# National Prevalence of Self-Reported Coronary <br> Heart Disease and Chronic Stable Angina Pectoris 

Factor Analysis of the Underlying Cardiometabolic
Risk Factors in the SuRFNCD-2011

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#### Abstract

Background: Coronary heart disease (CHD) is one of the most common causes of mortality worldwide. The national prevalence remains unclear in most of the developing countries. Objective: This study sought to estimate national prevalence of self-reported CHD and chronic stable angina pectoris in the general adult population of Iran using data from the fourth round of the Surveillance of Risk Factors of Non-Communicable Diseases (SuRFNCD-2011) survey. Methods: The analysis comprised data of 11,867 civilian, nonhospitalized and noninstitutionalized residents ages 6 to 70 years of age. The calculated prevalence of self-reported CHD and chronic stable angina pectoris were extrapolated to the Iranian adult population who were $>20$ years old using the complex sample analysis. The factor analysis was performed for clustering of the associated cardiometabolic risk factors among people ages $>40$ years of age.

Results: The estimated national prevalence of self-reported CHD and chronic stable angina pectoris were $5.3 \%$ ( $95 \%$ confidence interval: 4.6 to 5.9 ) and $7.7 \%$ ( $95 \%$ confidence interval: 4.6 to 8.7 ), respectively. Higher prevalence of these conditions were observed among the older people, urban residents, and women. Factor analysis generated 4 distinct factors that were mainly indicators of dyslipidemia, hypertension, central obesity, hyperglycemia, and tobacco smoking. The factor incorporating hypertension was a significant correlate of selfreported CHD.

Conclusions: We report concerning prevalence of self-reported CHD and chronic stable angina pectoris in the adult population of Iran. The constellation of raised systolic and diastolic blood pressures was significantly predictive of the presence of self-reported CHD.


Coronary heart disease (CHD) is the single largest cause of death in the developed countries and is one of the leading causes of disease burden in the developing nations [1]. In 2013, there were in the excess of 54 million deaths reported globally, with $32 \%$ of these deaths (around 17 million) being attributable to cardiovascular disease; the majority of these deaths due to cardiovascular disease were attributable to CHD and cerebrovascular disease [2]. At least 350,000 deaths occur per annum in Iran, with the rough estimate of 70,000 deaths from this total figure suggested to be caused by CHD [3]. By comparison, CHD alone caused an estimated 1 of every 7 deaths ( $14 \%$ ) in the United States for a total of 375,295 Americans in the year 2011 [4]. As of now, no data from national or subnational
levels have been provided for estimating the prevalence of CHD or chronic stable angina pectoris in Iran.

CHD results from the interaction of different genetic and environmental factors that are yet to be completely understood [5,6]. A cluster of cardiometabolic risk factors has been attributed to the development of arteriosclerosis and CHD, namely the components of central obesity, hyperglycemia, dyslipidemia, and hypertension, the combination of which is characterized as metabolic syndrome [7-9]. The prevalence of these risk factors has been shown to be rising in recent years in Iran [10,11]. However, the true contributions of these cardiometabolic risk factors in the development/ progression of CHD are controversial and diverse among different ethnic groups and nationalities [12,13].

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Started in 2005 and conducted since periodically in 2007 and 2011, the Surveillance of Risk Factors of NonCommunicable Diseases (SuRFNCD) is a nationwide survey of noncommunicable diseases and their associated risk factors among the Iranian adult population. In 2011, estimating the national prevalence of self-reported CHD and chronic stable angina pectoris became possible by asking direct questions associated with these conditions on the clinical study questionnaire. Addition of new itemized questions on the self-reported diagnosis of CHD and angina pectoris allows for tracking the ongoing secular changes in their prevalence over the long term.

In the present study, data from the fourth round of the SuRFNCD (SuRFNCD-2011) was accessed, aggregated, and analyzed to estimate the national prevalence of selfreported CHD and chronic stable angina pectoris in the civilian, nonhospitalized, and noninstitutionalized adult population of Iran. The contribution of different demographic, anthropometric, and biochemical factors to the prevalent self-diagnosed cases of CHD and angina pectoris were carefully investigated using the factor analysis. Factor analysis has been shown to be an appropriate statistical approach for distinguishing particular groups of risk factors that may lead the better understanding of underlying disease processes [14].

## METHODS

## Sampling protocol of SuRFNCD-2011

A randomized multistage cluster sampling framework was conceived to choose a representative sample of civilian, nonhospitalized, and noninstitutionalized Iranian individuals ages 6 to 70 years. Individuals residing in army bases or nursing homes or those hospitalized during the course of interview were not included. By using a 4 -step sampling scheme, 11,867 individuals were surveyed between May 22 and June 20, 2011, in each of the 30 provinces of the country.

Step 1. A total of 402 counties across the country were listed individually or as an assemblage of neighboring counties to designate 50 primary sampling units (PSUs) based on the probability proportional to size algorithm. For this purpose, a consecutive list of potential PSUs and their corresponding cumulative population size was complied. To select the PSU, the sampling interval was first calculated by dividing the population by 50 , which is the number of PSUs required. Then, a random number between 1 and the calculated sampling interval was generated. The unit where this random number fell was assigned as the first PSU. Subsequently, succeeding PSU were determined by adding the constant sampling interval.

Step 2. Within the territory of a selected PSU, individual urban and rural areas were designated as potential secondary sampling units (SSU). These potential SSU found a list, from which the 12 required SSU were selected
using the probability proportional to size method as previously described.

Step 3. For each of the SSU identified in the step 2, a list of households referenced by their postal addresses (10digit postal codes; framework provided by the Iranian Postal Service) was conceived. Twenty households were then selected using a simple random sampling method. In case of a selected postal code belonging to a commercial unit, the adjacent household to the right of the commercial unit was used as the alternative. Each address was then contacted and the inhabitants were registered in the survey.

