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Modifiable factors and 10-year and lifetime cardiovascular disease risk in adults with new-onset hypertension: insights from the Kailuan cohort

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Abstract

Background Preventing cardiovascular disease (CVD) in adults with hypertension is essential, but it remains uncertain whether optimizing modifiable factors can eliminate the excess CVD risk associated with new-onset hypertension.

Methods In this prospective cohort study, 29,597 adults with new-onset hypertension and no prior CVD (from 2006–2016 surveys) were each matched by age and sex to a normotensive control. Eight modifiable factors were assessed using the American Heart Association's Life's Essential 8 algorithm. We followed participants for incident CVD until December 2020, estimating 10-year and lifetime (age 25–95) CVD risks using the Fine-Gray competing risks model.

Results Over a median follow-up of 9.81 years, adults with new-onset hypertension had higher 10-year (8.97% vs. 6.31%) and lifetime CVD risks (45.55% vs. 34.98%) compared to normotensive controls. After adjusting for age, sex, and other unmodifiable factors, each additional favorable factor was associated with a stepwise reduction in CVD risk (P-trend < 0.05). Hypertensive participants with four or more favorable factors had a 17% lower 10-year CVD risk (HR 0.83; 95% CI 0.72–0.97) and a similar lifetime CVD risk (HR 0.90; 95% CI 0.78–1.05) compared to normotensive controls. Notably, the protective effect was weaker among those with early-onset (before age 45) hypertension than those with later-onset (age ≥ 60) hypertension (P-interaction < 0.05).

Conclusions In adults with new-onset hypertension, maintaining four or more modifiable factors at favorable levels was associated with a CVD risk comparable to that of normotensive individuals. However, young hypertensive adults may require more aggressive interventions to mitigate CVD risk.

Keyword Cardiovascular disease, Hypertension, Modifiable factors, Lifetime risk

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Background

Hypertension is a major contributor to cardiovascular disease (CVD) and premature death globally [1–4]. With an aging population and the increasing prevalence of unhealthy lifestyles, the incidence of hypertension is rising, particularly in low- and middle-income countries [5, 6]. Estimates indicate that 31.5% of adults (1.04 billion people) in low- and middle-income countries and 28.5% of adults (349 million people) in high-income countries have hypertension [7]. Preventing complications from hypertension remains a critical public health priority.

Epidemiological studies have shown that improving modifiable factors significantly reduces CVD risk in both general [8–10] and hypertensive populations [11–14]. However, previous studies often included participants with prevalent hypertension, potentially introducing recall bias due to inaccurate recollection of past exposures or behaviors and survivor bias by overlooking those who did not survive. While clinical trials have demonstrated that controlling blood pressure (BP), blood glucose, and blood lipids decreases CVD risk [15–17], few studies have explored the association between these modifiable factors and the lifetime risk of CVD. Notably, some adults with low short-term CVD risk may still face a heightened risk over their remaining lifespan [18]. Therefore, assessing lifetime risk is a valuable complement to short-term risk assessment in clinical practice [18–20].

Recently, the American Heart Association (AHA) issued Life's Essential 8 (LE8) to assess and promote cardiovascular health [21, 22], potentially reducing CVD risks among adults with hypertension. LE8 comprises modifiable factors such as diet, nicotine exposure, physical activity, sleep, body mass index (BMI), blood lipids, blood glucose, and BP. Leveraging data from the Kailuan cohort, collected biennially since 2006, we identified a population with new-onset hypertension and followed their risk of developing CVD. This study investigates whether improving modifiable factors in LE8 can mitigate the excess 10-year and lifetime risk of CVD associated with new-onset hypertension.

Methods

Study design and participants

The Kailuan study, located in the Kailuan community of Tangshan, China, is a prospective cohort. Detailed study design and procedures have been previously documented [23–26]. In brief, all employees and retirees affiliated with the Kailuan Group Company were biennially invited to participate in health check-up surveys initiated in 2006. Across six survey cycles conducted in 2006–2007, 2008–2009, 2010–2011, 2012–2013, 2014–2015, and 2016–2017, 134,131 participants underwent repeated assessments of their hypertension status (Fig. 1). Hypertension was defined as systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg or self-reported use of antihypertensive medication, and normal BP was defined as systolic BP < 130 mm Hg and diastolic BP < 85 mm Hg, according to the International Society of Hypertension Global Hypertension Practice Guidelines [27]. New-onset hypertension ($n = 38{,}257$) was defined as the development of hypertension in individuals confirmed as non-hypertensive in at least one prior survey. The onset time of hypertension was determined by the earliest recorded date of diagnosis or the first date when hypertension was detected. We excluded 2106 participants who were under 25 or over 95 years old or who had a history of cancer, atrial fibrillation, coronary heart disease (CHD), heart failure, stroke, or end-stage renal disease at baseline.

We matched each participant with new-onset hypertension to a normotensive control from the same survey. The matching criteria included identical age (in years), sex, absence of a history of cancer, atrial fibrillation, CHD, heart failure, stroke, or end-stage renal disease, and complete data on all covariates. For example, a 52-year-old male with new-onset hypertension identified in the 2008 survey was matched with a 52-year-old normotensive male from the same survey who did not have any of the mentioned medical conditions. Finally, 31,335 new-onset hypertension cases were successfully matched with controls, and 29,597 pairs without missing data were included in the primary analysis.

Modifiable factors assessment

Modifiable factors are defined as those that can be improved through lifestyle changes or medical interventions. Based on the American Heart Association LE8 algorithm [22], the modifiable factors in our study included eight key metrics: diet, nicotine exposure, physical activity, sleep, BMI, blood lipids, blood glucose, and BP. Trained and certified nurses conducted assessments using standardized procedures. Information regarding diet, nicotine exposure, physical activity, sleep, and medication history was collected using standardized questionnaires. Since sodium restriction is the most common dietary recommendation for preventing hypertension and reducing BP, and current guidelines advise reducing sodium intake [4, 12, 28], we chose salt consumption to assess diet health. Body weight and height were measured with participants wearing lightweight indoor clothing and no shoes. BMI was then calculated by dividing weight in kilograms by height in square meters. BP was measured three times using a calibrated sphygmomanometer with an appropriately sized cuff, following a 5-min rest in a seated position. The average of these three BP measurements was utilized in the analyses.

