associated with hypertension incidence in multivariable Weibull regression models by using methods described elsewhere (12). Continuous variables were divided into categories to facilitate risk estimation: Age was divided into 10-year groups; systolic blood pressure was divided into 5-mm Hg categories beginning with 110 mm Hg and ending with 139 mm Hg; and diastolic blood pressure was divided into 5-mm Hg categories beginning with 70 mm Hg and ending with 89 mm Hg. Body mass index was classified into 3 levels: less than 25 kg/m² (normal), 25 to less than 30 kg/m² (overweight), and 30 kg/m² or more (obesity).

We derived 1-, 2- and 4-year rates of hypertension incidence directly from the Weibull model (Appendix, available at www.annals.org). We chose to assess 4-year risk because examination cycles in the Framingham Offspring Study occurred approximately every 4 years, and we were interested in assessing a short-term risk score for hypertension. We present 1- and 2-year risks for hypertension because we believe that these short-term risk estimates may also serve to motivate patients and providers. We assessed performance of the risk prediction models by evaluating discrimination through use of the overall c-statistic, described by Harrell and colleagues (13) and Pencina and D'Agostino (14), and we evaluated calibration by using the modified Hosmer-Lemeshow chi-square statistic (15). We used uniform resampling of event times to account for the interval-censored nature of our data and presented medians of 199 resamples. To assess the degree of overoptimism introduced by evaluating our model on the same data on which it was developed, we did a bootstrap simulation by adapting a method described by Harrell and colleagues (13).

All analyses were done with SAS/STAT software, version 9.1 (SAS Institute, Cary, North Carolina). All reported *P* values are 2-sided, and a *P* value less than 0.05 was considered statistically significant.

RESULTS

Sample and Hypertension Incidence

Table 1 shows the baseline characteristics of our sample. In total, 1717 individuals (54% women) contributed 5814 person-examinations of observation. For 60% of participants at baseline, both parents had documented hypertension. During follow-up (median, 3.8 years), 796 persons developed new-onset hypertension. The Appendix Table (available at www.annals.org) shows persons at risk and the proportion who developed hypertension, by baseline systolic and diastolic blood pressure.

Table 2. Multivariable-Adjusted Hazard Ratios for Hypertension*

Variable	Weibull β -Coefficient (\pm SE) [†]	Hazard Ratio (95% CI)	P Value
Age	-0.15641 \pm 0.0474	1.195 (1.089–1.312)	<0.001
Women (vs. men)	-0.20293 \pm 0.0709	1.260 (1.091–1.456)	0.004
Systolic blood pressure	-0.05933 \pm 0.0093	1.070 (1.060–1.080)	<0.001
Diastolic blood pressure	-0.12847 \pm 0.0338	1.158 (1.087–1.234)	<0.001
Current smoker (vs. nonsmoker)	-0.19073 \pm 0.0766	1.243 (1.058–1.460)	0.013
Parental hypertension, per category increment [‡]	-0.16612 \pm 0.0673	1.209 (1.047–1.395)	0.014
Body mass index	-0.03388 \pm 0.0078	1.039 (1.025–1.054)	<0.001
Age by diastolic blood pressure [§]	0.00162 \pm 0.0006	0.998 (0.997–0.999)	0.005

* Variables for which $P < 0.05$ are included. Weibull scale parameter = 0.87692; intercept = 22.94954. Hazard ratios are for a unit increment in continuous variables.

[†] Weibull regression uses an opposite metric to other proportional hazard models and results in opposite sign and interpretation of regression coefficients.

[‡] Hazard ratio for each category increment in parental hypertension (1 parent or both parents) versus no parental hypertension as the reference category.

[§] The effect of increasing diastolic blood pressure on the incidence of hypertension decreases with age.

Multivariable Models

Table 2 shows the results of stepwise, interval-censored Weibull regression analysis for incident hypertension. After multivariable adjustment, age, sex, systolic blood pressure, diastolic blood pressure, body mass index, parental hypertension, and cigarette smoking remained significantly associated with onset of hypertension. The interaction between age and diastolic blood pressure was significant ($P = 0.005$). Increasing levels of diastolic blood pressure were associated with an increased risk for incident hypertension across all age groups, but the slope of the association was steeper in younger groups than in older groups (Figure 2). The interactions of diastolic blood pressure by sex, systolic blood pressure by sex, body mass index by smoking, parental hypertension by age, parental hypertension by sex, and parental hypertension by body mass index were not statistically significant. Because prehypertension is a risk factor for cardiovascular disease (1), we repeated our analyses after excluding 84 individuals who developed cardiovascular disease between heart study examinations. These analyses did not change the results. Coefficients for the variables in Table 2 remained largely unchanged (data not shown).

