Gogoba *et al. BMC Cardiovascular Disorders* (2025) 25:573
https://doi.org/10.1186/s12872-025-05006-x

BMC Cardiovascular Disorders

RESEARCH

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Sex differences in adiposity and hemodynamic parameters as cardiovascular risk indicators among South African university staff: a descriptive cross-sectional study

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Abstract

Background Cardiovascular diseases (CVDs) are the leading cause of death worldwide, with their prevalence continuing to rise each year. Adiposity indexes and hemodynamic parameters have been established as effective predictors of CVDs when analysed separately. However, the impact of sex differences on the distribution and combined use of these predictors remains largely unexplored, particularly in Sub-Saharan Africa. This study aimed to investigate the sex differences in the distribution of adiposity indexes (AI) and hemodynamic parameters (HP), as well as their associated indicators of cardiovascular diseases risks among staff members at Walter Sisulu University (WSU).

Methods This cross-sectional descriptive quantitative study was conducted on 100 healthy adults (50 males, 50 females) aged 18–65 years. AI were assessed using a stadiometer, body composition monitor, and tape measure, while HP were measured with a stethoscope and sphygmomanometer.

Results The study's findings revealed that mean values for AI, including height, visceral adiposity index, and waist circumference, were higher in males compared to females, while weight, body mass index, and hip circumference were greater in females. Additionally, the study indicated that mean values for HP, such as systolic blood pressure, diastolic blood pressure, and mean arterial pressure, were elevated in males, whereas pulse pressure was higher in females. Notably, heart rate was consistent across both sexes.

Conclusion This study provides useful information about the sex-based patterns of adiposity indices and hemodynamic distribution among selected South African populations.

Keywords Cardiovascular diseases, Adiposity indexes, Hemodynamic parameters, Sexual dimorphism

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Introduction

Cardiovascular diseases (CVDs) are the leading cause of death worldwide and have emerged as a significant public health concern with serious implications [1]. These diseases affect the heart that pumps blood and the blood vessels that carry blood [1, 2]. Among many others, most of these diseases are myocardial infarction, peripheral artery diseases, stroke, cardiomyopathy, rheumatic heart disease, aortic aneurysm and dissection, heart failure, coronary artery diseases, cerebrovascular disease, arrhythmia, hypertension, and congenital heart defects [3–5]. Available data revealed that CVDs cause 20 million deaths in 2024, indicating 34% of all deaths worldwide [6]. This trend shows that the global crude prevalence will increase from the current 598 million in 2025 to 1.14 billion in 2050, with the global death rates projected to be increased to 35.6 million (73.4%) by 2050 [6]. In sub-Saharan Africa, more than 1 million CVD-related deaths were recorded in 2019, accounting for 5.4% of the deaths caused by CVDs [7, 8]. More so, South Africa is usually regarded as the epicenter of human immunodeficiency virus (HIV)/AIDS and tuberculosis [9, 10]. The prevailing data have shown that CVDs remain a major health concern ranking second leading cause of death in South Africa responsible for over 1 in 6 deaths in the country, 22.4% of death in South Africa is attributed to CVDs [11].

Several predisposing factors to CVDs have been recorded, and these include: unhealthy dietary habits, smoking, physical inactivity, sedentary lifestyle, and alcohol consumption [12, 13]. More so, age, diabetes mellitus, genetic disposition, dyslipidemia, socioeconomic status, and gender have been highlighted as other predisposing factors to CVDs, especially in sub-Saharan Africa [14, 15]. Some of the risk factors, like metabolic syndromes, have been linked to the changes from rural to urban shift, which is now evident in most countries in sub-Saharan Africa, including South Africa [16]. Consequently, this has brought about dietary changes, sedentary lifestyles, and contributed to an increase in the cases of overweight and obesity among adults and adolescents in South Africa [16].

The predictors of CVDs include HP and AI, as well as the visceral adiposity index (VAI), which are more sensitive and specific in terms of cardiovascular risk. They also serve as important indicators of central obesity, which is considered a more reliable predictor of cardiovascular diseases [17]. The hemodynamic parameters such as total peripheral resistance (TPR), systolic blood pressure (SBP), diastolic blood pressure (DBP), Heart rate (HR), stroke volume (SV), cardiac output (CO), Pulse Pressure (PP), and electrocardiogram (ECG) have been recorded as appropriate predictors of CVDs [18]. Also, body mass index (BMI), waist circumference (WC), waist-hip-ratio (WHR), waist-height ratio (WHtR), visceral adiposity index (VAI), body adiposity index (BAI), lipid accumulation product index (LAPI) are the most widely used adiposity indices [19].