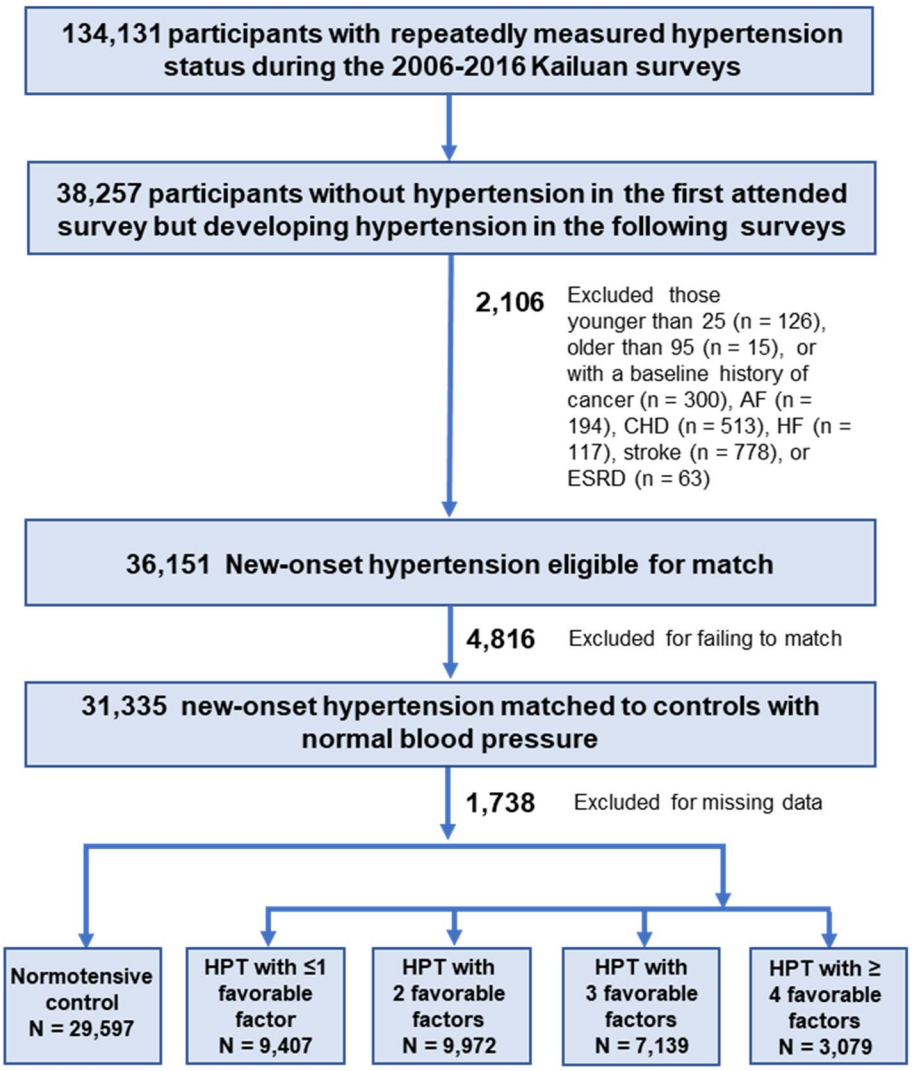

Fig. 1 Cohort identification and analysis grouping flowchart. Abbreviations: AF denotes atrial fibrillation, CHD denotes coronary heart disease, HF denotes heart failure, ESRD denotes end-stage renal disease, and HPT denotes new-onset hypertension

Blood samples were obtained following an overnight fast of 8–10 h and analyzed at the central laboratory of Kailuan Hospital using an auto-analyzer (Hitachi 747; Hitachi, Tokyo, Japan). Fasting blood glucose (FBG) levels were measured using the hexokinase/glucose-6-phosphate dehydrogenase method, while total cholesterol levels were measured using the enzymatic method. High-density lipoprotein cholesterol (HDL-c) levels were measured using the direct method. Non-high-density lipoprotein cholesterol (nHDL-c) was calculated as the difference between total and HDL-c levels.

Modifiable factors were scaled to a 0–100 score range using the LE8 algorithm [22], with detailed scoring criteria provided in Additional file 1: Table S1. Factors with scores exceeding 80 were classified as favorable. Examples include never smoking, obtaining 7–10 h of sleep per night, a BMI < 23 kg/m^2, nHDL-c < 3.36 mmol/L, FBG < 5.60 mmol/L, and BP < 120/ < 80 mm Hg.

Outcomes assessment

The primary outcome in this study was CVD events, defined as fatal and nonfatal CHD (including myocardial infarction and coronary revascularization) [23, 26], heart failure hospitalization [24], and stroke (ischemic and hemorrhagic) [25]. Secondary outcomes included the individual components of the primary outcome, analyzed separately as CHD, heart failure hospitalization, and stroke.

To follow these events among participants, we linked them via unique identification codes to the medical

record systems of both social medical insurance institutions in Tangshan City and Kailuan Hospitals. Additionally, to capture cases potentially missed by these systems, we incorporated data from biennial health examinations, where we collected histories of myocardial infarction and stroke via questionnaire surveys. Furthermore, all study subjects were connected to the death registration system to gather relevant information regarding mortality.

For potential CHD, heart failure, and stroke outcomes, the endpoint evaluation committee of Kailuan Hospital reviewed and adjudicated case-related information without access to baseline data of the study subjects. The adjudication of CHD was based on changes in biochemical markers of myocardial necrosis accompanied by ischemic symptoms, pathological Q waves, ST-segment elevation or depression, or coronary revascularization. Heart failure cases considered in our study pertained to chronic systolic heart failure, diagnosed through clinical manifestations, laboratory tests, and imaging assessments aligned with European Society of Cardiology guidelines [29]. Diagnosis of strokes relied on rapidly developing signs of focal (or global) disturbances in cerebral function lasting more than 24 h without an apparent nonvascular cause.

Covariates assessment

Demographic characteristics, including sex (male or female), date of birth, education levels (more than 9 years or 9 years or less), marital status (married, single, widowed, divorced, or separated individuals), work environment (working in the mine or not), and family history of CVD (present or absent), were collected through a standardized questionnaire. The same questionnaire also gathered medical histories, including hypertension, diabetes, CHD, stroke, or end-stage renal disease, as well as information on medications such as antihypertensive medications. These data were consistently updated biennially to ensure accuracy and relevance.

Statistical analysis

All statistical analyses were conducted using SAS version 9.4, employing two-sided tests with a significance level set at $P<0.05$. Continuous variables were reported as means with standard deviations, and categorical variables were presented as frequencies with proportions. We employed a paired design to compare baseline characteristics between the new-onset hypertension group and normotensive controls. Linear trends among groups of favorable factors in adults with new-onset hypertension were investigated using general linear models for continuous variables and logistic models for categorical variables.

We calculated person-years for each participant from the date of hypertension onset until the earliest of the following events: the first recorded outcome, death, or the end of the follow-up period (December 31, 2020). Incidence rates were presented as events per 100 person-years of observation, along with exact 95% Poisson confidence intervals [30]. Examination of log(–log[survival]) curves indicated that the proportional hazards assumption was not violated. Initially, we employed proportional hazard models, using the matched pair ID as the stratification variable, to estimate the hazard ratio and quantify the excess CVD risk associated with new-onset hypertension. These models included adjustments for additional covariates beyond those used in the matching process, such as educational level, marital status, work environment, family history of CVD, and baseline antihypertensive drug use. The partial population-attributable risks were calculated to describe the proportion of CVD, which could be prevented if everyone maintained the normotensive status [31].