Step 4. For each of the households selected in the step 3, 2 individuals, 1 younger and 1 older than 50 years of age, were drawn from the World Health Organization (WHO) Kish [15] tables (http://www.who.int/healthinfo/survey/ whslongversion-appendices.pdf, accessed December 25, 2016), and they were visited at their respective households. After 3 attempts, if a sampling individual was not available or refused to participate, the label "nonresponse" was applied. Cluster sampling was conducted under the direction and supervision of Iran's Center for Disease Control (CDC). The final stage was performed by trained interviewers and was overseen by 43 medical universities across the country. For interviews, a Persian translation of the WHO STEPs Chronic Disease Risk Factor Surveillance (http://www.who.int/chp/ steps/instrument/STEPS_Instrument_V3.1.pdf?ua=1, accessed December 25 , 2016) was used. At the beginning of each interview, a consent form was read by the interviewer to the interviewee, and acceptance or refusal to participate was formally recorded.

All procedures described herein were conducted in accordance with the guidelines and standards laid down in the current revision of the Declaration of Helsinki. The CDC Board of Ethics approved the study protocol.

The nationwide SuRFNCD-2011 survey had 3 steps: 1) collection of demographics and the associated healthrelated characteristics; 2) measurement of anthropometrics and clinical metrics; and 3) measurements of biochemical indices. Age, sex, history of diabetes, hypertension, dyslipidemia, chronic stable angina pectoris, and CHD were assessed among the recruited participants.

## Physical examinations

In the household visit and following the interview, physical examinations were performed by trained interviewers and included measurement of weight, height, waist circumference, and blood pressure. Weight was measured on a portable digital scale with the participants wearing light clothing and was recorded to the nearest 0.1 kg . An inflexible measurement tape was used to measure height while the individual standing still, with no shoes or socks on, and was recorded with a precision of 0.1 cm . Body mass index (BMI) was calculated as weight ( kg ) divided by height $\left(\mathrm{m}^{2}\right)$. After resting for at least $10 \mathrm{~min}, 3$ blood pressure measurements, 5 min apart, were taken by trained health
staff using the calibrated Omron M7 digital sphygmomanometers (Hoofddorp, The Netherlands) with the appropriate size cuff covering at least $80 \%$ of the right arm. The first reading was discarded and mean values for systolic and diastolic blood pressures (SBP and DBP, respectively) were determined by averaging the second and third readings.

## Laboratory evaluations

Participants were instructed to fast overnight for 12 to 14 h prior to blood sampling the following morning. At each center, 10 ml of venous blood was drawn and collected from each of the participants, sampled in cold biochemistry tubes (4 to $8^{\circ} \mathrm{C}$ ), and sent within 4 h to representative collaborating laboratories. Samples were then immediately centrifuged (1,500 revolutions $/ \mathrm{min}$ for 10 min at standard room temperature: $21^{\circ} \mathrm{C}$ ) and the extracted serum was used for laboratory evaluations. Fasting plasma glucose (FPG) was measured with enzymatic calorimetric methods using the glucose oxidase test. Serum concentrations of triglycerides, total cholesterols, lowdensity lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were determined by enzymatic methods. All measurements were performed using the approved, quality-controlled commercially available kits (Pars Azmun, Karaj, Iran) provided and distributed by the Central Reference Laboratory (Iran's CDC, Tehran, Iran). Random samples were also sent to the Central Reference Laboratory to check for the accuracy of measurements and, if significant deviations were flagged, the results were discarded.

## Definition of outcome measures

Recruited participants were considered to have CHD if they answered "yes" to the following question: "Have you ever been told by a physician or a health care professional that you have coronary heart disease/diseases of heart vessels?" Participants were specifically asked on being told about the diagnosis of "myocardial infarction" or "heart attack" as the follow-up ascertainments to answering "yes" this question. Previous studies confirmed the accuracy of this approach for epidemiological assessments [16-19]. Participants were adjudged to have selfreported chronic stable angina pectoris if they answered "yes" to all of the following 4 questions: 1) "Have you ever had feelings of pain, discomfort, pressure or tightness running on the center of your chest or just below your breastbone (sternum)?"; 2) "Have you ever experienced chest pain while you are walking fast or slow or walking up a hill?"; 3) "Does the pain relieve with changing positions (i.e., standing, seating, walking slowly) or by taking using sublingual pills?"; 4) "Does the pain relieve in less than 10 minutes?". By definition, self-reported diagnosis of heart disease other than ischemic heart disease including the rheumatic valvular heart disease, and other types of cardiovascular diseases including stroke or the intracranial hemorrhage are not included in the present study. We also utilized related components of the Global Physical Activity Questionnaire to define intensity of physical activity according to a previous SuRFNCD-2011 study [20]. Hypertension was defined as $\mathrm{SBP} \geq 130 \mathrm{~mm} \mathrm{Hg}$ and/or DBP $\geq 85 \mathrm{~mm} \mathrm{Hg}$ or
treatment of previously diagnosed hypertension. Diabetes was defined according to the American Diabetes Association guidelines. The diagnosis of metabolic syndrome was based on the International Diabetes Federation criteria [21].

## Statistical analysis

Estimated national prevalence of self-reported CHD and chronic stable angina pectoris. The Stata software version 12.0 for Windows (Stata Corporation, College Station, Texas) was utilized to perform the complex sample survey analysis. Both the design weight (probability of the presence of each participant according to the design scheme) and post-sampling weights were utilized to extrapolate findings to the 2011 adult population of Iran. Post-sampling weights were generated according to Iran population in different strata of age (10-year strata: 20 to 29,30 to 39,40 to 49,50 to 59 , and 60 to 69), sex, and area of residence (urban, rural). Results of the national census of Iran in 2011 (available at the website of Statistical Centre of Iran) were used for the extrapolation. A total of 8,191 participants were included in the analysis and the prevalence of self-reported CHD and angina pectoris were extrapolated to the overall Iranian adult population older than 20 years ( $n=47,950,243$ ).

Clustering of the self-reported CHD and chronic stable angina pectoris risk factors. Factor analysis was carried out using the SPSS software version 21.0 for Windows (IBM Inc., Armonk, New York). Factor analysis synthesizes a large set of variables into a smaller set of factors or components to facilitate simpler interpretations. These factors comprise sets of variables with optimal correlation to the overall variances of the factors. Principle component analysis with varimax rotation method was utilized and factors with eigenvalues $>1.0$ were extracted. Loading values $>0.32$ were considered statistically significant; this cutoff point reveals that variables have at least $10 \%$ shared variance with factors. Variables with greater loadings values indicate higher correlations with specific factors. Factor analysis generates factor scores indicating the rank of each individual for each factor. The factor score will not be calculated for those participants with missing data for any variables of that particular factor. These scores are standardized values with mean of 0 and SD of 1 .