Model Performance

Our model had very good discrimination (c-statistic, 78.8 [95% CI, 73.3 to 80.3]) and was well calibrated (Hosmer–Lemeshow chi-square statistic, 4.35 [values <20 indicate good calibration]; P value for lack of fit = 0.88. The degree of overoptimism based on bootstrap simulation was estimated at 0.003.

Risk Scores

The Framingham risk calculator for new-onset hypertension is available at www.annals.org. Figure 2 shows the risk scores for hypertension onset at 1, 2, and 4 years. An individual's risk for hypertension can be calculated on the basis of risk factor information. Figure 3 shows the 4-year predicted probabilities of developing hypertension, which range from 9% in a woman with a baseline blood pressure of 120/80 mm Hg who lacks most risk factors to 64% if the woman had risk factors, including parental hypertension,

smoking, obesity, and a blood pressure of 135/85 mm Hg.

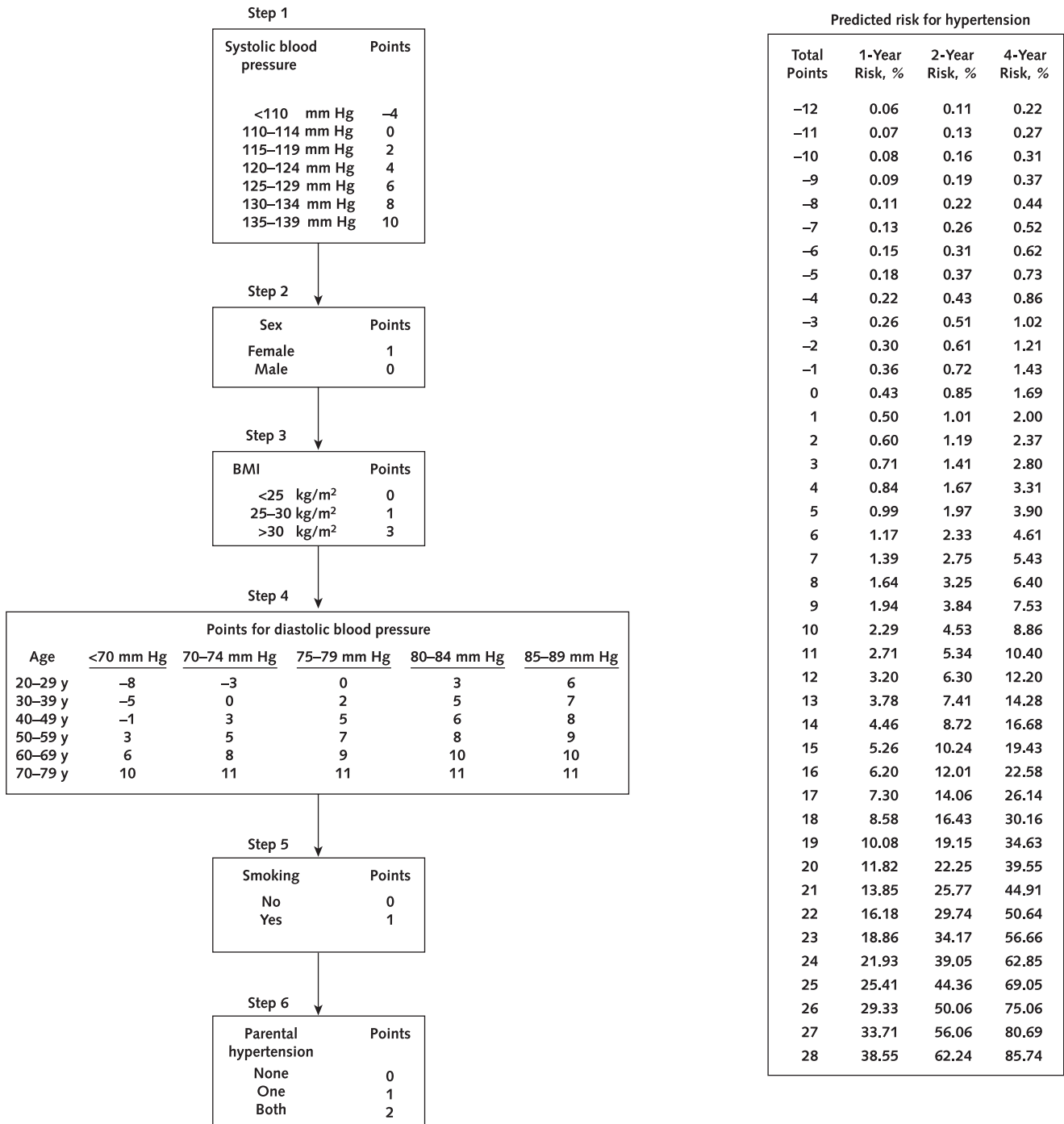
DISCUSSION

We examined clinical predictors of hypertension onset among individuals with new-onset hypertension in the community and developed a risk score that included the following components, which are easily assessed in a physician's office in primary care settings: age, sex, systolic blood pressure, diastolic blood pressure, body mass index, parental hypertension, and cigarette smoking. Our risk score for hypertension had very good discrimination and calibration, and assessment of overoptimism suggests that the model will probably do well when applied to a different sample. Physicians can use the hypertension risk score to measure an individual's estimated risk for hypertension, to inform patients of their risks and help guide their choice of nonpharmacologic measures to prevent hypertension, and to aid in clinical counseling and decision making. In addition, the risk prediction score may be useful in designing interventions to prevent high blood pressure by aiding in selection of participants at the highest risk for hypertension, who are most likely to benefit from treatment. Given that hypertension was measured on only 1 occasion, however, our score may somewhat overestimate an individual's true risk for hypertension.

Risk Factors for Hypertension Onset

Whereas several studies have examined specific risk factors for hypertension, we found no studies on risk scores for high blood pressure in an English-language MEDLINE search up to September 2007 using the search terms *hypertension*, *blood pressure*, *risk tool*, and *risk score*. Our findings that baseline systolic and diastolic blood pressure are critical determinants of hypertension onset are consistent with results from previous investigations (2). Age was positively associated with hypertension risk, probably because vascular stiffness increases with age (16). Increasing diastolic blood pressure was associated with greater risk for hypertension; this effect was more pronounced at younger ages