To explore the potential for reducing CVD risk in individuals with new-onset hypertension through improvements in modifiable factors, we evaluate CVD risk based on the status of individual modifiable factors and the cumulative number of favorable factors at baseline, comparing findings with those of normotensive controls. The number of favorable factors categorized hypertensive adults. Participants with one or fewer favorable factors were grouped to ensure sufficient statistical power, while those with four or more were placed in a separate group. To account for the competing risk of death from other causes before CVD, we applied the Fine-Gray model [18–20], using follow-up years as the time scale to estimate the 10-year risk. In the first model, we adjusted for age and sex. In the second model, we included additional adjustments for unmodifiable factors such as educational level, marital status, work environment, family history of CVD, and antihypertensive drug use at baseline. Furthermore, we used age as the time scale to assess the lifetime risk of CVD, from age 25 to 95. The CVD-free years gain was estimated to be the difference in the areas under the survival curves [32].

Comprehensive analyses were conducted to evaluate whether the number of favorable factors influenced CVD risk differently across age of hypertension onset, sex, and BP levels at baseline. Stratified analyses were performed based on the age of hypertension onset (<45 years, 45–59 years, or ≥60 years), sex (male or female), and BP levels (normotensive, BP 140–159/90–99 mmHg, or BP≥160/100 mmHg) at baseline. Likelihood ratio tests were used to assess potential statistical interactions between the number of favorable factors and hypertension onset age, sex, or BP levels about the risk of CVD.

We conducted several sensitivity analyses to evaluate the robustness of our findings. First, we excluded individuals with incomplete data from the primary analyses since less than 2% of matched hypertensive adults had missing information (Additional file 1: Table S2). To address potential bias from missing data, we also performed a sensitivity analysis using multiple imputations that included all participants [33]. Second, participants with a BMI below 18.5 kg/m^2 were excluded to reduce confounding effects related to underweight status. Third, we excluded outcome events occurring within the first 3 months of follow-up to mitigate the possibility of reverse causation. Fourth, we restricted the analysis to individuals diagnosed with hypertension less than 2 years prior to baseline to minimize the influence of longer hypertension duration. Fifth, we redefined hypertension and normal BP according to the ACC/AHA guidelines [34]. Sixth, we excluded participants using antihypertensive medications at baseline to reduce potential bias associated with these treatments. Seventh, since BP levels and antihypertensive drugs during follow-up can significantly influence CVD risk, we further adjusted for these factors among participants with available data. Eighth, acknowledging that changes in modifiable factors during follow-up may affect CVD risk, we also adjusted for changes in the number of favorable factors. In addition, we examined the relationship between changes in the number of modifiable factors and subsequent outcomes, accounting for the baseline number of favorable factors.

Results

At baseline, the mean age of the participants was 52.8 ± 11.1 years, with 19.0% female (Table 1). Adults with new-onset hypertension had similar age, sex, education level, marital status, and mine worker proportion compared to normotensive controls. However, they were more likely to have a family history of CVD and less likely to have favorable levels of diet health, nicotine exposure, sleep health, BMI, blood lipids, FBG, and BP. Among adults with new-onset hypertension, 31.8% had one or fewer favorable factors, 33.7% had two, 24.1% had three, and 10.4% had four or more (Additional file 1: Table S3). Those with more favorable factors tended to be older, female, work at non-mine, and have higher education levels.

New-onset hypertension association with high CVD risk

Over a median follow-up of 9.81 years (interquartile range 7.01 to 11.61 years), 2423 cases of CVD were recorded among adults with new-onset hypertension (Additional file 1: Table S4). As shown in Fig. 2, these adults had a 10-year CVD risk of 8.97% (95% CI 8.63% to 9.31%) and a lifetime CVD risk of 44.55% (95% CI 43.46% to 47.74%). In comparison, there were 1768 CVD cases among normotensive controls, with a 10-year CVD risk of 6.31% (95% CI 6.00% to 6.63%) and a lifetime CVD risk of 34.98% (95% CI 33.15% to 36.91%). After accounting for age, sex, education level, marital status, work environment, family history of CVD, and antihypertensive treatment at baseline, adults with new-onset hypertension had

Table 1 Baseline characteristics of participants with new-onset hypertension and normotensive controls in the Kailuan Study

Items	Normotensive controls	New-onset hypertension	*P*-value
Participant, *n*(%)	29,597	29,597	
Age, year, mean ± SD	52.8 ± 11.1	52.8 ± 11.1	1.00
Female, *n*(%)	5624(19.0)	5624(19.0)	1.00
Education > 9 years, *n*(%)	7534(25.5)	7477(25.3)	0.59
Married status, *n*(%)	29,208(98.7)	29,207(98.7)	0.97
Mine worker, *n*(%)	8715(29.4)	8757(29.6)	0.71
Family CVD history, *n*(%)	1024(3.46)	1226(4.14)	< 0.001
Favorable diet health, *n*(%)	4238(14.3)	4000(13.5)	0.005
Favorable nicotine exposure, *n*(%)	17,257(58.3)	16,813(56.8)	< 0.001
Favorable physical activity, *n*(%)	4433(15.0)	4581(15.5)	0.09
Favorable sleep health, *n*(%)	20,455(69.1)	20,073(67.8)	< 0.001
Favorable BMI level, *n*(%)	11,143(37.6)	6699(22.6)	< 0.001
Favorable blood lipids level, *n*(%)	14,309(48.3)	12,051(40.7)	< 0.001
Favorable blood glucose level, *n*(%)	20,993(70.9)	17,538(59.3)	< 0.001
Favorable blood pressure level, *n*(%)	11,322(38.3)	0(0.00)	< 0.001
Antihypertensive therapy (yes)	0(0.00)	5134(17.35)	< 0.001

Abbreviations: *CVD* Cardiovascular disease; *BMI* Body mass index; *SD* Standard deviation. Diet health, nicotine exposure, physical activity, sleep health, BMI, blood lipids, blood glucose, and blood pressure were assessed according to the Life's Essential 8 algorithm, and a favorable level was defined as ≥ 80 scores