Considering the theoretical concepts of the cardiometabolic risk factors, the following case-mix variables were selected for the factor analysis: waist circumference, BMI, SBP, DBP, FPG, HDL-C, LDL-C, triglyceride, total cholesterol, and tobacco smoking. All variables except for the SBP and DBP violated the assumption of normality; in order to maintain the normal distribution of variables, the natural logarithmic transformation was used. Among all participants, 3,464 had nonmissing data for the interested variables and were included in the factor analysis.

The participants with a factor score in the top quartile were considered as being high risk for the extracted factors. Baseline and multivariable adjusted models of logistic

TABLE 1. Estimated prevalence of self-reported CHD and chronic stable angina pectoris in the SuRFNCD-2011 ( $\mathrm{n}=8,191$ )

|  | Self-Reported CHD |  | Chronic Stable Angina Pectoris |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Estimated Number (Thousands) | Prevalence (95\% CI) | Estimated Number (Thousands) | Prevalence (95\% CI) |
| Age, yrs |  |  |  |  |
| 20-29 | 288 | 1.7 (0.7-2.6) | 759 | $4.4(3.3-5.6)$ |
| 30-39 | 321 | 2.6 (1.8-3.3) | 777 | 6.2 (4.7-7.7) |
| 40-49 | 620 | 6.9 (5.2-8.7) | 948 | 10.6 (8.7-12.5) |
| 50-59 | 736 | 11.9 (10.3-13.4) | 757 | 12.2 (10.4-14.0) |
| 60-69 | 558 | 17.4 (15.4-19.3) | 486 | 15.2 (12.9-17.5) |
| Sex |  |  |  |  |
| Male | 1,149 | 4.7 (3.9-5.6) | 1,426 | 5.9 (4.8-7.0) |
| Female | 1,374 | 5.7 (4.9-6.5) | 2,301 | 9.6 (8.4-10.9) |
| Residential area |  |  |  |  |
| Urban | 1,973 | 5.6 (4.9-6.3) | 2,784 | 7.9 (6.8-9.0) |
| Rural | 550 | 4.3 (3.4-5.2) | 943 | 7.4 (6.1-8.7) |
| Total | 2,523 | 5.3 (4.6-5.9) | 3,727 | 7.7 (4.6-8.7) |

CHD, coronary heart disease; Cl , confidence interval; Surveillance of Risk Factors of NonCommunicable Diseases.
regression analysis with predicting continuous and categorical variables were utilized to assess whether each factor is correlated with elevated risks of self-reported CHD and chronic stable angina pectoris. The factor analysis and logistic regression analysis models are limited to the Iranian adults aged more than 40 years because of low prevalence of CHD or angina pectoris in individuals younger than 40 years.

## RESULTS

## Estimated national prevalence of self-reported CHD and chronic stable angina pectoris

The estimated national prevalence of self-reported CHD was 5.3\% ( $95 \%$ confidence interval [CI]: 4.6 to 5.9),
corresponding to the total figure of 2.523 million Iranian adults aged 20 to 69 years old. The prevalence of CHD was higher among older age classes, urban areas, and women (Table 1). The calculated prevalence of chronic stable angina pectoris was $7.7 \%$ ( $95 \%$ CI: 4.6 to 8.7 ), which translates to the estimated number of 3.727 Iranian adults with chronic stable angina pectoris residing in the country in 2011. Older people, urban residents, and women recorded higher prevalence of angina pectoris than their younger, rural, and male counterparts, respectively (Table 1). Distribution of common cardiometabolic risk factors by age and sex in the SuRFNCD-2011 database is displayed in Table 2.

## Clustering of the self-reported CHD and chronic stable angina pectoris risk factors

According to the Bartlett test of sphericity, the present data were suitable for performing factor analysis ( $p<0.001$ ). Significant correlations were observed in the univariate analysis between each of the studied variables except for the HDL-C and DBP (Online Table 1). Tobacco smoking was defined and included in the factor analysis as a categorical variable with 3 layers (i.e., never smokers, past smokers, and current smokers). Factor analysis of 10 variables resulted in extracting 4 factors with the eigenvalue scores $>1.0$. The first factor (i.e., hypercholesterolemia and hypertriglyceridemia) comprised total cholesterol, triglyceride, and LDL-C, which explained approximately $26.4 \%$ of the variance in the measured variables. The second factor (i.e., central obesity and tobacco smoking) included BMI, waist circumference, and tobacco smoking and explained $16.9 \%$ of total variance. The SBP and DBP were loaded as the third factor (i.e., raised blood pressure) and explained $14.2 \%$ of the total variance. The fourth factor (i.e., dyslipidemia, tobacco smoking, and hyperglycemia) was determined by FPG, triglyceride, HDL-C, and tobacco smoking and explained $12.5 \%$ of the total variance. These

TABLE 2. Distribution of cardiometabolic risk factors by age and sex in the SuRFNCD 2011 ( $\mathrm{n}=8,191$ )