Figure 2. Calculation of scores to predict 1-, 2-, and 4-year risk for new-onset hypertension.



(as reflected by the negative slope of the significant interaction term). Our finding that body mass index was associated with hypertension onset has been reported in the Framingham and other cohorts (17, 18). Because weight loss decreases blood pressure (19, 20), overweight individuals with high risk scores could be aggressively targeted for intervention.

Whether the incidence of hypertension is higher in men or women is controversial. We found that the incidence of hypertension was slightly higher in women than in men, which other investigators have noted (21, 22). However, some analyses have demonstrated a similar (23) or higher (24-26) hypertension incidence in men compared with women. Finally, 1 previous study demonstrated

higher rates of new-onset isolated systolic hypertension in women but higher rates of new-onset isolated diastolic hypertension in men (27).

Parental Hypertension and Risk in Offspring

Our findings were consistent with those of previous investigations demonstrating that parental hypertension is a risk factor for hypertension in offspring and that risk for hypertension in offspring is greater if both parents (rather than only 1 parent) have a history of hypertension (28–35). Furthermore, hypertension is heritable and has important genetic determinants (36). Shared environmental influences between parents and children may also contribute to the observed association. Some studies relating parental hypertension to hypertension in the offspring have been limited by self-reported family history data (30, 31, 34, 35). A study demonstrated that the accuracy of self-reported parental occurrence of hypertension was 86.2%, with a specificity of 92.9% and a sensitivity of 68.2% (37). These data suggest that self-reported hypertension in parents may be accurate in that it has a high negative predictive value (that is, it identifies nonhypertensive parents) but may be less accurate in identifying hypertensive parents. Although we used validated parental hypertension measures and our estimates were therefore not influenced by the relatively low sensitivity of self-reported parental hypertension, we recognize that medical records are not used to validate patient reports of family history in most clinical settings.

