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Obesity measures and heart disease risks—the risks of overtraining and being underweight

Benjamin Hubert[1] [B-D], Aroued Khelifi[2] [E-G], Paul Matthieu[1] [F], Hakan Lane[1] [A-F], Shreyansh Shailaan[3] [E-G], Samuel Engst[4] [E-G], Charalambos Plousiou[5] [F-G], April Htoon[6] [F-G], Huda Amer[6] [F-G]

[1] Johannes Gutenberg University, Mainz, Germany
[2] University of Sousse, Higher Institute of Management of Sousse, Department of Business Intelligence, Sousse, Tunisia
[3] Sir Seewoosagur Ramgoolam Medical College, Department of Public Health, Vacoas-Phoenix, Mauritius
[4] Stockholm Technological Institute, Department of Health Data Analytics, Stockholm, Sweden
[5] National and Kapodistrian University of Athens, Athens, Greece
[6] Atilim University, Faculty of Medicine, Ankara, Turkey

Original article

Abstract

Purpose: Obesity and abnormal fat distribution are major components of the Cardiovascular–Kidney–Metabolic (CKM) syndrome and are strongly associated with cardiovascular disease (CVD). However, the comparative performance of different anthropometric measures and the modifying role of physical activity remain incompletely defined. We aimed to examine the associations between body mass index (BMI), body fat percentage (BFP), waist-to-height ratio (WHtR), physical activity, and prevalent CVD in a large population-based cohort.

Materials and methods: We conducted a cross-sectional analysis of UK Biobank participants aged 40–69 years. After excluding individuals with missing data, N = 489909 participants were included. Prevalent CVD was defined as a history of angina, myocardial infarction, or stroke at baseline assessment. Multivariable logistic regression models were used to evaluate associations between anthropometric measures and CVD, adjusting for age, sex, smoking status, and major comorbidities. Physical activity was analyzed by type and duration. Results are reported as odds ratios (OR) with 95% confidence intervals (CI).

Results: BMI showed a U-shaped association with CVD, with higher prevalence observed at both low (<18.5 kg/m^2) and high (>25 kg/m^2) values. WHtR was positively associated with CVD across its range, whereas BFP provided little additional predictive value after adjustment. Moderate physical activity was associated with lower odds of CVD, while very high volumes of vigorous activity showed no additional benefit and were associated with slightly higher CVD prevalence.

Keywords

- cardiovascular disease
- body mass index
- waist-to-height ratio
- physical activity
- CKM syndrome
- obesity

Contribution

A – Preparation of the research project
B – Assembly of data
C – Conducting of statistical analysis
D – Interpretation of results
E – Manuscript preparation
F – Literature review
G – Revising the manuscript

Corresponding author

Hakan Lane
e-mail: Hakan.Lane@nightingaleheart.com
Johannes Gutenberg University
Mainz, Germany

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Conclusions: In this large cross-sectional study, BMI and WHtR were robust markers associated with prevalent CVD, while BFP added limited incremental value. Moderate physical activity was associated with lower CVD prevalence, whereas very high volumes of vigorous self-reported activity did not confer further benefit. These findings support the use of simple anthropometric measures in CKM risk stratification and reinforce the importance of balanced physical activity.

Introduction

Cardiovascular disease (CVD) is the leading cause of global mortality, with obesity emerging as a critical modifiable risk factor.[1] Global prevalence of obesity, defined by elevated body mass index (BMI ≥30 kg/m^2),[1] has tripled since 1975, paralleling the rising burden of CVD.[2] While epidemiological studies associate higher BMI with increased CVD risk, pathophysiological mechanisms underlying this relationship remain multifaceted.