|  | $\begin{aligned} & \text { FPG } \\ & (\mathrm{mg} / \mathrm{dl}) \end{aligned}$ | $\begin{aligned} & \text { LDL-C } \\ & (\mathrm{mg} / \mathrm{dl}) \end{aligned}$ | Triglyceride (mg/dl) | $\begin{aligned} & \text { HDL-C } \\ & (\mathrm{mg} / \mathrm{dl}) \end{aligned}$ | Total Cholesterol (mg/dl) | $\begin{gathered} \mathrm{BMI} \\ \left(\mathrm{~kg} / \mathrm{m}^{2}\right) \end{gathered}$ | Waist <br> Circumference (cm) | $\begin{gathered} \text { SBP } \\ (\mathrm{mm} \mathrm{Hg}) \end{gathered}$ | $\begin{gathered} \text { DBP } \\ (\mathrm{mm} \mathrm{Hg}) \end{gathered}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Age, yrs |  |  |  |  |  |  |  |  |  |
| 20-29 | $87.4 \pm 15.6$ | $92.1 \pm 26.1$ | $121.8 \pm 70.5$ | $44.9 \pm 10.9$ | $167.3 \pm 37.6$ | $24.1 \pm 4.8$ | $81.2 \pm 13.0$ | $115.8 \pm 13.4$ | $73.8 \pm 9.5$ |
| 30-39 | $91.3 \pm 20.6$ | $97.6 \pm 25.6$ | $140.5 \pm 95.4$ | $45.0 \pm 11.1$ | $176.9 \pm 42.1$ | $26.5 \pm 5.2$ | $87.9 \pm 13.3$ | $118.8 \pm 14.0$ | $77.5 \pm 10.9$ |
| 40-49 | $101.4 \pm 40.8$ | $104.2 \pm 26.6$ | $153.7 \pm 98.7$ | $45.8 \pm 11.7$ | $187.3 \pm 40.3$ | $27.7 \pm 5.2$ | $91.6 \pm 13.4$ | $125.8 \pm 18.0$ | $80.9 \pm 11.7$ |
| 50-59 | $108.9 \pm 43.4$ | $109.9 \pm 27.7$ | $172.0 \pm 106.6$ | $44.9 \pm 11.5$ | $195.2 \pm 43.2$ | $27.7 \pm 5.3$ | $94.0 \pm 14.3$ | $133.4 \pm 19.8$ | $83.0 \pm 11.8$ |
| 60-69 | $113.2 \pm 48.7$ | $108.8 \pm 29.2$ | $157.7 \pm 85.6$ | $46.1 \pm 12.3$ | $193.7 \pm 46.2$ | $27.5 \pm 5.2$ | $94.4 \pm 14.6$ | $139.1 \pm 22.5$ | $83.5 \pm 13.0$ |
| Sex |  |  |  |  |  |  |  |  |  |
| Male | $100.5 \pm 36.0$ | $101.0 \pm 26.5$ | $155.6 \pm 94.5$ | $41.7 \pm 10.4$ | $179.2 \pm 41.1$ | $25.5 \pm 4.8$ | $89.7 \pm 14.6$ | $127.4 \pm 17.7$ | $78.9 \pm 11.5$ |
| Female | $102.5 \pm 40.2$ | $105.0 \pm 28.7$ | $149.8 \pm 95.8$ | $47.5 \pm 11.6$ | $189.4 \pm 44.5$ | $27.1 \pm 5.6$ | $88.7 \pm 14.7$ | $124.6 \pm 20.9$ | $79.4 \pm 12.2$ |
| Total | $101.8 \pm 38.7$ | $103.5 \pm 28.0$ | $152.0 \pm 95.3$ | $45.3 \pm 11.5$ | $185.6 \pm 43.6$ | $26.5 \pm 5.3$ | $89.1 \pm 14.7$ | $125.7 \pm 19.7$ | $79.2 \pm 11.9$ |

[^0]TABLE 3. Factor loadings of variables in 4 extracted factors

|  | Factor |  |  |  |
| :--- | ---: | ---: | ---: | ---: |
|  | 1 | 2 | 3 | 4 |
| FPG | 0.182 | 0.266 | 0.085 | $\mathbf{0 . 3 4 6}$ |
| LDL-C | $\mathbf{0 . 9 1 1}$ | 0.052 | 0.051 | -0.017 |
| Triglyceride | $\mathbf{0 . 3 6 4}$ | 0.239 | 0.063 | $\mathbf{0 . 7 1 7}$ |
| HDL-C | 0.299 | -0.011 | -0.012 | $-\mathbf{0 . 7 9 9}$ |
| Total cholesterol | $\mathbf{0 . 9 5 3}$ | 0.090 | 0.065 | 0.041 |
| BMI | 0.064 | $\mathbf{0 . 8 5 3}$ | 0.104 | 0.094 |
| Waist circumference | 0.042 | $\mathbf{0 . 7 9 4}$ | 0.091 | 0.226 |
| SBP | 0.053 | 0.086 | $\mathbf{0 . 9 1 3}$ | 0.084 |
| DBP | 0.060 | 0.102 | $\mathbf{0 . 9 1 3}$ | 0.007 |
| Tobacco smoking | -0.061 | $\mathbf{- 0 . 4 9 3}$ | -0.013 | $\mathbf{0 . 4 3 9}$ |
| \% of variance explained | $26.4 \%$ | $16.9 \%$ | $14.2 \%$ | $12.5 \%$ |
| by each factor |  |  |  |  |

Data is correlation coefficients between variable and factor. Factor loadings $>0.32$ are bold.
Abbreviations as in Table 2.

4 factors accounted for the combined $70.0 \%$ of total variance (Table 3). We alternatively measured progressive levels of tobacco exposure among people with available pack-year data and rerun the factor analysis by calculating a multilevel variable using the quintiles of tobacco smoking pack-years; however, factor loadings were similar across both definitions of tobacco smoking (data not shown).

Scores on the first extracted factor (i.e., hypercholesterolemia and hypertriglyceridemia) ranged between -4.9 and 3.0. A total of 3,465 participants $>40$ years old had available data to determine the first factor, from those 866 individuals ( $25.0 \%$ ) at the upper quartile (score $>0.683$ ) were designated as the high-risk class. Scores for the second extracted factor (i.e., central obesity and tobacco smoking) ranged between -4.1 and 3.4. For the third factor (i.e., raised blood pressure), scores ranged between -3.1 and 4.8 . In the fourth extracted factor (i.e., dyslipidemia, tobacco smoking, and hyperglycemia), the scores ranged between -3.0 to 4.3. The mean values of variables that formed 4 extracted factors for the low- and high-risk classes are presented in the Table 4.

By classification of study participants into the low- and high-risk classes of factors, different models of logistic regression analysis were run to identify independent risk factors for the presence of self-reported CHD and angina pectoris using continuous and categorical models of extracted factors. The results of logistic regression analysis are summarized in Table 5. The raised blood pressure (i.e., third extracted factor) was a significant correlate of the presence of self-reported CHD (baseline continuous model: $\mathrm{p}=0.010$; odds ratio $=1.19 ; 95 \%$ CI: 1.04 to 1.35 ; adjusted continuous model: $\mathrm{p}=0.014$; odds ratio $=1.18$; $95 \%$ CI: 1.03 to 1.35 ; respectively). The first, second, and the fourth factors-which were the respective indicators of hypercholesterolemia and hypertriglyceridemia; central obesity and tobacco smoking; and dyslipidemia, tobacco
smoking, and hyperglycemia-were not significantly associated with the presence of self-reported CHD or angina pectoris (Table 5).