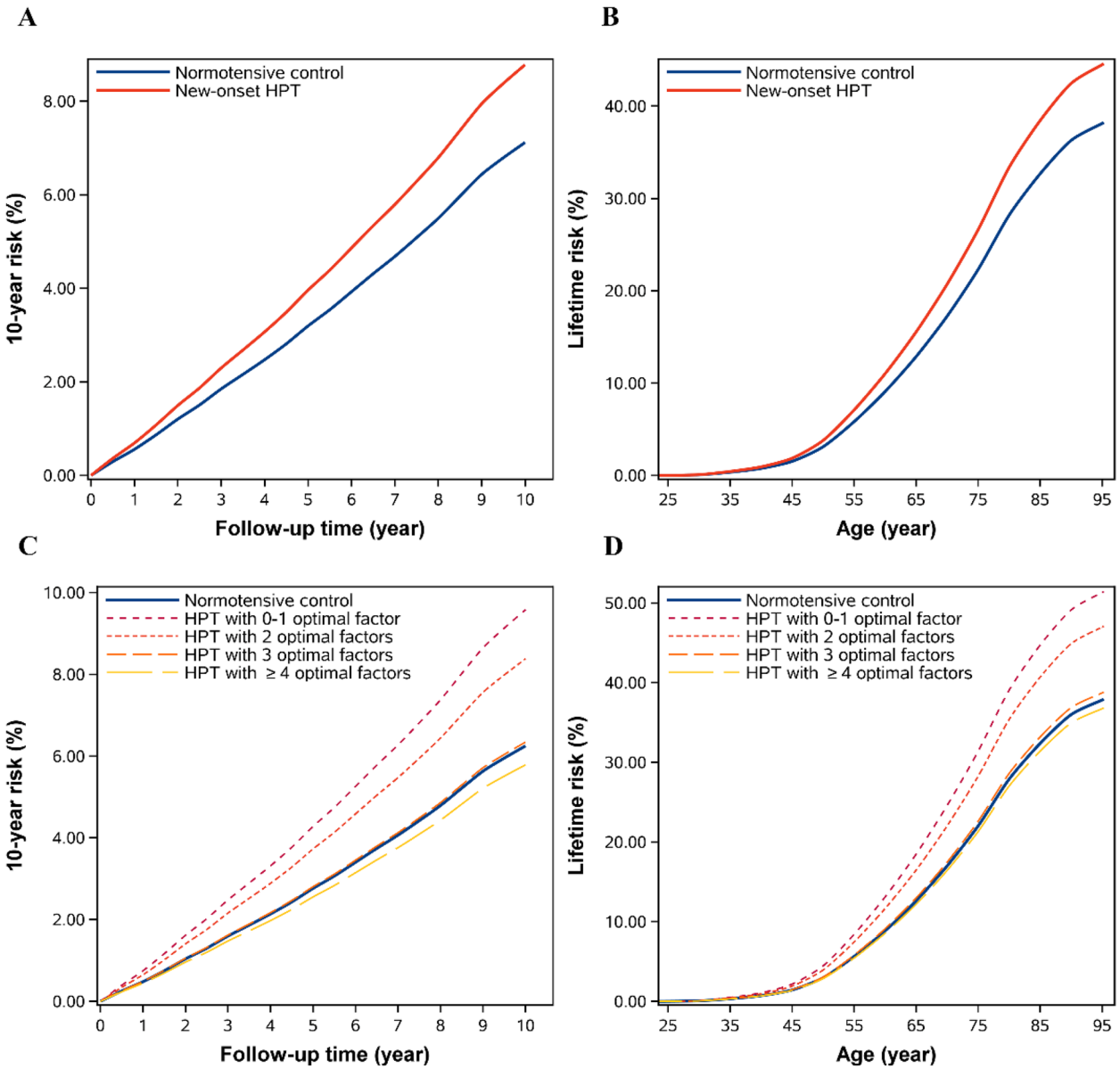

Fig. 2 The age and sex-adjusted 10-year and lifetime risk of cardiovascular disease (CVD) of new-onset hypertension compared with normotensive controls. Panel **A** presents the 10-year risk of CVD for all participants with new-onset hypertension compared to normotensive controls. Panel **B** depicts the lifetime risk of CVD for those with new-onset hypertension in comparison to controls. In Panel **C**, the 10-year risk of CVD is shown for new-onset hypertension cases with varying numbers of favorable factors. Panel **D** displays the lifetime risk of CVD for new-onset hypertension cases with different numbers of favorable factors. HPT denotes new-set hypertension. The risks were adjusted for age (continuous) and sex (male or female). Modifiable factors include diet, nicotine exposure, physical activity, sleep, body mass index, blood lipids, blood glucose, and blood pressure, with a favorable level defined as a Life's Essential 8 score ≥ 80

a 23% higher 10-year and lifetime risk of CVD (HR 1.23, 95% CI 1.15 to 1.31) compared to normotensive controls (Additional file 1: Table S5). The population-attributable risk of new-onset hypertension for CVD risk was 8.90% (95% CI 4.90–12.8%). Additionally, adults with new-onset hypertension tended to have a higher risk of CHD, heart failure, and stroke compared to normotensive controls.

Modifiable factors and attenuated hypertension-related risk of CVD

Examining modifiable factors individually, participants with unfavorable diet health, nicotine exposure, physical activity, sleep health, BMI, blood lipids, and blood glucose appeared to have a relatively high risk of CVD compared with normotensive controls (Additional file 1:

Table S6). Integrating the modifiable factors, an increasing number of favorable factors was associated with a stepwise decrease in CVD risk among adults with new-onset hypertension (*P*-trend < 0.05, Fig. 2 and Additional file 1: Table S7). The 10-year CVD risk, adjusted for age and sex, was 9.48% (95% CI 8.91–10.08%) for those with one or fewer favorable factors, decreasing to 7.87% (95% CI 7.29–8.50%) with two factors, 6.45% (95% CI 5.95–7.00%) with three, and 5.19% (95% CI 4.56–5.92%) with four or more factors, compared to 5.52% (95% CI 5.29%–5.76%) in normotensive controls.

Compared to normotensive controls, the 10-year CVD risk increased by 53% (HR 1.53, 95% CI 1.41–1.67) for individuals with new-onset hypertension and one or fewer favorable factors and by 25% (HR 1.25, 95% CI 1.15–1.37) for those with two favorable factors, after adjusting for age, sex, education level, marital status, work environment, family history of CVD, and antihypertensive treatment (Table 2). However, those with three favorable factors (HR 1.03, 95% CI 0.93 to 1.14) and four or more favorable factors (HR 0.83, 95% CI 0.72 to 0.97) did not have a significantly higher 10-year risk of CVD.

Those with three favorable factors (HR 1.06, 95% CI 0.96–1.17) and four or more favorable factors (HR 0.90, 95% CI 0.78–1.05) did not exhibit a significantly higher lifetime risk of CVD. Analysis of differences in areas under the survival curves revealed that individuals with new-onset hypertension and four or more favorable factors had 2.76 additional CVD-free years compared to normotensive controls. Furthermore, those with new-onset hypertension and four or more favorable factors did not have a higher risk of CHD, heart failure, or stroke compared to normotensive controls (Additional file 1: Table S8).