A J-shaped relationship between BMI and CVD mortality has been demonstrated, with both underweight (BMI <18.5 kg/m^2) and obesity (BMI ≥30 kg/m^2) in adults conferring elevated risks.[3] Excess adiposity drives systemic inflammation, characterized by elevated circulating levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and C-reactive protein (CRP), which promote endothelial dysfunction and atherosclerosis.[4] Adipose tissue, particularly visceral fat, secretes bioactive molecules (adipokines) that disrupt insulin signaling pathways, leading to hyperinsulinemia and insulin resistance—a precursor to metabolic syndrome and CVD.[5] Dysregulated adipokine production (e.g., reduced adiponectin and elevated leptin) exacerbates oxidative stress and vascular remodeling, accelerating coronary artery disease.[6]

Context and significance

Lifestyle factors, including poor dietary habits and physical inactivity, are drivers of BMI elevation. Diets high in saturated fats and refined carbohydrates promote visceral adiposity and chronic low-grade inflammation, while sedentary behavior reduces energy expenditure, compounding metabolic dysregulation.[7] Conversely, sustained weight loss through caloric restriction and regular exercise improves endothelial function and reduces inflammatory biomarkers, commenting on the potential reversibility of obesity-related CVD risk.[8] Despite the known risks of obesity, a paradoxical phenomenon has been observed. "Obesity paradox", whereby overweight or mildly obese individuals (BMI 25–35 kg/m^2) exhibit lower mortality rates following acute CVD events compared to normal-weight counterparts.[9] This paradox may reflect limitations in BMI as a sole adiposity marker, as it fails to distinguish between lean mass and fat distribution. For instance, visceral adiposity, quantified by waist-to-hip ratio, shows a stronger correlation with CVD outcomes than BMI alone.[10] Additionally, confounding factors such as smoking, undiagnosed malignancies, or cardiotoxic treatments in normal-weight populations may skew mortality data. There has also been an interrelationship now formally recognized as Cardiovascular-Kidney-Metabolic (CKM) syndrome, a systemic disorder driven by the pathophysiological interplay of obesity, metabolic dysregulation, and cardiovascular disease.[11]

Research problems and questions

There is no consensus on optimal BMI thresholds for CVD risk across diverse populations, given genetic, cultural, and environmental variability.[12] Longitudinal evidence on how body composition changes affect CVD progression remains limited.

This study seeks to address the following questions:

1. Which anthropometric measure most strongly correlates with CVD risk?
2. What type of physical activity is most effective in reducing this risk?
3. At what BMI levels does being underweight or overweight increase the likelihood of CVD?

Objective and hypothesis

The aim of this study is to clarify the relationships between anthropometric indices, physical activity, and CVD risk. Through a comparative literature analysis, we evaluate which indicators and interventions are most predictive and protective. We hypothesize that visceral adiposity measures (e.g., waist-to-hip ratio) better predict CVD risk than BMI and that moderate-intensity aerobic activity provides the most significant risk reduction.

Importance of study

Identifying stronger predictors of CVD beyond BMI and evaluating the most effective types of physical activity may enhance public health strategies and clinical guidelines. This study contributes to more precise CVD risk stratification and targeted prevention, especially in diverse global populations.

Obesity indicators

Obesity is a complex and multifactorial condition, characterized by an excessive accumulation of body fat that poses health risks. To assess and quantify obesity, several anthropometric indicators are employed in both clinical and epidemiological settings. This study focuses on three primary indicators: Body Mass Index (BMI), Body Fat Percentage, and Waist-to-Hip Ratio (WHR). Each of these has its theoretical foundation, measurement methodology, and limitations.

Body Mass Index (BMI)

BMI is a widely used screening tool that classifies individuals into categories such as underweight, normal weight, overweight, and obese. It is calculated as follows:

$$\text{BMI} = \frac{\text{weight [kg]}}{\text{height [m]}^2}$$

The World Health Organization (WHO) classification is as follows: "underweight (BMI < 18.5), normal weight (18.5–24.9), overweight (25–29.9), and obese (≥30)."[13] Despite its ease of use and low cost, BMI does not differentiate between fat and lean body mass. For example, muscular individuals may be misclassified as overweight, while those with low muscle mass but high fat levels may be misclassified as normal weight.[14]

Body Fat Percentage (BFP)

BFP measures the proportion of body mass composed of fat. Gold standard methods for determining BFP include Dual-Energy X-ray Absorptiometry (DEXA), hydrostatic weighing, and air displacement plethysmography.[15] While these methods are precise, they are often expensive and less accessible for routine use. More commonly used alternatives like bioelectrical impedance analysis (BIA) and skinfold measurements have varying accuracy, with BIA showing errors ranging from 3–9% depending on conditions and device type.[16]