Table 6 summarizes associations of various lifestyle/ behavioral, clinical, and laboratory cardiometabolic risk factors with the presence of self-reported CHD and chronic stable angina pectoris in the SuRFNCD-2011 using separate models of univariate logistic regression analysis. Importantly, significantly lower odds for the diagnosis of self-reported CHD and/or chronic stable angina pectoris were noted among people with their diets containing higher weekly units of vegetables/fruits, and those with increased, intense, and vigorous physical activities. Conversely, self-reported CHD/chronic stable angina pectoris was more frequently observed among people who engaged in a more sedentary lifestyle, consumed fewer weekly units of vegetables and fruits, had increased waist circumference, waist/hip ratio, triglyceride, and total cholesterol, and were diagnosed with diabetes, hypertension, or metabolic syndrome (Table 6).

## DISCUSSION

In the present study, nationally representative data from the periodic SuRFNCD-2011 survey was analyzed to estimate national prevalence of self-reported CHD and chronic stable angina pectoris in the adult population of Iran. Complex sample survey analysis of the WHO STEP-WISE SuRFNCD-2011 estimated national prevalence of $5.3 \%$ (95\% CI: 4.6 to 5.9 ) for CHD and $7.7 \% ~(95 \% \mathrm{CI}=4.6$ to 8.7) for chronic stable angina pectoris in the year 2011 in Iran. The presence of self-reported CHD and angina pectoris were more frequent among the elder, urbanized, and female Iranian adults. Factor analysis indicated people with

TABLE 4. Mean $\pm$ SD of variables in 4 extracted factors among the low- and high-risk groups

|  | Low-Risk Group <br> (First Quartile) | High-Risk Group <br> (Fourth Quartile) |
| :---: | :---: | :---: |
| Factor 1 | $\mathrm{n}=2,599$ | $\mathrm{n}=866$ |
| LDL-C, $\mathrm{mg} / \mathrm{dl}$ | $97.0 \pm 20.7$ | $141.4 \pm 19.8$ |
| Triglyceride, $\mathrm{mg} / \mathrm{dl}$ | $149.2 \pm 87.1$ | $205.27 \pm 118.10$ |
| Total cholesterol, $\mathrm{mg} / \mathrm{dl}$ | $174.8 \pm 28.8$ | $246.8 \pm 35.5$ |
| Factor 2 | $\mathrm{n}=2,598$ | $\mathrm{n}=866$ |
| BMI, $\mathrm{kg} / \mathrm{m}^{2}$ | $25.5 \pm 3.6$ | $34.1 \pm 4.5$ |
| Waist circumference, cm | $89.3 \pm 11.1$ | $108.0 \pm 11.3$ |
| Tobacco smoking | $\mathrm{N} / \mathrm{A}$ | $\mathrm{N} / \mathrm{A}$ |
| Factor 3 | $\mathrm{n}=2,600$ | $\mathrm{n}=866$ |
| SBP, mm Hg | $125.3 \pm 13.7$ | $160.6 \pm 17.7$ |
| DBP, mm Hg | $78.0 \pm 8.4$ | $97.5 \pm 9.5$ |
| Factor 4 | $\mathrm{n}=2,599$ | $\mathrm{n}=867$ |
| FPG, mg/dl | $101.6 \pm 34.4$ | $129.6 \pm 62.6$ |
| Triglyceride, mg/dl | $132.5 \pm 55.5$ | $255.4 \pm 135.9$ |
| HDL-C, mg/dl | $49.1 \pm 10.9$ | $34.9 \pm 7.0$ |
| Tobacco smoking | $\mathrm{N} / \mathrm{A}$ | $\mathrm{N} / \mathrm{A}$ |

N/A, not available; other abbreviations as in Table 2.

TABLE 5. Baseline and multivariable adjusted logistic regression models of the associations among extracted risk categories of factor analysis and self-reported CHD and chronic stable angina pectoris in the SuRFNCD-2011 ( $\mathrm{n}=8,191$ )