These parameters have been widely employed as predictors of the occurrence and severity of CVDs in various populations [20–22]. Nevertheless, the current trends show that most AI and HP have been used independently to predict the incidence and severity of cardiovascular diseases [23], suggesting that important pathophysiological indicators of cardiovascular diseases may have been overlooked. This is on account that adiposity parameters predominantly reveal body fat distribution but not cardiovascular function, while hemodynamic indices measure the blood circulation system, not fat-related function [24, 25]. Consequently, results in weaker sensitivity and predictability of CVDs and inappropriate treatment [26–28]. This gap in knowledge on underexplored combination utilization and sex distribution of these two indices (AI and HP) spurred this investigation that seeks to explore the distribution of these markers among Walter Sisulu University staff in the Eastern Cape province of South Africa.

Materials and methods

Study location

This study was carried out at Walter Sisulu University, both Nelson Mandela Drive and the Faculty of Medicine and Health Science, Sissong Street campus, located in a town called Mthatha, one of the Universities in the Eastern Cape Province.

Research approach

In this cross-sectional descriptive quantitative study, sex differences in the distribution of adiposity indexes, hemodynamic parameters [Systolic blood pressure (mmHg), Diastolic blood pressure (mmHg), Heart rate (bpm), Pulse pressure (mmHg), Mean arterial pressure (mmHg)], among Walter Sisulu University staff members were investigated using a cross-sectional descriptive quantitative study design to collect data from participants at a specific time.

Methodological design

The study was performed between July and September 2024, and the measurements of the AI and HP were taken between 09:00 h and 11:00 h daily in the month of July, 2024 from volunteered participants.

Population and sampling

The sample size was calculated using the below formular;

$$n = n_0/(1 + n_0/N) \tag{1}$$

where:

where n is the adjusted sample size, N is population size and n_0 is initial sample size based on 95% level of confidence.

Both males and females were considered in this study and 100 staff members (50 male and 50 female) of Walter Sisulu University were recruited by verbal invitation and visiting them in their offices and residences. The quota sampling method was used for this study because this was an efficient method for recruiting participants because of the criteria that we had. Only staff members who were willing to participate in the study were used; no participant was forced to participate.

Research instruments

An Omron Digital sphygmomanometer (3 Series® Upper Arm Blood Pressure Monitor BP7150 by Omron Healthcare Inc.®) was used to measure hemodynamic parameters such as blood pressure (systolic and diastolic blood pressure), heart rate, pulse rate (PR) and mean arterial pressure (MAP).

A body composition analyser (Omron BF511 Body Composition Monitor) was used to measure adiposity indexes such as body mass index (BMI), visceral adiposity index (VAI), and weight (W). Also, a stadiometer was used for height (H) measurements, while tape measure was used for waist circumference and hip circumference.

Data collection procedure

A cross-sectional study design was used, and data were collected from participants at a specific time of the day (9:00–11:00 AM). The participants were asked to rest on a comfortable chair with their arms resting on a table and their feet resting flat on the floor. The cuff was placed around the arm, ensuring that it was at the same level as the participant's heart. For adiposity indexes, an Omron body composition monitor was used; the participant had to stand on the analyzer's footpads and grip the handgrips.

Inclusion and exclusion criteria

Inclusion criteria

- Adult males and females of Walter Sisulu University employees between the ages of 18 and 65 years were considered in this study.
- The participant's medical conditions were intact for him/her to participate in the study.

Exclusion criteria

- No student or community member was allowed to participate in this study.
- Staff members of other universities were not allowed to participate in this study even if they were willing to participate.
- Pregnant women were not allowed to participate.
- The staff members on chronic medication were excluded, because it may create a bias in the data.
- Participants who did not sign the consent form were excluded.

Ethical considerations

Ethical approval was sought and obtained from the Human Research Ethics Committee (HREC) of Walter Sisulu University (WSU) with approval number: 070/2024 before the commencement of this study. Also, necessary gatekeeper permissions were obtained before the start of this study. All the experiments were performed in accordance with the Helsinki declaration.