Waist-to-Height Ratio (WHtR)

WHtR is calculated by dividing waist circumference by height. A value greater than 0.5 is generally indicative of increased cardiovascular risk.[17] A large-scale cohort study of over 468,000 participants found that WHtR had a stronger linear association with cardiovascular events than BMI.[18]

Exercise

Physical inactivity is a major global health concern, contributing to approximately 3.2 million deaths annually and accounting for 6% of coronary heart disease (CHD) cases worldwide.[19] Regular physical activity is one of the most effective non-pharmacological strategies to prevent cardiovascular disease (CVD). It lowers blood pressure, body weight, LDL cholesterol, and improves insulin sensitivity and glucose metabolism.[20] A post-exercise reduction in blood pressure, known as post-exercise hypotension, occurs due to reductions in oxidative stress, inflammation, and vascular resistance.[19]

The American Heart Association recommends that adults perform at least 150 minutes of moderate-intensity aerobic exercise per week (e.g., brisk walking, light cycling) or 75 minutes of vigorous-intensity aerobic activity (e.g., running, swimming) per week, ideally spread across 3 to 5 sessions.[21] The most consistent benefits are observed when sessions last 30 to 60 minutes, performed 5 times per week for moderate intensity, or 3 times per week for vigorous intensity.[21] For additional benefits, 300 minutes of moderate or 150 minutes of vigorous activity weekly are recommended.[21]

High-Intensity Interval Training (HIIT), short bouts of intense activity followed by rest (e.g., 4 × 4 minutes at 85–95% of max heart rate with 3-minute active rest), has shown superior improvements in endothelial function, cardiorespiratory fitness, and insulin sensitivity, particularly in high-risk individuals.[22] In addition to aerobic activity, resistance (anaerobic) training performed 2–3 times per week improves muscle mass, metabolism, and reduces LDL cholesterol and triglycerides.[20] Combining aerobic and anaerobic training leads to the greatest reduction in cardiovascular risk factors.[20] Studies also suggest that accumulating exercise in bouts of at least 10 minutes throughout the day still confers measurable health benefits.[21]

Table 1. Key recommendations

Exercise type	Frequency	Duration per Session	Weekly target
Moderate aerobic	5 days/ week	30–60 minutes	≥150 minutes
Vigorous aerobic	3 days/ week	20–30 minutes	≥75 minutes
Resistance training	2–3 days/ week	30–45 minutes	≥75 minutes
HIIT	2–3 days/ week	15–25 minutes	≥75 minutes

Being underweight as a risk for heart disease

While obesity is a well-known risk factor for cardiovascular disease, being underweight also carries significant cardiovascular risk. Underweight individuals (BMI < 18.5 kg/m^2) are often overlooked in public health discussions. However, research shows that insufficient body weight can be linked to elevated CVD morbidity and mortality. Potential mechanisms include:

- Malnutrition and micronutrient deficiencies, which impair immune and cardiovascular function.
- Reduced muscle mass, leading to decreased metabolic reserves and cardiovascular stress under physical strain.
- Lower levels of protective fat that normally regulate inflammatory processes and produce essential hormones like leptin and adiponectin.[23]

A 2017 meta-analysis of 51 studies found a U-shaped association between BMI and CVD mortality, with the lowest risk observed in individuals with a BMI between 22–24.9 kg/m^2. Both underweight and obese individuals had significantly higher risks.[24] Moreover, underweight status has been correlated with arrhythmias, cardiac muscle atrophy, and hypotension, particularly in elderly or frail populations.[25] Further evidence suggests that underweight individuals are more vulnerable to sarcopenia, which compromises heart function during times of physiological stress and increases the risk of heart failure.[26]