Having more favorable factors was consistently associated with a lower risk of CVD in both early-onset hypertension (before age 45) and later-onset hypertension (age 60 or older) (Fig. 3 and Additional file 1: Table S9). However, the magnitude of risk reduction was less pronounced among adults with early-onset hypertension (*P*-interaction: 0.002). Compared to normotensive controls, adults with early-onset hypertension and three favorable factors had a 45% higher 10-year relative risk of CVD (HR 1.45, 95% CI 0.92–2.29). In contrast, adults with later-onset hypertension showed no significant difference in 10-year relative CVD risk (HR 1.00, 95% CI 0.86–1.16). Furthermore, no significant interaction was observed between the number of favorable factors and sex about either 10-year or lifetime CVD risk (Additional file 1: Table S10, all *P*-interaction > 0.2). Additionally, the risk reduction associated with favorable factors was attenuated in individuals with BP ≥ 160/100 mmHg (Additional file 1: Table S11, *P*-interaction: 0.07 for 10-year risk; 0.04 for lifetime risk).

The association between having more favorable factors and a lower hypertension-related risk of CVD remained robust across several sensitivity analyses (Additional file 1: Table S12). These analyses included reintroducing participants with missing data through multiple imputations, excluding individuals classified as underweight, excluding outcome events occurring within the first 3 months of follow-up, restricting the analysis to individuals whose hypertension status was confirmed by a check-up survey within a 2-year window, redefining hypertension and normal BP according to ACC/AHA guidelines, excluding participants receiving antihypertensive medications at baseline, further adjusting for BP and antihypertensive

Table 2 Compared with normotensive controls, adjusted cardiovascular disease risk among participants with new-onset hypertension based on the number of modifiable factors at favorable levels

Items	Population(case)	Incidence(/100pys)	10-year risk (%)	10-year HR	Lifetime risk (%)	Lifetime HR
Normotensive control	29,597(1768)	0.66(0.64 to 0.68)	5.77(5.49 to 6.07)	1(ref.)	35.19(33.28 to 37.21)	1(ref.)
HPT with 0–1 favorable factor	9407(896)	1.11(1.06 to 1.16)	8.72(8.22 to 9.24)	1.53(1.41 to 1.67)	46.80(44.11 to 49.65)	1.45(1.33 to 1.59)
HPT with 2 favorable factors	9972(824)	0.95(0.91 to 1.00)	7.17(6.72 to 7.65)	1.25(1.15 to 1.37)	41.90(39.39 to 44.57)	1.25(1.15 to 1.37)
HPT with 3 favorable factors	7139(509)	0.81(0.77 to 0.87)	5.91(5.40 to 6.48)	1.03(0.93 to 1.14)	36.85(34.33 to 39.56)	1.06(0.96 to 1.17)
HPT with ≥ 4 favorable factors	3079(194)	0.72(0.65 to 0.79)	4.83(4.22 to 5.52)	0.83(0.72 to 0.97)	32.35(28.83 to 36.30)	0.90(0.78 to 1.05)
P value for the trend	–		–	< 0.001	–	< 0.001

Abbreviations: *HPT* Denotes new-onset hypertension. Population(case) indicates population size with case number. HR denotes hazard ratio

The 10-year risk and lifetime risk were adjusted for age (continuous), sex (male or female), education level (<9 or ≥9 years), marital status (married or not), work environment (mine or not), family history of cardiovascular diseases (present or absent)

Modifiable factors include diet, nicotine exposure, physical activity, sleep, body mass index, blood lipids, blood glucose, and blood pressure, with a favorable level defined as a Life's Essential 8 score ≥ 80

The *P* value for the trend assessed the linear effect of the number of favorable factors on CVD risk among adults with new-onset hypertension