| Predictor | Self-Reported CHD |  | Chronic Stable Angina Pectoris |  |
| :---: | :---: | :---: | :---: | :---: |
|  | p Value | OR (95\% CI) | $p$ Value | OR (95\% CI) |
| Baseline Models |  |  |  |  |
| Model I ( $\mathrm{n}=3,465$ ) |  |  |  |  |
| First factor: hypercholesterolemia and hypertriglyceridemia |  |  |  |  |
| Continuous | 0.761 | 1.02 (0.88-1.18) | 0.408 | 1.05 (0.93-1.20) |
| Categorical | 0.615 | 1.03 (0.91-1.18) | 0.293 | 1.06 (0.95-1.19) |
| First quartile, low risk |  |  |  |  |
| Second quartile | 0.962 | 0.99 (0.65-1.50) | 0.727 | 0.94 (0.65-1.37) |
| Third quartile | 0.823 | 1.05 (0.69-1.58) | 0.546 | 1.12 (0.78-1.60) |
| Fourth quartile, high risk | 0.664 | 1.09 (0.73-1.65) | 0.437 | 1.15 (0.81-1.64) |
| Model II ( $\mathrm{n}=3,464$ ) |  |  |  |  |
| Second factor: central obesity and tobacco smoking |  |  |  |  |
| Continuous | 0.630 | 1.04 (0.89-1.21) | 0.933 | 1.01 (0.88-1.15) |
| Categorical | 0.876 | 0.99 (0.87-1.13) | 0.845 | 0.99 (0.88-1.11) |
| First quartile, low risk |  |  |  |  |
| Second quartile | 0.044 | 1.51 (1.01-2.25) | 0.992 | 1.00 (0.70-1.42) |
| Third quartile | 0.735 | 0.93 (0.59-1.44) | 0.380 | 0.85 (0.59-1.22) |
| Fourth quartile, high risk | 0.515 | 1.15 (0.75-1.75) | 0.937 | 1.01 (0.71-1.44) |
| Model III ( $\mathrm{n}=3,466$ ) |  |  |  |  |
| Third factor: raised blood pressure |  |  |  |  |
| Continuous | 0.007 | 1.21 (1.05-1.39) | 0.084 | 1.11 (0.99-1.26) |
| Categorical | 0.010 | 1.19 (1.04-1.35) | 0.193 | 1.08 (0.96-1.21) |
| First quartile, low risk |  |  |  |  |
| Second quartile | 0.775 | 0.94 (0.59-1.47) | 0.357 | 0.84 (0.58-1.22) |
| Third quartile | 0.078 | 1.44 (0.96-2.18) | 0.552 | 1.11 (0.78-1.58) |
| Fourth quartile, high risk | 0.043 | 1.53 (1.01-2.31) | 0.379 | 1.17 (0.82-1.66) |
| Model IV ( $\mathrm{n}=3,466$ ) |  |  |  |  |
| Fourth factor: dyslipidemia, tobacco smoking, and hyperglycemia |  |  |  |  |
| Continuous | 0.490 | 0.95 (0.82-2.10) | 0.807 | 0.93 (0.52-1.64) |
| Categorical | 0.552 | 0.96 (0.84-1.09) | 0.803 | 0.99 (0.88-1.10) |
| First quartile, low risk |  |  |  |  |
| Second quartile | 0.754 | 1.07 (0.71-1.60) | 0.421 | 0.86 (0.60-1.24) |
| Third quartile | 0.387 | 1.19 (0.80-1.78) | 0.462 | 1.14 (0.81-1.61) |
| Fourth quartile, high risk | 0.389 | 0.83 (0.54-1.27) | 0.432 | 0.86 (0.60-1.24) |
| Adjusted Model ( $\mathrm{n}=3,464$ ) |  |  |  |  |
| Continuous |  |  |  |  |
| First factor: hypercholesterolemia and hypertriglyceridemia | 0.759 | 1.02 (0.88-1.18) | 0.405 | 1.06 (0.93-1.20) |
| Second factor: central obesity and tobacco smoking | 0.561 | 1.05 (0.90-1.22) | 0.874 | 1.01 (0.89-1.15) |
| Third factor: raised blood pressure | 0.006 | 1.21 (1.05-1.39) | 0.086 | 1.11 (0.98-1.26) |
| Fourth factor: dyslipidemia, tobacco smoking, and hyperglycemia | 0.492 | 0.95 (0.82-1.10) | 0.386 | 0.94 (0.83-1.07) |
| Categorical |  |  |  |  |
| First factor: hypercholesterolemia and hypertriglyceridemia | 0.579 | 1.04 (0.91-1.18) | 0.326 | 1.06 (0.94-1.19) |
| Second factor: central obesity and tobacco smoking | 0.888 | 0.99 (0.87-1.13) | 0.775 | 0.98 (0.88-1.10) |
| Third factor: raised blood pressure | 0.014 | 1.18 (1.03-1.35) | 0.227 | 1.07 (0.96-1.20) |
| Fourth factor: dyslipidemia, tobacco smoking, and hyperglycemia | 0.649 | 0.97 (0.85-1.10) | 0.823 | 0.99 (0.88-1.10) |
| For all models, n is the number of people included in the analysis. Self-reported CHD or chronic stable angina pectoris were considered as dependent variables in all logistic regression models. OR, odds ratio; other abbreviations as in Table 1. |  |  |  |  |

TABLE 6. Associations of lifestyle/behavioral, clinical, and laboratory risk factors with the presence of self-reported CHD and chronic stable angina pectoris in the SuRFNCD-2011 ( $\mathrm{n}=8,191$ )

| Variable | n* | Self-Reported CHD |  | Chronic Stable Angina Pectoris |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | $p$ Value ${ }^{\dagger}$ | OR (95\% CI) ${ }^{\dagger}$ | p Value ${ }^{\dagger}$ | OR (95\% CI) ${ }^{\dagger}$ |
| Lifestyle/behavioral |  |  |  |  |  |
| Fruit consumption, weekly units, tertiles | 8,191 | 0.003 | 0.85 (0.77-0.95) | $<0.001$ | 0.72 (0.66-0.79) |
| Reference |  |  |  |  |  |
| Second |  | 0.200 | 0.89 (0.76-1.06) | $<0.001$ | 0.65 (0.56-0.75) |
| Third |  | 0.002 | 0.71 (0.57-0.88) | $<0.001$ | 0.56 (0.47-0.67) |
| Vegetable consumption, weekly units, tertiles | 8,191 | 0.538 | 0.97 (0.88-1.07) | 0.001 | 0.87 (0.81-0.94) |
| Reference |  |  |  |  |  |
| Second |  | 0.128 | 0.86 (0.72-1.04) | <0.001 | 0.76 (0.66-0.88) |
| Third |  | 0.533 | 0.94 (0.78-1.13) | 0.001 | 0.77 (0.66-0.89) |
| Physical activity |  |  |  |  |  |
| Levels of PA, high/low | 8,191 | <0.001 | 0.72 (0.62-0.85) | $<0.001$ | 0.76 (0.67-0.87) |
| Intensity of PA, vigorous/nonvigorous | 8,191 | 0.001 | 0.73 (0.62-0.88) |  |  |
| Total PA, MET $\times \mathrm{min} /$ weeks, tertiles | 8,191 | <0.001 | 0.81 (0.74-0.89) | $<0.001$ | 0.87 (0.80-0.93) |
| Reference |  |  |  |  |  |
| Second |  | 0.029 | 0.82 (0.68-0.98) | 0.002 | 0.79 (0.68-0.92) |
| Third |  | $<0.001$ | 0.82 (0.68-0.98) | $<0.001$ | 0.76 (0.65-0.88) |
| Sedentary behavior, min/day, tertiles | 8,191 | 0.026 | 1.12 (1.01-1.24) | 0.766 | 1.01 (0.93-1.10) |
| Reference |  |  |  |  |  |
| Second |  | 0.987 | 1.00 (0.82-1.21) | 0.045 | 0.86 (0.74-1.00) |
| Third |  | 0.034 | 1.24 (1.02-1.51) | 0.854 | 1.01 (0.86-1.19) |
| Tobacco smoking | 7,613 | 0.335 | 1.06 (0.94-1.21) | 0.928 | 1.00 (0.89-1.13) |
| Never smokers |  |  |  |  |  |
| Past smokers |  | 0.074 | 1.44 (0.96-2.16) | 0.795 | 1.06 (0.70-1.59) |
| Current smokers |  | 0.564 | 1.08 (0.83-1.41) | 0.975 | 1.00 (0.79-1.28) |
| Clinical/laboratory |  |  |  |  |  |
| Waist circumference, cm | 8,191 | $<0.001$ | 1.02 (1.02-1.03) | $<0.001$ | 1.02 (1.01-1.02) |
| Waist-hip ratio | 8,191 | $<0.001$ | 10.19 (5.35-19.41) | $<0.001$ | 3.14 (1.80-5.47) |
| Triglyceride, mg/dl | 5,426 | 0.144 | 1.00 (1.00-1.01) | 0.046 | 1.00 (1.00-1.00) |
| HDL-C, mg/dl | 5,423 | 0.024 | 0.99 (0.98-1.00) | 0.710 | 1.00 (0.99-1.00) |
| LDL-C, mg/dl | 5,422 | <0.001 | 1.00 (1.00-1.00) | 0.004 | 1.00 (1.00-1.00) |
| Triglyceride-HDL-C ratio | 5,417 | 0.003 | 1.04 (1.01-1.06) | 0.068 | 1.02 (1.00-1.04) |
| Total cholesterol, mg/dl | 5,423 | 0.177 | 1.00 (1.00-1.00) | 0.007 | 1.00 (1.00-1.00) |
| Diabetes, yes/no | 5,403 | $<0.001$ | 2.83 (2.29-3.50) | $<0.001$ | 2.05 (1.71-2.45) |
| Hypertension, yes/no | 5,403 | $<0.001$ | 2.74 (2.22-3.39) | $<0.001$ | 1.77 (1.52-2.07) |
| Metabolic syndrome, yes/no | 5,403 | $<0.001$ | 2.62 (2.16-3.19) | $<0.001$ | 1.70 (1.46-1.97) |