Theoretical models linking obesity and cardiovascular disease

1. **Inflammatory Model.** Adipose tissue, especially visceral fat, acts as an endocrine organ, releasing pro-inflammatory cytokines such as TNF-α and IL-6. These molecules promote systemic inflammation, endothelial dysfunction, and atherogenesis, which are central to CVD pathogenesis.[27]
2. **Insulin Resistance Model.** Obesity leads to insulin resistance, which in turn contributes to a cluster of conditions known as metabolic syndrome (hyperglycemia, dyslipidemia, hypertension), all of which significantly increase CVD risk.[27]
3. **Lipotoxicity Model.** In obese individuals, excess free fatty acids accumulate in non-adipose tissues like the heart and liver. This ectopic fat deposition disrupts cellular metabolism, induces apoptosis, and contributes to organ dysfunction, especially in the myocardium.[27]

Materials and methods

Data source and study population

This study used data from the UK Biobank, a large prospective population-based cohort comprising over 500,000 participants aged 40–69 years recruited between 2006 and 2010 across the United Kingdom. All participants provided written informed consent, and the UK Biobank received ethical approval from the appropriate research ethics committees.27

For the present analysis, we included participants with available baseline data on anthropometric measures, physical activity, and cardiovascular outcomes. Individuals with missing data for any of the main variables of interest were excluded. After these exclusions, the final analytical sample consisted of N = 489909 participants. A flowchart describing the study sample selection is provided in Figure X.

Definition of cardiovascular disease

Prevalent cardiovascular disease (CVD) was defined at baseline as the presence of at least one of the following conditions: angina pectoris, myocardial infarction, or stroke. These diagnoses were identified using a combination of self-reported doctor diagnoses and hospital record data available in the UK Biobank. The outcome was treated as a binary variable.

Anthropometric measures

The following anthropometric indicators were evaluated:

- Body Mass Index (BMI): calculated as weight (kg) divided by height squared (m^2).
- Body Fat Percentage (BFP): derived from bioelectrical impedance measurements.
- Waist-to-Height Ratio (WHtR): calculated as waist circumference divided by height.

BMI was analyzed both as a continuous variable and using standard categories (underweight <18.5 kg/m^2, normal weight 18.5–24.9 kg/m^2, overweight 25–29.9 kg/m^2, obese ≥30 kg/m^2).

Physical activity assessment

Physical activity was assessed using self-reported questionnaire data at baseline. Participants reported the frequency and duration of different types of activity, including moderate physical activity, heavy do-it-yourself (DIY) activity, and strenuous sports. For each activity type, weekly duration was calculated in minutes per week and analyzed as a continuous variable. Extremely implausible values (e.g., reports exceeding 24 hours per day) were excluded prior to analysis.

Statistical analysis

Descriptive statistics were used to summarize baseline characteristics of the study population.

Multivariable logistic regression models were used to examine the associations between anthropometric measures (BMI, BFP, WHtR) and prevalent CVD. Separate models were fitted for each anthropometric measure as well as combined models where appropriate. Physical activity variables were added to the models to assess associations with CVD.

Results are presented as odds ratios (OR) with 95% confidence intervals (CI). Nonlinear associations for BMI were explored using categorical analyses and graphical inspection. Analyses were performed using a combination of the R and Python programming tools.

Results

Descriptives

Table 2. Descriptives

Feature	Average / Prevalence
BMI	27.4 kg/m^2
Waist to Height Ratio	0.54
Body Fat Percentage	31.4%
Angina	3.1%
Heart Attack	2.2%
Stroke	1.5%

Obesity indicators

For each indicator, the analysis was divided into low (<18.5) and medium (>18.5) BMI, all studied cardiovascular conditions and the three obesity measures Body Fat Percentage (BFP), Body Mass Index (BMI), and Waist to Height Ratio (WTH). The results for underweight BMI is shown in Table 3.