Permission for inclusion as a participant

Permission to be included in this study was obtained verbally from the prospective participant.

Anonymity and confidentiality

Anonymity and confidentiality of the medical information of the participant were always maintained.

Rights of participants

- Participants were given the right to leave if they felt uncomfortable during the study.
- Every participant was given the right to ask questions if they did not understand what had already been explained to them.

Informed consent

The informed consent was sought and obtained from the participant.

Statistical analysis

All the collected information (Adiposity indexes and Haemodynamic parameters) was entered into Microsoft Excel for storage and analysis. Descriptive statistics were presented as means ± SD. An independent T-test was used to compare the means of adiposity indexes and haemodynamic parameters between males and females of participants. Statistically significant data was considered at $p < 0.05$.

Results

Demographics

The study included 100 participants, with an equal distribution of 50 males, age 31 ± 10 and 50 females, age 32 ± 10, all of whom were staff members at Walter Sisulu University.

Adiposity index and hemodynamic parameters assessments for males and female

The study's findings indicated that the mean values for adiposity indexes in males were as follows: height

Table 1 The sex differences in the distribution of adiposity indexes in males and female of Walter Sisulu university staff members

Adiposity indexes	Males (n = 50) Mean ± SD	Females (n = 50) Mean ± SD	P values
Height (cm)	171 ± 6	158 ± 6	0.00002***
Weight (kg)	77 ± 2	79 ± 2	0.6011
Body Mass Index (kg/m2)	26 ± 5	31 ± 7	0.000022**
Visceral Adiposity index	9 ± 6	8 ± 2	0.3578
Waist circumference (cm)	91 ± 1	90 ± 2	0.8228
Hip circumference (cm)	103 ± 1	114 ± 1	0.03282*

P values < 0.05

***extreme significant

**highly significant

*significant difference

171 ± 6 cm, weight 77 ± 18 kg, body mass index 26 ± 5 kg/m^2, visceral adiposity index 9 ± 6, waist circumference 91 ± 14 cm, and hip circumference 103 ± 12 cm (see Table 1). Also, the study's findings revealed that the recorded data for adiposity indexes in females were as follows: height 158 ± 6 cm, weight 79 ± 19 kg, body mass index 31 ± 7 kg/m^2, visceral adiposity index 8 ± 2, waist circumference 90 ± 18 cm, and hip circumference 114 ± 11 cm (see Table 1). The mean hemodynamic parameters recorded were systolic blood pressure 129 ± 21 mmHg, diastolic blood pressure 82 ± 13 mmHg, heart rate 71 ± 7 bpm, pulse pressure 71 ± 11 mmHg, and mean arterial pressure 58 ± 0.1 mmHg (refer to Table 2). In addition, the hemodynamic parameters recorded for females were: systolic blood pressure 120 ± 17 mmHg, diastolic blood pressure 78 ± 9 mmHg, heart rate 71 ± 6 bpm, pulse pressure 77 ± 11 mmHg, and mean arterial pressure 53 ± 6 mmHg (Table 2).

Discussions

The global prevalence of cardiovascular diseases has risen significantly, posing a major public health challenge [29]. Key factors contributing to this increase include population growth, aging, and escalating levels of modifiable risk factors such as high blood pressure, high cholesterol, poor diet, tobacco use, alcohol consumption, physical inactivity, and air pollution [30]. While the AI and HP are effective and sensitive markers for predicting the risk of cardiovascular diseases, they have typically been used independently, neglecting many of the pathophysiological indicators associated with these conditions [23]. In addition, hemodynamic parameters differ between males and females due to variations in body composition, hormones, and cardiovascular structure, which may impact ischemic and bleeding risks in each sex [31, 32]. Furthermore, the sex-based distribution of these markers remains underexplored, particularly in sub-Saharan Africa. Therefore, this study aims to investigate the sex differences in the distribution of these markers within this study population.