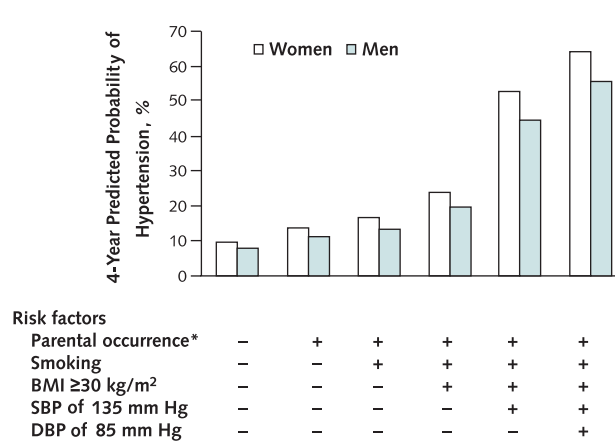
Cigarette Smoking and Hypertension

Our finding that current cigarette smoking is a risk factor for hypertension onset is consistent with several studies (25, 38–40). Yet, other analyses have demonstrated that individuals who smoke may have similar (41, 42) or lower (43–46) blood pressure compared with nonsmoking individuals. Studies of ambulatory blood pressure measurements have shed light on these discrepant findings by demonstrating that the average daily blood pressure of smokers is higher than that of nonsmokers, although blood pressure measured on a single occasion may be similar or lower in smokers than in nonsmokers (47–49). The association between cigarette smoking and elevated blood pressure is probably mediated by mechanisms that increase sympathetic nervous system activation (50, 51) and may be influenced by mechanisms leading to arterial stiffness (52, 53). Of note, smoking cessation has been linked to increased blood pressure (54, 55), although this correlation may be related to weight gain after smoking cessation (54). Therefore, lifestyle modification efforts aimed at reducing hypertension onset among individuals with high hypertension risk scores should target both smoking cessation and weight control.

Rationale for Predicting Cardiovascular Risk Factors

Most existing risk scores used in clinical practice and research are aimed at prevention of secondary and tertiary health outcomes, such as cardiovascular disease (56) or

Figure 3. Four-year predicted probability of hypertension in men and women, by selected risk factors.



Blood pressure was 120/80 mm Hg, unless otherwise indicated. Plus and minus signs below graph indicate the presence or absence of risk factors. BMI = body mass index; DBP = diastolic blood pressure; SBP = systolic blood pressure. *Both parents with hypertension.

cardiovascular disease–related death (57). Risk factors that serve as components of existing risk scores (for example, blood pressure) are often modifiable, and when blood pressure is abnormal in the setting of a high risk score, it is a recommended target for treatment (1, 58). Hypertension is also associated with a shorter life expectancy (59) and antedates most cases of heart failure (60). Thus, prevention of new-onset hypertension (referred to as *primordial prevention*) in turn prevents the emergence of a risk factor and addresses important primary and secondary prevention concerns.

Strengths and Limitations

The strengths of our investigation include the community-based sample and standardized assessment of cardiovascular risk factors, including blood pressure measurement. Our study also has several limitations. The sample is limited geographically and ethnically, consisting of primarily white individuals. Nevertheless, the relationship between cardiovascular risk factors and vascular events in the Framingham data set has been validated in ethnically and geographically diverse populations, which suggests that our risk score may be applicable in other populations (61). However, given the ethnic and racial variation in hypertension incidence and prevalence, our risk equation needs to be validated and may require recalibration for other groups. Similarly, our risk equation was derived from individuals 20 to 69 years of age and may not be generalizable to older or younger persons. We chose to include risk factors that can easily be measured in a clinician’s office, with the aim of developing a simple and usable risk score. Therefore, our risk score does not take into account other known predictors of hypertension that may not be routinely measured. We did not account for the possible con-

founding effects of medications, such as nonsteroidal anti-inflammatory drugs, which have been related to incident hypertension in some reports (62–64). However, in other studies, nonsteroidal anti-inflammatory drugs were associated with incident hypertension (65), or their effects on mean blood pressure were modest (66).