Table 3. Logistic regression coefficients

Measure	BFP	BMI	WTH
Angina	0.85	1.9E-7 (0.52)	403 (0.57)
Heart Attack	0.31 (0.77)	2.9E-23 (0.007)	812 (0.57)
Stroke	0.0015 (0.09)	1.9E-19(0.02)	898 (0.50)

Table 4. Logistic regression coefficients high BMI (*p*-value)

Measure	BFP	BMI	WTH
Angina	2.3 (<0.001)	270 (0.001)	2440 (<0.001)
Heart Attack	0.41 (<0.001)	120 (<0.001)	1640 (<0.001)
Stroke	2.3 (<0.001)	60 (<0.001)	450 (<0.001)

Exercise

Table 5 represents the duration and frequency of each type of activity (moderate activity, heavy DIY, strenuous sport) found in different medical conditions (angina, heart attack, stroke), under best-case (lowest CVD prevalence) and worst-case (highest prevalence).

Table 5. Best and worst practices of exercise for three diseases

Condition	Duration (hours)		Frequency (days per week)	
	Best	Worst	Best	Worst
Moderate activity				
Angina	0.5–1.0	1.5–2.0	1	0
Heart Attack	0.5–1.0	1.5–20	5	0
Stroke	0.25–0.5	1.0–1.5	1	0
Heavy DIY				
Angina	1.0–1.5	< 0.25	1	4–5
Heart Attack	1.0–1.5	< 0.25	1	4–5
Stroke	1.0–1.5	0.5–1.0	1	7
Strenuous sport				
Angina	0.5–1.0	2–3	1–3	7
Heart Attack	0.5–1.0	2–3	4–5	1
Stroke	0.5–1.0	> 3	2–3	7

The results show that for moderate physical activity, engaging in at least some activity each week is beneficial, and regular participation (especially in the case of heart attack survivors) can be particularly positive. For heavy DIY activities, the data suggest that they should be performed for longer durations, but less frequently, to avoid potential negative outcomes. In contrast, for strenuous sport, practicing it for extended durations and at very high frequencies appears to carry certain risks, especially for individuals with cardiovascular conditions.

The following three figures illustrate the probability of developing cardiovascular disease, as predicted by logistic regression models, based on the different types of physical activity.

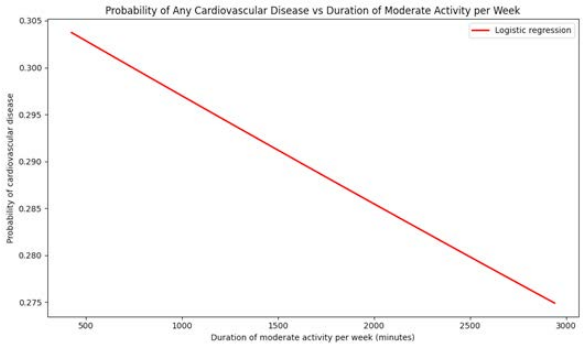

Figure 1. Probability of any cardiovascular disease by duration of moderate activity per week.

There is a clear inverse relationship between the duration of moderate physical activity and the probability of cardiovascular disease, supporting existing evidence that moderate exercise has protective cardiovascular effects.

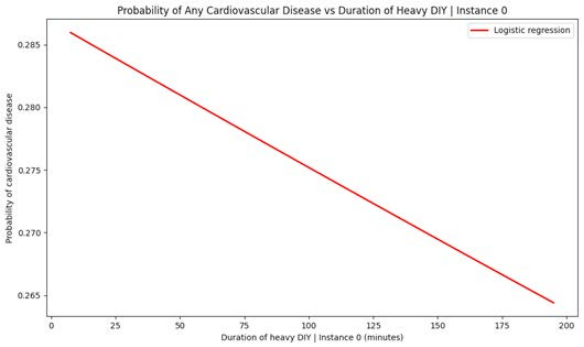

Figure 2. Probability of any cardiovascular disease by duration of heavy DIY per week

The graph shows a slight decrease in the probability of cardiovascular disease with increased time spent on heavy DIY tasks, indicating that regular intense manual labor may contribute positively to cardiovascular health.