This study found no statistically significant difference between age and sex, which aligns with the findings of Ball, Løchen [33], who also reported no significant relationship between age and sex, although the average age for females was slightly higher than that for males. This result suggests that age did not influence the sex-based distributions of the adiposity index and hemodynamic parameters in this study population; however, other factors may serve as major determinants. Height is one of the most critical adiposity parameters, and this study identified a significant difference in mean height between males and females, which is consistent with previous research [34]. Yusuf, Hawken [34] demonstrated a significant difference in height between males and females, indicating that height can be used to assess body fat distribution, making it a better predictor of cardiovascular diseases. This significant difference in height may be attributed to hormonal, genetic, and evolutionary factors. Empirical evidence suggests that sex-determining genes located on the Y chromosome play a role in controlling male development and differences in stature [35]. Additionally, this significant difference may be linked to the effects of testosterone, which has been shown to exert a more effective anabolic effect on bones, thereby increasing bone density and length [36].

Table 2 The sex differences in the distribution of hemodynamic parameters in males and female of Walter Sisulu university staff members

Hemodynamic parameters	Males (n = 50) Mean ± SD	Females (n = 50) Mean ± SD	P values
Systolic blood pressure (mmHg)	129 ± 2	120 ± 2	0.001233***
Diastolic blood pressure (mmHg)	82 ± 1	78 ± 9	0.07026
Heart Rate (bpm)	71 ± 7	71 ± 6	0.06991
Pulse pressure (mmHg)	71 ± 1	77 ± 1	0.0231*
Mean arterial pressure (mmHg)	88 ± 0	83 ± 6	0.01344**

P values < 0.05

***extreme significant

**highly significant

*significant difference

Although the mean weights of males and females were not significantly different, a slight difference was observed, with females exhibiting a slight increase in weight. This finding is consistent with similar investigations that attribute such slight differences to the distribution of fat in areas such as the breasts, buttocks, and thighs, which are typically more pronounced in females [37]. This factor may contribute to the slight increase in weight observed in females compared to males. Excessive weight gain and fat accumulation have been linked to an increased risk of atherosclerosis and cardiovascular diseases, particularly acute coronary diseases [38]. Furthermore, a significant difference in mean body mass index (BMI) was found in females, indicating excessive body fat. A similar study by Schorr, Dichtel [39] reported higher BMI in females, linking it to excessive body fat deposition with detrimental health effects. Recently, research by Fahed, Aoun [40] associated higher BMI with an increased risk of developing hypertension and cardiovascular diseases. Previous studies have documented patterns of body fat distribution in both sexes, revealing that males tend to accumulate fat around the trunk and abdominal organs, while females typically have more body fat around the hips and thighs [41].

In this study, the mean visceral adiposity index and mean waist circumference were not found to be significant, suggesting that the pattern of distribution may be specific to this population. These findings regarding the mean visceral adiposity index does not agree with previous studies, which reported a significant difference in visceral adiposity, indicating that males are more likely to develop central obesity due to increased fat distribution around abdominal organs [42, 43]. This discrepancy highlights the need for further research to understand the factors influencing adiposity patterns in different populations. Consequently, males with greater percentages of visceral adiposity have been associated with higher levels of triglycerides (TG), free fatty acids (FFAs), postprandial insulin and at high risk of developing CVDs [44]. The insignificant results obtained on mean waist circumference concurs with a similar investigation which reveals that the waist in both males and females contribute similarly to predicting cardiovascular diseases, but males tend to have large waist than females as they have more body fat distribution in the trunk and abdominal organs [45]. Furthermore, significant differences obtained on the mean hip circumference between males and females in this study revealed a sex-specific adiposity index distribution. Similar studies have documented larger mean hip circumference in females compared to males and attributes these differences to the high fat distribution around thighs, and buttocks in females [39, 46, 47].

The significant difference in mean systolic blood pressure between male and female participants in this study aligns with previous research that attributes higher mean systolic blood pressure in males to variations in hormonal, genetic, and anatomical factors [48]. Additionally, it has been shown that blood pressure tends to increase with age in both normotensive individuals, regardless of sex [49]. Generally, males exhibit higher blood pressure than females until older age, at which point the values tend to converge. In females, the rise in blood pressure is often associated with menopause and estrogen depletion [49, 50].

Conversely, the mean diastolic blood pressure for males and females was not significantly different in this study, suggesting that the participants may have similar levels of systemic vascular resistance, a key marker in diastolic pressure, as previously reported [51]. These results imply that while factors such as environmental influences, genetics, and lifestyle may be comparable, sex may not be a relevant determinant in the regulation of diastolic blood pressure [49].