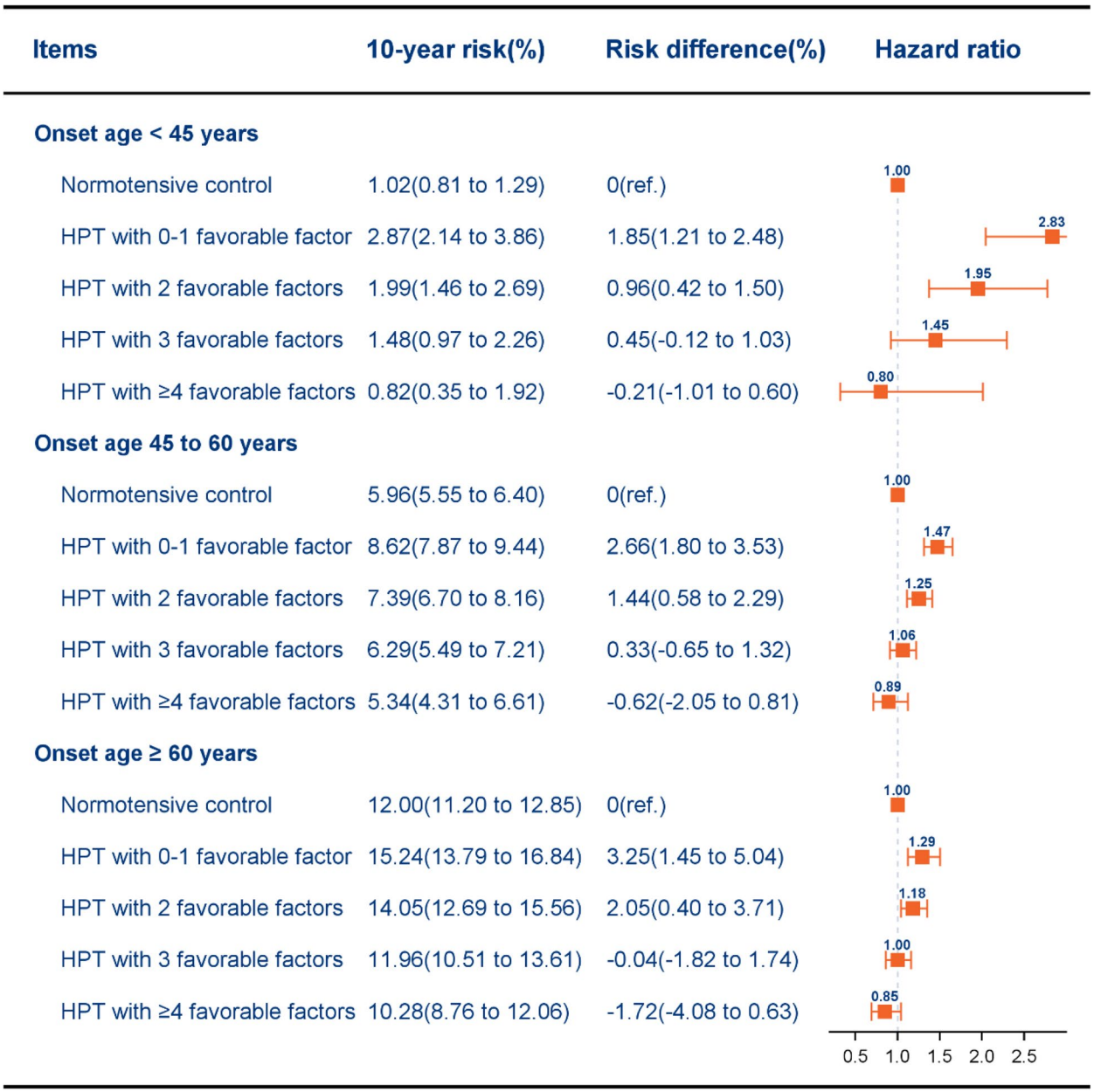

Fig. 3 Adjusted cardiovascular disease (CVD) risk according to favorable factors number in adults with new-onset hypertension, compared to normotensive controls, stratified by hypertension onset age, HPT denotes new-set hypertension. Error bars represent 95% confidence intervals, and dashed lines depict the risk of CVD among normotensive controls. The 10-year risk was adjusted for age (continuous), sex (male or female), education level (<9 or ≥9 years), marital status (married or not), work environment (mine or not), family history of cardiovascular diseases (present or absent), and antihypertensive treatment (yes or no). Modifiable factors include diet, nicotine exposure, physical activity, sleep, body mass index, blood lipids, blood glucose, and blood pressure, with a favorable level defined as a Life's Essential 8 score≥ 80. The *P*value for interaction between favorable factors status and hypertension onset age concerning the 10-year risk of cardiovascular disease was 0.002

treatment during follow-up, and accounting for follow-up changes in favorable factors. Moreover, in addition to the baseline number of modifiable factors at favorable levels associated with CVD risk, improvements in these factors during the follow-up period were also associated with a reduced CVD risk (Additional file 1: Table S13).

Discussion

In this large prospective community cohort, we identified 29,597 new-onset hypertension cases matched with normotensive controls of the same age and sex. Over a median follow-up of 9.81 years, individuals with new-onset hypertension had a 23% higher risk of CVD compared to controls. However, those maintaining four or more favorable factors showed no excess CVD risk. This protective effect remained robust across various sensitivity analyses, underscoring the importance of optimizing modifiable factors even after hypertension onset. Notably, young adults under 45 with new-onset hypertension may require more aggressive strategies to mitigate elevated CVD risk.

Consistently, a recent study pooled data from 1,518,028 individuals across 112 cohort studies, concluding that 57.2% of CVD cases in women and 52.6% in men could be attributed to five modifiable risk factors: BMI, systolic BP, nHDL-c, smoking, and diabetes [8]. Our study, however, focused specifically on adults with new-onset hypertension. We additionally included salt consumption, physical activity, and sleep as modifiable factors, recognizing their strong association with hypertension and cardiovascular health [12]. Based on these modifiable factors, the AHA developed the LE8 to assess and promote cardiovascular health [22]. Our study utilized the LE8 factors and found that maintaining four or more at favorable levels can mitigate the excess CVD risk associated with new-onset hypertension. In addition, our findings are partly supported by the results among patients with diabetes [35, 36], where the excess diabetes-related risk of CVD was decreased with increasing degrees of risk factor control.

Data from the UK Biobank investigated the association between controlling BP, BMI, low-density lipoprotein cholesterol, hemoglobin A1c, albuminuria, smoking, and physical activity with heart failure risk [9]. This study found that hypertensive adults who controlled six risk factors had a lower heart failure risk than non-hypertensive controls (HR 0.79; 95% CI 0.67–0.94). Our study observed that adults with new-onset hypertension and four favorable factors had a lower 10-year heart failure risk (HR 0.73; 95% CI 0.54–1.01) than normotensive controls. Differences in the specific modifiable factors, selection criteria for hypertension and control groups, and racial or ethnic backgrounds might account for the differing number of factors needed to mitigate hypertension-related risk. Because hemoglobin A1c and albuminuria are not routinely included in health check-ups, we used the LE8 factors endorsed by the AHA [22]. Additionally, our study focused on adults with new-onset hypertension, who likely have a lower risk of heart failure due to the shorter disease duration.

Our findings align with prior research indicating that maintaining favorable levels of modifiable factors correlates with reducing the long-term risk of CVD. Lifetime risk offers a more comprehensive perspective on the cumulative burden of CVD over an individual's lifespan, particularly for younger individuals. While their 10-year risk may appear low due to age, they can still face significant long-term risk if modifiable factors remain unfavorable. A meta-analysis of data from 18 cohort studies involving 257,384 individuals revealed that individuals having a favorable risk-factor profile experienced markedly lower risks of death from CVD between 50 and 80 years than participants with two or more major risk factors (4.7% vs. 29.6% among men, 6.4% vs. 20.5% among women) [37]. In addition, a Framingham Heart Study, encompassing 3564 men and 4362 women, reported substantially lower lifetime risks (5.2% versus 68.9% in men, 8.2% versus 50.2% in women) for participants with favorable levels compared to those with two or more risk factors [38]. Our study contributes empirical evidence regarding the impact of modifiable factors, particularly among adults with new-onset hypertension. Furthermore, our research demonstrates that adults with new-onset hypertension and four or more favorable factors had a comparable lifetime risk of CVD to normotensive controls.

Early-onset hypertension is consistently associated with a higher relative risk of CVD, as shown in previous studies [39–41]. A recent meta-analysis indicated that while the absolute risk of adverse outcomes from high BP is low at a young age, the relative risk is significantly elevated; in contrast, absolute risk increases, and relative risk diminishes with age [42]. Although adults with early-onset hypertension have a lower absolute 10-year CVD risk compared to those with later-onset hypertension, their lifetime risk highlights a concerning overall risk trajectory. Our study found that more favorable modifiable factors were associated with reduced CVD risk among individuals with early-onset hypertension. However, the effect was less pronounced than in those with later-onset hypertension. Given the rising prevalence of early-onset hypertension over the past three decades [43, 44] and limited awareness of the heightened lifetime CVD risk [45], we stress the urgent need to promote intensive lifestyle interventions in this population.

Our study found that only 10.4% of adults with new-onset hypertension had four or more favorable factors outlined in LE8. This prevalence aligns with findings from previous studies [21, 46, 47]. For example, an analysis of National Health and Nutrition Examination Survey (NHANES) data from 2003 to 2008 in the USA revealed that only 1% of adults met the ideal levels for all seven metrics in Life's Simple 7 [46]. Similarly, NHANES data

from 2013 to 2018 indicated that overall cardiovascular health in the US population remains suboptimal, as assessed by LE8 [47]. Given the substantial global surge in hypertension prevalence, particularly in China, where rates range from 23 to 45% [45, 48], our findings underscore a significant public health opportunity. Since a more favorable factor profile is associated with lower CVD risk, improving these factors over time has been shown to reduce CVD risk, underscoring the importance of dynamic management. We recommend initiatives focused on managing these modifiable factors among adults with hypertension. Additionally, since improvements in CVD risk among hypertension patients cannot be solely attributed to the traditional risk factors assessed in our study, it is essential to evaluate non-traditional risk factors to manage individuals with hypertension effectively [49].