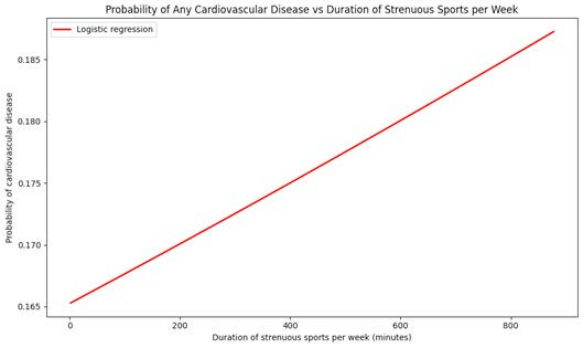

Figure 3. Probability of any cardiovascular disease by duration of strenuous sports per week

We observe that the probability of having cardiovascular disease slightly increases with the duration of strenuous sports per week, which may suggest that excessive high-intensity exercise could potentially have negative effects, especially if the training is not adapted to the individual's metabolism.

Table 6 below summarizes the results of the logistic regression analysis, showing the impact of various physical activities on the likelihood of cardiovascular disease (CVD), along with regression coefficients, intercepts (bias), sample sizes, and the number of observed CVD cases.

Table 6. Logistic Regression coefficients exercise

	Odds ratio	Intercept (Bias)
Duration of moderate activity per week	0.999944	−0.81
Duration of heavy DIY per week	0.999	−0.91
Duration of strenuous sports per week	1.000	−1.62

Dangerous BMI

Figure 4 shows the proportion of individuals with cardiovascular disease as a function of their Body Mass Index (BMI).

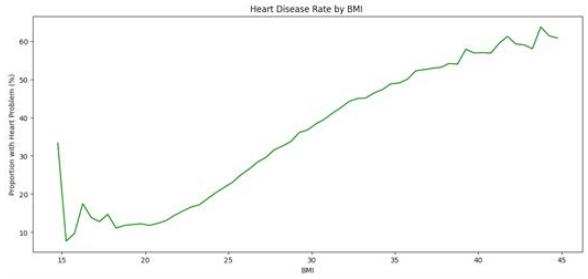

Figure 4. Heart disease rate by BMI

The proportion of individuals with heart disease increases with BMI values ranging from 14.53 to 45. An early spike appears at BMI ≈ 14.7 (33%), followed by a sharp drop to 7.9% at BMI ≈ 15.1 and a secondary peak at 16.3 (17.3%). From BMI ≈ 22 onward, prevalence rises steadily, reaching 30% at BMI ≈ 27.5, 45% at BMI ≈ 32.3, and peaking at 64.8% near BMI ≈ 43.5. This trend highlights a strong positive association between BMI and heart disease risk, especially above the overweight threshold (BMI > 25).

To further explore the relationship between body weight and cardiovascular disease, Figure 5 shows the proportion of individuals with cardiovascular disease, focusing specifically on those who are currently underweight (BMI < 18.5).

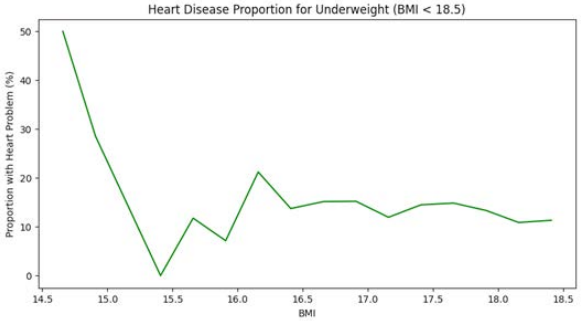

Figure 5. Heart disease rate for underweight (BMI < 18.5)

The highest heart disease proportion is 50% at BMI 14.6, dropping sharply to 28% at BMI 14.9 and reaching 0% at BMI 15.5. A secondary peak of 21.2% occurs at BMI 16.1, with rates stabilizing between 12–16% for BMIs from 16.5 to 18.4. Despite sample variability, these findings underscore a significant cardiovascular burden among underweight individuals.