The results regarding the mean heart rate of males and females in this study indicate no significant difference, suggesting that there are sex-based similarities in sympathetic and parasympathetic activities within this population. This finding implies that sex may not be a critical factor in assessing resting heart rate, consistent with previous reports [52]. However, these findings contrast with those of Koenig and Thayer [53], who documented a greater mean heart rate in female participants, attributing their results to sex-based differences in autonomic activities. It is important to note that a higher heart rate has been associated with an increased risk of developing cardiovascular diseases and abnormal functioning of heart muscles [54]. This discrepancy highlights the need for further investigation into the factors influencing heart rate variations across different populations and the potential implications for cardiovascular health.

In this study, both mean pulse rate and mean arterial pressure were found to be significant among male and female participants, indicating a sex-based difference in these cardiovascular markers. The mean pulse rate was higher in females, which aligns with previous research [55]. Factors such as hormonal influences, autonomic activities, and heart size have been linked to these sex-based differences in mean pulse rate [56, 57]. This suggests that females may experience greater aortic stiffness, potentially leading to an increased pulse rate and a higher risk of developing cardiovascular diseases as they age [58].

Additionally, the significant difference observed in mean arterial pressure between males and females, with males exhibiting higher mean values, further underscores the sex-based differences in arterial blood pressure. This finding is consistent with previous studies that attribute these differences to vascular and hormonal factors [49].

The higher mean arterial pressure in males increases their likelihood of developing cardiovascular diseases compared to females, as mean arterial pressure is associated with target organ damage and cardiovascular disorders [59].

Conclusion

This study provides useful information about the sex-based patterns of adiposity indices and hemodynamic distribution among selected South African populations, an area that remains underexplored. This substantiates the need for sex-tailored evaluation and management modalities for CVDs, especially in this study population. Further studies are necessary to establish the correlation between the patterns of distribution and its use in predicting the risk of cardiovascular diseases.

Limitations

The primary limitation of this study was small sample size, hence, a large sample size with more than two racial groups for future studies. The study was not specific to race, and exercise. Also performing this investigation outside Walter Sisulu University such as in the Eastern Cape Province or all over South Africa will be of great interest. More so, this study did not consider sex differences in chest shape conformation. However, previous studies indicate that individuals with a concave-shaped chest wall conformation and/or narrow antero-posterior thoracic diameter are predominantly females with increased prevalence of mitral valve prolapse and good outcome over mid-to-long-term follow-up [60, 61]. Based on this evidence, further studies are needed for evaluating the prognostic role of chest shape conformation in South African males vs. females.

Abbreviations

CVDs	Cardiovascular diseases
AI	Adiposity Indexes
HP	Hemodynamic Parameters
WSU	Walter Sisulu University
VAI	Visceral Adiposity Index
TPR	Total Peripheral Resistance
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
HR	Heart Rate
SV	Stroke Volume
CO	Cardiac Output
PP	Pulse Pressure
ECG	Electrocardiogram
BMI	Body Mass Index
WC	Waist Circumference
WHR	Waist-Hip-Ratio
WHtR	Waist-Height Ratio
BAI	Body Adiposity Index
LAPI	Lipid Accumulation Product Index

Acknowledgements

The authors would like to appreciate the participants for their time and efforts in making this study a big success.

Authors' contributions

Conceptualization: SOO, BAA, SG, SKL, OA, AIJ, OOA; Experimentation: SG, SOO, BAA; Data collection: SG, SOO, BAA; Data analysis: SG, SOO, BAA, SKL; Manuscript writing; SOO, BAA, SG, SKL, OA, AIJ, OOA; Manuscript Editing: SOO, BAA, SKL, OA, AIJ, OOA.

Funding

The authors received no funding for this study.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Participants were given a consent form explaining the procedure of the experiment. The consent form clearly explains what was going to happen during the experiment and the study explained to them that their details wouldn't be used for any other purpose outside this research study. The participant's details were confidential and were not exploited in any way. All participants were allowed to leave whenever they felt like they no longer wanted to participate in the study.

Consent for publication

All authors gave permission for publication.

Competing interests

The authors declare no competing interests.