Limitations

In our study, several limitations merit consideration. First, we determined participants' hypertension status by measuring their BP multiple times and inquiring about their use of antihypertensive medications. We did not differentiate between essential and secondary hypertension. However, since only 5 to 10% of hypertension cases are secondary [50], this likely had minimal impact on our results. Second, we adapted the LE8 algorithm to fit our data—for example, using salt consumption to assess dietary health. Nonetheless, we implemented stringent quality control measures and observed robust outcomes across various sensitivity analyses, consistent with previous studies [10]. Third, we could not completely rule out residual and unmeasured confounders such as genetic predisposition, medication use, and psychological status. Lastly, our study population predominantly consisted of employees and retirees of the Kailuan Group Company, with a higher proportion of males, which may limit the generalizability of our findings. However, this homogeneity may reduce unmeasured confounding and enhance internal validity concerning factors like healthcare access, surveillance bias, structural influences, and self-reported health behaviors.

Conclusions

Our study demonstrates that adults with new-onset hypertension have higher 10-year and lifetime risks of CVD compared to normotensive controls. These excess risks are eliminated when individuals maintain four or more favorable factors. This finding underscores the critical importance of proactive lifestyle interventions, particularly for adults with early-onset hypertension, to mitigate elevated CVD risk.

Abbreviations

AHA	American Heart Association
BMI	Body mass index
BP	Blood pressure
CHD	Coronary heart disease
CI	Confidence interval
CVD	Cardiovascular disease
HDL-c	High-density lipoprotein cholesterol
HR	Hazard ratio
LE8	Life's Essential 8
nHDL-c	Non-high-density lipoprotein cholesterol

Supplementary Information

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Supplementary Material 1

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Authors' Twitter handles

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Authors' contributions

SL.W and YX.W: Original draft preparation, Conceptualization, Resources, and Methodology; C.J and JW.T: Writing—Review with Editing, Funding acquisition, Methodology, and Conceptualization; LM. L, JS.W, and FR.L: Formal analysis, Methodology, and Writing—Review with Editing; J.F, CY.R, and ZF.N: Investigation, Data curation, and Validation. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study's protocols and procedures obtained approval from the Ethics Committee of the Kailuan Medical Group (20060012). All participating individuals provided written informed consent during each survey cycle.

Consent for publication

Written informed consents were obtained from all participants for publication of this study.

Competing interests

The authors declare no competing interests.

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References

1. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: a systematic analysis for the global burden of disease study 2017. Lancet. 2018;392(10159):1923–94.
2. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: update from the GBD 2019 study. J Am Coll Cardiol. 2020;76(25):2982–3021.
3. Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, et al. Global burden of hypertension and systolic blood pressure of at least 110 to 115 mm Hg, 1990–2015. JAMA. 2017;317(2):165–82.
4. Wang JG, Zhang W, Li Y, Liu L. Hypertension in China: epidemiology and treatment initiatives. Nat Rev Cardiol. 2023;20(8):531–45.
5. NCD Risk Factor Collaboration. Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants. Lancet. 2021;398(10304):957–80.
6. NCD Risk Factor Collaboration. Long-term and recent trends in hypertension awareness, treatment, and control in 12 high-income countries: an analysis of 123 nationally representative surveys. Lancet. 2019;394(10199):639–51.
7. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K, et al. Global disparities of hypertension prevalence and control: a systematic analysis of population-based studies from 90 countries. Circulation. 2016;134(6):441–50.
8. Global Cardiovascular Risk Consortium, Magnussen C, Ojeda FM, Leong DP, Alegre-Diaz J, Amouyel P, et al. Global effect of modifiable risk factors on cardiovascular disease and mortality. N Engl J Med. 2023;389(14):1273–85.
9. Kou M, Wang X, Ma H, Li X, Heianza Y, Qi L. Degree of joint risk factor control and incident heart failure in hypertensive patients. JACC Heart Fail. 2023;11(6):678–88.
10. Jin C, Li J, Liu F, Li X, Hui Y, Chen S, et al. Life's essential 8 and 10-year and lifetime risk of atherosclerotic cardiovascular disease in China. Am J Prev Med. 2023;64(6):927–35.
11. Kou M, Wang X, Ma H, Li X, Heianza Y, Qi L. Degree of risk factor control and incident cardiovascular diseases in patients with hypertension. Mayo Clin Proc. 2024;99(3):387–99.
12. Valenzuela PL, Carrera-Bastos P, Galvez BG, Ruiz-Hurtado G, Ordovas JM, Ruilope LM, et al. Lifestyle interventions for the prevention and treatment of hypertension. Nat Rev Cardiol. 2021;18(4):251–75.
13. Xie H, Li J, Zhu X, Li J, Yin J, Ma T, et al. Association between healthy lifestyle and the occurrence of cardiometabolic multimorbidity in hypertensive patients: a prospective cohort study of UK Biobank. Cardiovasc Diabetol. 2022;21(1):199.
14. Zhao M, Yu H, He S, He X, Chen J. Association of life's essential 8 score with risk of all-cause and cardiovascular disease-related mortality in individuals with hypertension. BMC Cardiovasc Disord. 2024;24(1):465.
15. Zhang W, Zhang S, Deng Y, Wu S, Ren J, Sun G, et al. Trial of intensive blood-pressure control in older patients with hypertension. N Engl J Med. 2021;385(14):1268–79.
16. Kosiborod M, Lam CSP, Kohsaka S, Kim DJ, Karasik A, Shaw J, et al. Cardiovascular events associated with SGLT-2 inhibitors versus other glucose-lowering drugs: the CVD-REAL 2 study. J Am Coll Cardiol. 2018;71(23):2628–39.
17. Deedwania P, Murphy SA, Scheen A, Badariene J, Pineda AL, Honarpour N, et al. Efficacy and safety of PCSK9 inhibition with evolocumab in reducing cardiovascular events in patients with metabolic syndrome receiving statin therapy: secondary analysis from the FOURIER randomized clinical trial. JAMA Cardiol. 2021;6(2):139–47.
18. Berry JD, Liu K, Folsom AR, Lewis CE, Carr JJ, Polak JF, et al. Prevalence and progression of subclinical atherosclerosis in younger adults with low short-term but high lifetime estimated risk for cardiovascular disease: the coronary artery risk development in young adults study and multi-ethnic study of atherosclerosis. Circulation. 2009;119(3):382–9.
19. Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. J Am Stat Assoc. 1999;94(446):496–509.
20. Beiser A, D'Agostino RB Sr, Seshadri S, Sullivan LM, Wolf PA. Computing estimates of incidence, including lifetime risk: Alzheimer's disease in the Framingham study. The Practical Incidence Estimators (PIE) macro. Stat Med. 2000;19(11–12):1495–522.
21. Annual Percent Change (APC) and Confidence Interval. National Cancer Institute Division of Cancer Control & Population Sciences. Available from: https://surveillance.cancer.gov/help/joinpoint/setting-parameters/method-and-parameters-tab/apc-aapc-tau-confidence-intervals/estimate-average-percent-change-apc-and-confidence-interval.
22. Lloyd-Jones DM, Allen NB, Anderson CAM, Black T, Brewer LC, Foraker RE, et al. Life's essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. Circulation. 2022;146(5):e18–43.
23. Wu S, Huang Z, Yang X, Zhou Y, Wang A, Chen L, et al. Prevalence of ideal cardiovascular health and its relationship with the 4-year cardiovascular events in a northern Chinese industrial city. Circ Cardiovasc Qual Outcomes. 2012;5(4):487–93.
24. Wang J, Wu M, Wu S, Tian Y. Relationship between body roundness index and the risk of heart failure in Chinese adults: the Kailuan cohort study. ESC Heart Failure. 2022;9(2):1328–37.
25. Li W, Jin C, Vaidya A, Wu Y, Rexrode K, Zheng X, et al. Blood pressure trajectories and the risk of intracerebral hemorrhage and cerebral infarction: a prospective study. Hypertension. 2017;70(3):508–14.
26. Jin C, Chen S, Vaidya A, Wu Y, Wu Z, Hu FB, et al. Longitudinal change in fasting blood glucose and myocardial infarction risk in a population without diabetes. Diabetes Care. 2017;40(11):1565–72.
27. Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al. 2020 International Society of Hypertension global hypertension practice guidelines. Hypertension. 2020;75(6):1334–57.
28. Neal B, Wu Y, Feng X, Zhang R, Zhang Y, Shi J, et al. Effect of salt substitution on cardiovascular events and death. N Engl J Med. 2021;385(12):1067–77.
29. Swedberg K, Cleland J, Dargie H, Drexler H, Follath F, Komajda M, et al. Guidelines for the diagnosis and treatment of chronic heart failure: executive summary (update 2005): the task force for the diagnosis and treatment of chronic heart failure of the European Society of Cardiology. Eur Heart J. 2005;26(11):1115–40.
30. Coxe S, West SG, Aiken LS. The analysis of count data: a gentle introduction to poisson regression and its alternatives. J Pers Assess. 2009;91(2):121–36.
31. Spiegelman D, Hertzmark E, Wand HC. Point and interval estimates of partial population attributable risks in cohort studies: examples and software. Cancer Causes Control. 2007;18(5):571–9.
32. Chudasama YV, Khunti K, Gillies CL, Dhalwani NN, Davies MJ, Yates T, et al. Estimates of years of life lost depended on the method used: tutorial and comparative investigation. J Clin Epidemiol. 2022;150:42–50.
33. Sterne JA, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338: b2393.
34. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. J Am Coll Cardiol. 2018;71(19):e127–248.
35. Wang T, Lu J, Su Q, Chen Y, Bi Y, Mu Y, et al. Ideal cardiovascular health metrics and major cardiovascular events in patients with prediabetes and diabetes. JAMA Cardiol. 2019;4(9):874–83.
36. Rawshani A, Rawshani A, Franzen S, Sattar N, Eliasson B, Svensson AM, et al. Risk factors, mortality, and cardiovascular outcomes in patients with type 2 diabetes. N Engl J Med. 2018;379(7):633–44.