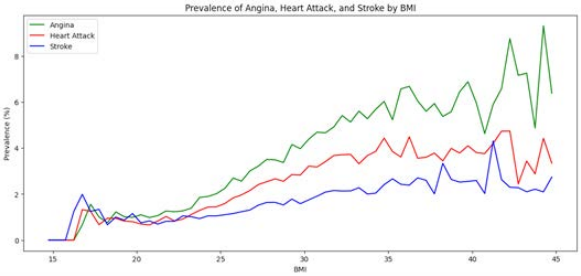

Figure 6. Prevalence of angina, heart attack and stroke by BMI

Across BMI values from 14.53 to 45.00, all three conditions, angina, heart attack, and stroke, show increasing prevalence with higher BMI. Angina peaks early at 1.5% (BMI ≈ 17), rises above 2% at BMI ≈ 25, and reaches 9.3% at BMI ≈ 44.5. Heart attack starts rising at BMI ≈ 17, increases steadily from BMI ≈ 23, and peaks at 4.73% around BMI ≈ 42. Stroke shows a peak of 2.0% at BMI ≈ 16.7, climbs gradually, and reaches 4.31% near BMI ≈ 41, remaining below 3% for most BMI values. These trends indicate a strong positive link between BMI and cardiovascular risk, especially for angina.

Discussion

The main conclusions were:

1. BMI is the most consistent measure of heart disease risk related to obesity when looking at both underweight and overweight. BMI exhibited a classic U-shaped relationship with CVD in our data. Figure 8 shows that very low BMIs (<16 kg/m^2) were associated with unexpectedly high prevalence of heart disease (~30–33%), followed by a decline in risk at normal BMI, and then a steep rise in risk above the normal range. In other words, both underweight and overweight extremes carried elevated risk. This pattern has been reported before.
2. These findings on BMI and WHtR provide empirical evidence for the early stages of the contemporary CKM staging model, where Stage 1 is defined by excess adiposity and Stage 2 by the emergence of metabolic risk factors.[11] This reinforces the utility of simple anthropometrics for initial risk stratification within a broader syndemic framework.
3. Finding thus reinforces that the relationship of BMI to CVD is not purely linear; there is a J/U-shaped curve with lowest risk in the mid-normal range (BMI ~22–25) and rising risk at both tails.[29]
4. This underscores that clinicians should not overlook the underweight population. Malnutrition, sarcopenia, and other health issues in underweight individuals likely contribute to the increased risk, as described in other studies, while the very high-BMI group's risk is driven by comorbid obesity conditions.
5. Body fat percentage alone added little independent predictive value. In our regression, BFP had only a tiny coefficient and did not improve model accuracy when combined with BMI. In effect, knowing someone's percent body fat provides no clear advantage beyond what BMI and waist measures already tell us.
6. Routine risk assessment can rely on simpler metrics (BMI or WHtR) without needing costly body composition tests. (That said, some literature does link high adiposity to risk factors, but as a stand-alone predictor BFP was weak in our analysis.)
7. Exercise exhibited a protective trend, which varied according to intensity. Moderate exercise and heavy DIY both reduced the prevalence of heart disease, however this does not scale proportionally. Greater minutes of moderate-intensity exercise were associated with lower heart disease prevalence, whereas the incremental benefit of heavy/vigorous activity was more modest. In practical terms, promoting a habitual regimen of moderate exercise (walking, cycling, etc.) should be a cornerstone of CVD prevention.
8. An underweight condition was associated with high risks of CVD with a peak of BMI between 14 and 15.

Correlation to the research question

This study examined the predictive value of anthropometric indicators, BMI, BFP, and WHtR, for cardiovascular disease (CVD) risk, as well as the influence of physical activity. Among these, BMI emerged as the most robust and consistent predictor, demonstrating a U-shaped relationship with CVD. WHtR also provided added utility, particularly in combination with BMI, while BFP offered minimal incremental value. These results underscore the practicality and reliability of using accessible anthropometric measures in clinical and public health settings.

Moderate physical activity was inversely associated with CVD prevalence, confirming its protective role. Notably, high-intensity physical activity did not yield proportional risk reduction and may, in some cases, have adverse effects, highlighting the need to tailor exercise recommendations to individual capacity. In fact, we can observe that engaging in high-intensity physical activity like strenuous sport for extended durations may also carry certain risks, highlighting that too much exercise is not always better, especially in vulnerable populations.