Received: 16 May 2025 / Accepted: 30 June 2025
Published online: 02 August 2025

References

1. Gaidai O, Cao Y, Loginov S. Global cardiovascular diseases death rate prediction. Curr Probl Cardiol. 2023;48(5):101622.
2. Мирзаев M. The cardiovascular system: structure of the heart and blood vessels. Международный Мультидисциплинарный Журнал Исследований И Разработок. 2025;1(2):269–72.
3. Hupp WS. Cardiovascular diseases. The ADA practical guide to patients with medical conditions. 2015. pp. 25–42.
4. Laranjo L, et al. World heart federation roadmap for secondary prevention of cardiovascular disease: 2023 update. Global Heart. 2024;19(1):8.
5. Victor G, et al. The global burden of cardiovascular disease in adults: a mapping review. J Cardiovasc Nurs. 2024;10:10–97.
6. Chong B, et al. Global burden of cardiovascular diseases: projections from 2025 to 2050. Eur J Prev Cardiol. 2024:zwae281. https://doi.org/10.1093/eurjpc/zwae281.
7. Keates AK, et al. Cardiovascular disease in Africa: epidemiological profile and challenges. Nat Rev Cardiol. 2017;14(5):273–93.
8. Bulto LN, Hendriks JM. The burden of cardiovascular disease in africa: prevention challenges and opportunities for mitigation. Eur J Cardiovasc Nurs. 2023;23(6):e88–90.
9. Organization W.H. WHO operational handbook on tuberculosis. Module 6: tuberculosis and comorbidities. Geneva: World Health Organization; 2025.
10. UNAIDS GH. AIDS statistics—fact sheet. 2022. Available on: https://www.unaids.org/sites/default/files/media_asset/UNAIDS_FactSheet_en.pdf. 2023.
11. Ricci C, et al. Determinants of mortality status and population attributable risk fractions of the North West province, South African site of the international PURE study. Arch Public Health. 2024;82(1):102.
12. Chikafu H, Chimbari MJ. Cardiovascular disease healthcare utilization in sub-Saharan Africa: a scoping review. Int J Environ Res Public Health. 2019;16(3):419.
13. Mudie K, et al. Non-communicable diseases in sub-Saharan Africa: a scoping review of large cohort studies. J Global Health. 2019;9(2):020409.
14. Mozaffarian D, Wilson PW, Kannel WB. Beyond established and novel risk factors: lifestyle risk factors for cardiovascular disease. Circulation. 2008;117(23):3031–8.