MET, metabolic equivalent; PA, physical activity; other abbreviations as in Tables 1, 2, and 5.
*Denotes the number of people included in the analysis.
${ }^{\dagger}$ Presented data denote results of the univariate logistic regression models.
raised blood pressures are at the increased risk for selfreported diagnosis of CHD.

## Comparison of the estimated national prevalence of self-reported CHD and chronic stable angina pectoris with other regions

The Chinese National Diabetes and Metabolic Disorder Study reported the prevalence of self-reported CHD to be $0.6 \%$ ( $95 \%$ CI: 0.49 to 0.79 ) of the entire adult population who were at least 20 years of age, with increased
prevalence of CHD in the elders and men [22]. The selfreported CHD was present in as low as in $<0.01 \%$ of people aged between 20-29 years old and $0.1 \%$ of those aged between 30-39 years old [22]. It should be noted that prevalence of self-reported CHD in the 40 to 49,50 to 59 and 60 to 69 age categories were $0.3 \%, 1.1 \%$, and $1.4 \%$, respectively [22]. The corresponding figures of selfreported CHD in the Iranian adult population were $6.9 \%, 11.9 \%$, and $17.4 \%$, respectively. Based on the results of NHANES (National Health and Nutrition Examination Survey), the age-adjusted prevalence of self-reported CHD
in the U.S. adults older than 18 years is $6.0 \%$ ( $95 \% \mathrm{CI}: 5.9$ to 6.1) [23], demonstrating a significant decline from $6.7 \%$ ( $95 \%$ CI: 6.5 to 6.9 ) in 2006 ( p value for linear trend $<0.001$ ) [24]. Analyzed data from the 2013 CPRD GOLD (The UK Clinical Practice Research Datalink) database suggest that almost 2.3 people were living with some forms of CHD or angina pectoris in the United Kindgom, with hospital-diagnosed prevalence of CHD ranging from $2.1 \%$ to $4.5 \%$ across different regions; and $3.05 \%$ and $1.79 \%$ for angina pectoris in men and women, respectively [25]. The population-based INTERHEART study of cardiovascular risk and events in 17 low-, middle-, and high-income countries and comprising 388,796 community-dwelling participants recorded 1,736 total events (1.1\%) of myocardial infarction, with the low-income countries of Bangladesh, India, Pakistan, and Zimbabwe suffering the highest rates [26]. These data categorically attest that the estimated national prevalence of self-reported CHD and angina pectoris are alarmingly high in Iran, significantly more prevalent than the respective figures in China [22], the United Kingdom [25] and the average global estimates [26], but comparable to that of the U.S. adult population [24]. In the noncommunicable diseases database of India, the prevalence of angina pectoris according to the selfreport of diagnosed cases and the standardized measures of angina were $3.1 \%$ and $6.9 \%$, respectively [27]. Despite the self-reported designation of chronic stable angina pectoris as a major health concern in this SuRFNCD-2011 study, the estimated national prevalence of $7.7 \%$ is significantly higher than the Indian estimates [27] of selfreported or medically documented cases.

The diagnosis of self-reported CHD and chronic stable angina pectoris was more prevalent in urban settings, associated with lower weekly units of fruit and vegetable consumption, and was more frequently observed among people with decreased physical activity. These results are in accordance with previous reports, concluding that the sedentary pattern of lifestyle, lack of vigorous physical activity and characteristic environmental biohazards may contribute to the increased prevalence of CHD in urban settings [28,29]. Consistently, recent SuRFNCD-2011 data demonstrated that the prevalence of the metabolic syndrome and hypertension as the main risk factors of atherosclerosis and major adverse cardiac events are substantially higher in urban than in rural areas of Iran [21,30].

## Clustering of the self-reported CHD and chronic stable angina pectoris risk factors

A previous study on clustering of the cardiovascular disease risk factors was performed by Goodman et al. [31], which revealed 4 clusters among 11 cardiometabolic risk factors. Goodman et al. [31] investigated 1,578 healthy adolescents, and blood pressure was measured in only 212 participants. They also did not evaluate the correlations among clusters of cardiometabolic risk factors and the risk
of cardiovascular disease in their young study population [31]. Despite these methodological differences, results of their factor analysis were consistent with those of the present study and the variables were loaded in similarly extracted factors.