Comparison with previous studies

Our results align with epidemiological literature that describes a U- or J-shaped relationship between BMI and cardiovascular risk, with elevated risk in both underweight and obese individuals.[30] The lowest CVD risk was observed in individuals with BMI values in the range of 22–25 kg/m^2.

The limited contribution of BFP observed in our data is supported by earlier research suggesting that waist-based measures and BMI often outperform BFP in predicting cardiometabolic outcomes in public health settings.[30] While BFP has more physiological specificity, it appears redundant for broad screening when BMI and WHtR are available and easier to measure reliably.[30]

Regarding physical activity, our results reaffirm the protective role of moderate-intensity exercise, echoing

the American Heart Association's recommendation of 150 minutes per week. Interestingly, increased time spent in vigorous activity did not proportionally reduce CVD risk, suggesting a plateau or diminishing return effect, consistent with findings from other population-based studies.

Explanation of unexpected results

BMI was the most consistent measure of heart disease risk related to obesity, showing a classic U-shaped relationship with CVD in our data. Very low BMIs (<16 kg/m^2) were associated with an unexpectedly high prevalence of heart disease (~30–33%), followed by a decline in risk at normal BMI values, and then a steep rise in risk above the normal range (Figure 8). Thus, both underweight and overweight extremes carried elevated CVD risk. This pattern has been previously reported, reinforcing that the relationship between BMI and CVD is not purely linear but follows a J/U-shaped curve with the lowest risk in the mid-normal range (BMI approximately 22–25) and rising risk at both tails.

This finding underscores that clinicians should not overlook the underweight population. The increased risk in underweight individuals is likely driven by factors such as malnutrition, sarcopenia, and other health conditions, which have been linked to cardiovascular vulnerability.[30] Conversely, the high risk observed in the very high BMI group is attributed to comorbid obesity-related conditions. Extremely low body mass may reflect not only insufficient fat but also loss of protective lean tissue, which undermines metabolic and vascular resilience, thereby increasing CVD risk.

The emergence of higher risks with very high levels of training was slightly unexpected, but the target of at least 1–2 hours per week in line with recommendations from the American Heart Association.[31]

Study limitations

One of the limitations of our study lies in the unbalanced distribution of physical activity within the cohort: a large proportion of participants reported low levels of physical activity while having no cardiovascular diseases, which may introduce bias and reduce our ability to detect meaningful associations. Additionally, since physical activity data is self-reported, it is subject to inaccuracies or exaggerations, for example, some participants claimed to engage in moderate activity for 1,440 minutes per day (i.e., 24 hours), requiring us to filter out such clearly unrealistic entries. The age range of only 40–69 years old further restricts the generalizability.

Implications and future research

Our findings contribute to a better understanding of how lifestyle factors such as physical activity levels and BMI are associated with the risk of developing cardiovascular diseases. This reinforces the importance of promoting balanced physical activity and maintaining a healthy weight as part of cardiovascular disease prevention strategies. Future research could focus on longitudinal studies to better capture causal relationships, integrate objective measures of physical activity (e.g., wearable devices), or explore genetic and metabolic interactions with lifestyle factors. From a practical standpoint, these insights can inform public health policies and personalized prevention programs targeting at-risk populations based on physical activity and BMI profiles and systematically assess social determinants of health (SDOH), as adverse SDOH are fundamental drivers of disparities in obesity, physical inactivity, and CVD access, and are central to equitable prevention strategies.[11]

Conclusion

In this large population-based cross-sectional study, body mass index and waist-to-height ratio were robustly associated with prevalent cardiovascular disease, demonstrating a U-shaped and monotonic relationship, respectively. In contrast, body fat percentage provided limited additional discriminatory value beyond these simpler anthropometric measures.

Moderate physical activity was associated with lower CVD prevalence, supporting its role as a cornerstone of cardiovascular prevention strategies. Very high volumes of vigorous self-reported activity were not associated with further reductions in CVD prevalence, although this finding should be interpreted cautiously given the observational and cross-sectional nature of the data.

Taken together, these findings support the use of simple, widely available anthropometric measures for early cardiometabolic risk stratification within the CKM syndrome framework and reinforce the importance of balanced, sustainable physical activity patterns in population health.

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