We measured body mass index on only 1 occasion and did not account for the contribution of weight change to hypertension onset, which has been reported (67, 68). Similarly, we measured alcohol and smoking on a single occasion and did not assess changes in these variables, which could be related to blood pressure changes on follow-up. We also considered smoking and alcohol intake as dichotomous variables rather than as continuous or multi-category variables. We chose this approach to keep the risk score simple and because the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood Pressure guidelines recommend use of the same binary cut-points for alcohol consumption (1). Although our information on parental occurrence of hypertension was validated, the extent to which self-reported family history decreases the accuracy of the risk score is unknown. We did not validate this risk prediction instrument in an independent cohort. We measured blood pressure on a single occasion, which may not be as accurate as several measurements (69). We did not model lifestyle factors known to be associated with incident hypertension, such as salt intake (70), vegetable and fat intake (71), or exercise (72). However, these factors would be harder to quantify in the setting of a single office-based visit, which was a key aim of our proposed risk score. We did not assess for nonlinear associations between several risk factors and hypertension incidence. Finally, our risk score was based on a sample from which we excluded individuals with diabetes; the risk score therefore may not be applicable to these persons.

In conclusion, we developed a simple risk prediction algorithm that estimates an individual's 1-, 2- and 4-year probability of developing hypertension. We believe that this score may facilitate targeting of prevention efforts, which is a key objective, given the large population at risk for hypertension. Studies are needed to validate our findings in multiethnic samples.

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APPENDIX

Calculating Risk for Hypertension Directly and Using the Score Sheet

Consider a 60-year-old woman with a systolic blood pressure (*SBP*) of 128 mm Hg and a diastolic blood pressure (*DBP*) of 85 mm Hg, who smokes (*smk*), has 2 parents with history of hypertension (*parhtm*), and whose body mass index is 32 kg/m². By using Table 2 directly, we can estimate her 4-year risk for hypertension:

$$4\text{-year risk} = 1 - \exp \left[- \exp \frac{\ln 4 - (22.9495 - 0.1564 \times \text{age} - 0.2029 \times \text{women} - 0.0593 \times \text{SBP} - 0.1285 \times \text{DBP} - 0.1907 \times \text{smk} - 0.1661 \times \text{parhtm} - 0.0339 \times \text{BMI} + 0.0016 \times \text{age} \times \text{DBP})}{0.8769} \right] =$$

$$1 - \exp \left[- \exp \frac{\ln 4 - (22.9495 - 0.1564 \times 60 - 0.2029 \times 1 - 0.0593 \times 128 - 0.1285 \times 85 - 0.1907 \times 1 - 0.1661 \times 2 - 0.0339 \times 32 + 0.0016 \times 60 \times 85)}{0.8769} \right] =$$

Appendix Table. Persons at Risk for and Development of Hypertension, by Baseline Systolic and Diastolic Blood Pressure Category

Baseline Blood Pressure	Participants at Risk, n	Participants Who Developed Hypertension, n (%)
Systolic		
<110 mm Hg	1705	46 (3)
110–119 mm Hg	1775	132 (7)
120–129 mm Hg	1526	291 (19)
130–139 mm Hg	808	327 (40)
Diastolic		
<75 mm Hg	2842	181 (6)
75–79 mm Hg	1354	171 (13)
80–84 mm Hg	1117	245 (22)
85–89 mm Hg	501	199 (40)

$$\left. \frac{\times 85}{0.8769} \right] = 0.5750 = 57.50\%$$

By using the score sheet from Figure 2, we assign 10 points for an age and diastolic blood pressure combination, 1 point for female sex, 6 points for systolic blood pressure, 1 point for smoking, 2 points for parental hypertension, and 3 points for body mass index, for a total of 23 points. The predicted risk corresponding to 23 points is 56.66%. By using Figure 2, we can also quickly see that if this person was a nonsmoker and had a body mass index less than 25 kg/m², the risk would be lower, at 34.63 (based on 19 points).

Similarly, 4-year risk for a 50-year-old man with a systolic blood pressure of 120 mm Hg and a diastolic blood pressure of 79 mm Hg who smokes, has 1 parent with a history of hypertension, and whose body mass index is 27.5 kg/m² can be calculated directly:

$$4\text{-year risk} = 1 - \exp \left[- \exp \frac{\ln 4 - (22.9495 - 0.1564 \times 50 - 0.2029 \times 0 - 0.0593 \times 120 - 0.1285 \times 79 - 0.1907 \times 1 - 0.1661 \times 1 - 0.0339 \times 27.5 + 0.0016 \times 50 \times 80)}{0.8769} \right] = 0.1489 = 14.89\%$$

By using Figure 2, his points would be calculated by assigning 7 for age and diastolic blood pressure, 0 for male sex, 4 for systolic blood pressure, 1 for smoking, 1 for parental hypertension, and 1 for body mass index, for a total of 14 points corresponding to a risk of 16.68%.