We found raised SBP and DBP to be significant factors underlying the self-reported diagnosis of CHD in SuRFNCD2011. This observation is consistent with a great body of evidence indicating hypertension as a major cardiometabolic risk factors [32]. A recent systematic review and meta-analysis of 123 studies with 613,815 participants showed blood pressure lowering to significantly reduce cardiovascular risk across various baseline blood pressure levels and comorbidities [33]. Results from this study provided strong support for the beneficial effects of lowering blood pressure to SBP $<130$ mm Hg and providing blood pressure-lowering treatment to individuals with a history of CHD [33]. Result from the global INTERHEART study in 52 countries recognized elevated blood pressure as 1 of the highest population-attributable risk factors of acute myocardial infarction [29].

In this SuRFNCD-2011 factor analysis, the first (i.e., LDL-C, triglyceride, and total cholesterol), second (i.e., BMI, waist circumference, and tobacco smoking), and fourth (i.e., FPG, triglyceride, HDL-C, and tobacco smoking) extracted factors did not predict the presence of selfreported CHD or chronic stable angina pectoris. Reverse causality could explain why the factors of dyslipidemia, central obesity, and hyperglycemia are not as strong as the factor of hypertension to predict self-reported diagnosis of CHD. It could be argued that people with hyperglycemia, hypercholesterolemia, or hypertriglyceridemia are more likely to have these risk factors treated and this may reduce strength of correlations among these risk factors and frequency of self-reported CHD or chronic stable angina pectoris.

In the present study, raised blood pressure had significant association with the presence of self-reported CHD in baseline and multivariable adjusted logistic regression models. It may be suggested that the adult population of Iran with self-reported CHD have poorer hypertension control compared with their management of hyperglycemia, dyslipidemia, and central obesity. The prevalence of awareness, treatment, and control of hypertension and prehypertension was recently investigated in the adult population of Iran in 2011 [30]. It was estimated that the rates of awareness, management, treatment, and control among people with hypertension were $43.2 \%$ ( $95 \%$ CI: 40.0 to 46.4 ), $40.3 \%$ ( $95 \%$ CI: 37.0 to 43.6 ), $34.8 \%$ ( $95 \%$ CI: 31.5 to 38.2 ), and $38.6 \%$ ( $95 \%$ CI: 33.1 to 44.2 ), suggesting that initiative strategies need to be undertaken to refine the status quo of hypertension awareness and control in Iran [30].

## Study limitations

First, the self-reported diagnosis of CHD and chronic stable angina pectoris leads to underestimation/overestimation of
the actual number of affected adult people and overall burden of disease in Iran [34]. Second, this study did not report the mortality rates of CHD and the proportion of all deaths attributable to CHD in the general adult population of Iran. These statistics are required to estimate the overall burden of CHD in the adult population of Iran. Last but not least, the cross-sectional design of SuRFNCD-2011 data is not suited to examine and determine the population-attributable fractions of cardiometabolic risk factors for self-reported diagnosis of CHD and chronic stable angina pectoris.

## Future directions

Despite the significant association of raised blood pressure with self-reported diagnosis of CHD, important correlations exist among a variety of modifiable lifestyle/behavioral risk factors and the presence of self-reported CHD/ chronic stable angina pectoris. Lower odds of self-reported CHD and chronic stable angina pectoris were observed among people who more frequently consumed fruits and vegetables and engaged in more vigorous physical activity. Higher odds of concurrent CHD and/or chronic stable angina pectoris were found among people with a more sedentary lifestyle and among those with their diets containing fewer average units of fruits and vegetables. These observations point to the enormous potential of active primary care preventative strategies for their roles in cardiometabolic risk reduction in our country, through counteracting deleterious effects of hypertension on the currently high prevalence of CHD among the general adult population of Iran.

## CONCLUSIONS

The current study reported high prevalence of self-reported CHD and chronic stable angina pectoris in the general adult population of Iran. Factor analysis of 10 anthropometric, laboratory and behavioral indices resulted in extracting 4 factors consisting of different sets of cardiometabolic risk variables. Various continuous and categorical models of logistic regression analysis recorded higher risks of self-reported CHD in people with raised blood pressures. In addition, we demonstrated significant associations among higher consumption units of fruit/ vegetables and healthier physical activity habits with lower odds for the presence of self-reported CHD or chronic stable angina pectoris, suggesting opportunities for lifestyle and/or behavioral change strategies to improve cardiometabolic profile through reversal of these modifiable risk factors among the adult population of Iran. The national CHD preventative programs are therefore urged to target reducing the prevalence of hypertension and introduce active primary care preventative measures to reduce the overall acute burden of CHD currently present in Iran. Future cross-sectional and longitudinal studies from the national SuRFNCD database are encouraged to examine temporal associations among these cardiometabolic risk
factors and self-reported diagnosis of CHD/chronic stable angina pectoris in order to estimate the populationattributable fractions of each specific risk factor to the overall risk of CHD and cardiovascular diseases in Iran.

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## APPENDIX

ONLINE TABLE 1. Correlation coefficients among the cardiometabolic risk factors

|  | FPG | LDL-C | Triglyceride | HDL-C | Total Cholesterol | BMI | Waist Circumference | SBP | DBP |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| LDL-C | 0.101* |  |  |  |  |  |  |  |  |
| Triglyceride | 0.281* | 0.208* |  |  |  |  |  |  |  |
| HDL-C | -0.075* | 0.167* | -0.379* |  |  |  |  |  |  |
| Total cholesterol | 0.146* | 0.816* | 0.399* | 0.226* |  |  |  |  |  |
| BMI | 0.162* | 0.133* | 0.254* | $-0.051^{\dagger}$ | 0.159* |  |  |  |  |
| Waist circumference | 0.178* | 0.132* | 0.268* | -0.131* | 0.134* | 0.615* |  |  |  |
| SBP | 0.164* | 0.088* | 0.151* | -0.054 ${ }^{\dagger}$ | 0.126* | 0.167* | 0.172* |  |  |
| DBP | 0.065* | 0.118* | 0.126* | -0.020 | 0.123* | 0.196* | 0.168* | 0.696* |  |
| Tobacco smoking | -0.065* | $-0.055^{\dagger}$ | -0.002 | -0.164* | -0.086* | -0.189* | -0.078* | $-0.038^{\dagger}$ | -0.062* |

BMI, body mass index; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure.
*p $<0.001$.
${ }^{\dagger} 0.01>\mathrm{p} \geq 0.001$.


[^0]:    Values represent mean $\pm$ SD of each cardiometabolic risk factor.
    BMI, body mass index; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure.