37. Berry JD, Dyer A, Cai X, Garside DB, Ning H, Thomas A, et al. Lifetime risks of cardiovascular disease. N Engl J Med. 2012;366(4):321–9.
38. Lloyd-Jones DM, Leip EP, Larson MG, d'Agostino RB, Beiser A, Wilson PW, et al. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. Circulation. 2006;113(6):791–8.
39. Zhao M, Song L, Sun L, Wang M, Wang C, Yao S, et al. Associations of type 2 diabetes onset age with cardiovascular disease and mortality: the Kailuan study. Diabetes Care. 2021;44(6):1426–32.
40. Chan JC, Lau ES, Luk AO, Cheung KK, Kong AP, Yu LW, et al. Premature mortality and comorbidities in young-onset diabetes: a 7-year prospective analysis. Am J Med. 2014;127(7):616–24.
41. Nanayakkara N, Curtis AJ, Heritier S, Gadowski AM, Pavkov ME, Kenealy T, et al. Impact of age at type 2 diabetes mellitus diagnosis on mortality and vascular complications: systematic review and meta-analyses. Diabetologia. 2021;64:275–87.
42. Li Y, Thijs L, Zhang ZY, Asayama K, Hansen TW, Boggia J, et al. Opposing age-related trends in absolute and relative risk of adverse health outcomes associated with out-of-office blood pressure. Hypertension. 2019;74(6):1333–42.
43. Li Y, Teng D, Shi X, Qin G, Qin Y, Quan H, et al. Prevalence of diabetes recorded in mainland China using 2018 diagnostic criteria from the American Diabetes Association: national cross sectional study. BMJ. 2020;369: m997.
44. Xu Y, Wang L, He J, Bi Y, Li M, Wang T, et al. Prevalence and control of diabetes in Chinese adults. JAMA. 2013;310(9):948–59.
45. Wang Z, Chen Z, Zhang L, Wang X, Hao G, Zhang Z, et al. Status of hypertension in China: results from the china hypertension survey, 2012–2015. Circulation. 2018;137(22):2344–56.
46. Shay CM, Ning H, Allen NB, Carnethon MR, Chiuve SE, Greenlund KJ, et al. Status of cardiovascular health in US adults: prevalence estimates from the National Health and Nutrition Examination Surveys (NHANES) 2003–2008. Circulation. 2012;125(1):45–56.
47. Lloyd-Jones DM, Ning H, Labarthe D, Brewer L, Sharma G, Rosamond W, et al. Status of cardiovascular health in US adults and children using the American Heart Association's new "Life's Essential 8" metrics: prevalence estimates from the National Health and Nutrition Examination Survey (NHANES), 2013 through 2018. Circulation. 2022;146(11):822–35.
48. Lu J, Lu Y, Wang X, Li X, Linderman GC, Wu C, et al. Prevalence, awareness, treatment, and control of hypertension in China: data from 1.7 million adults in a population-based screening study (China PEACE Million Persons Project). Lancet. 2017;390(10112):2549–58.
49. Martín-Timón I, Sevillano-Collantes C, Segura-Galindo A, del Cañizo-Gómez FJ. Type 2 diabetes and cardiovascular disease: have all risk factors the same strength? World J Diabetes. 2014;5(4):444.
50. Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? Eur Heart J. 2014;35(19):1245–54.

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