15. Negesa LB, et al. Patients' knowledge on cardiovascular risk factors and associated lifestyle behaviour in Ethiopia in 2018: a cross-sectional study. PLoS ONE. 2020;15(6):e0234198.
16. Rossouw HA, Grant CC, Viljoen M. Overweight and obesity in children and adolescents: the South African problem. South Afr J Sci. 2012;108(5):1–7.
17. Amato MC, et al. Visceral adiposity index: a reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care. 2010;33(4):920–2.
18. Saghiv MS, et al. Cardiovascular function. Basic Exerc Physiol. 2020:285–369.
19. Sun K, et al. Sex difference in the association between habitual daytime napping and prevalence of diabetes: a population-based study. Endocrine. 2016;52:263–70.
20. Schneider HJ, et al. Accuracy of anthropometric indicators of obesity to predict cardiovascular risk. J Clin Endocrinol Metab. 2007;92(2):589–94.
21. Yadav RL, et al. Association between obesity and heart rate variability indices: an intuition toward cardiac autonomic alteration–a risk of CVD. Diabetes Metab Syndr Obes. 2017;10:57–64.
22. Mansoori A, et al. Predictive properties of novel anthropometric and biochemical indexes for prediction of cardiovascular risk. Diabetol Metab Syndr. 2024;16(1):304.
23. Müller MJ, et al. Beyond BMI: conceptual issues related to overweight and obese patients. Obes Facts. 2016;9(3):193–205.
24. Hall JE, et al. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. Circul Res. 2015;116(6):991–1006.
25. Neeland IJ, Poirier P, Després J-P. Cardiovascular and metabolic heterogeneity of obesity: clinical challenges and implications for management. Circulation. 2018;137(13):1391–406.
26. Satoh M, et al. Lifetime risk of stroke and coronary heart disease deaths according to blood pressure level: EPOCH-JAPAN (Evidence for cardiovascular prevention from observational cohorts in Japan). Hypertension. 2019;73(1):52–9.
27. Piché M-E, Tchernof A, Després J-P. Obesity phenotypes, diabetes, and cardiovascular diseases. Circul Res. 2020;126(11):1477–500.
28. Yusuf S, et al. Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study. Lancet. 2020;395(10226):795–808.
29. Roth GA, 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.
30. Liu Y, et al. Inflammation biomarkers are associated with the incidence of cardiovascular disease: a meta-analysis. Front Cardiovasc Med. 2023;10:1175174.
31. Calabro P, et al. Are we ready for a gender-specific approach in interventional cardiology? Int J Cardiol. 2019;286:226–33.
32. Cesaro A, et al. Visceral adipose tissue and residual cardiovascular risk: a pathological link and new therapeutic options. Front Cardiovasc Med. 2023;10:1187735.
33. Ball J, et al. Sex differences in the impact of body mass index on the risk of future atrial fibrillation: insights from the longitudinal population-based Tromsø study. J Am Heart Assoc. 2018;7(9):e008414.
34. Yusuf S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937–52.
35. Stulp G, et al. Does natural selection favour taller stature among the tallest people on earth? Proc Biol Sci. 2015;282(1806):20150211.
36. Rogol AD, Clark PA, Roemmich JN. Growth and pubertal development in children and adolescents: effects of diet and physical activity. Am J Clin Nutr. 2000;72(2):S521–8.
37. Katch VL, et al. Contribution of breast volume and weight to body fat distribution in females. Am J Phys Anthropol. 1980;53(1):93–100.
38. Messerli FHG, Schmieder GE, Sundgaard-Riise RE, Nunez KIR, Amodeo BDC. Disparate cardiovascular findings in men and women with essential hypertension. Ann Intern Med. 1987;107(2):158–161.
39. Schorr M, et al. Sex differences in body composition and association with cardiometabolic risk. Biol Sex Differ. 2018;9:1–10.
40. Fahed G, et al. Metabolic syndrome: updates on pathophysiology and management in 2021. Int J Mol Sci. 2022;23(2):786.
41. Adab P, Pallan M, Whincup PH. Is BMI the best measure of obesity? London: British Medical Journal Publishing Group; 2018. p. 360.
42. Javed AA, et al. Age-appropriate BMI cut-points for cardiometabolic health risk: a cross-sectional analysis of the Canadian longitudinal study on aging. Int J Obes. 2022;46(5):1027–35.
43. Regensteiner JG, Reusch JE. Sex differences in cardiovascular consequences of hypertension, obesity, and diabetes: JACC focus seminar 4/7. J Am Coll Cardiol. 2022;79(15):1492–505.
44. Chang E, Varghese M, Singer K. Gender and sex differences in adipose tissue. Curr Diab Rep. 2018;18:1–10.
45. Wells JC, Treleaven P, Cole TJ. BMI compared with 3-dimensional body shape: the UK National sizing survey. Am J Clin Nutr. 2007;85(2):419–25.
46. Shungin D, et al. New genetic loci link adipose and insulin biology to body fat distribution. Nature. 2015;518(7538):187–96.
47. Kościński K. Assessment of waist-to-hip ratio attractiveness in women: an anthropometric analysis of digital silhouettes. Arch Sex Behav. 2014;43:989–97.
48. Dimkpa U, Ugwu A, Oshi D. Assessment of sex differences in systolic blood pressure responses to exercise in healthy, non-athletic young adults. J Exerc Physiol Online. 2008;11(2):18–25.
49. Reckelhoff JF. Gender differences in the regulation of blood pressure. Hypertension. 2001;37(5):1199–208.
50. Reckelhoff J, Cardozo LLY, Shawky N. Sex and gender differences in cardiovascular-renal-metabolic physiology and pathophysiology: sex, gender and function. London: Elsevier; 2024.
51. Hall ME, et al. Weight-loss strategies for prevention and treatment of hypertension: a scientific statement from the American Heart Association. Hypertension. 2021;78(5):e38-50.
52. Aubert AE, Seps B, Beckers F. Heart rate variability in athletes. Sports Med. 2003;33:889–919.
53. Koenig J, Thayer JF. Sex differences in healthy human heart rate variability: a meta-analysis. Neurosci Biobehav Rev. 2016;64:288–310.
54. Puig E, et al. Resting heart rate, cardiovascular events, and all-cause mortality: the REGICOR study. Eur J Prev Cardiol. 2022;29(5):e200–2.
55. Ryan SM, et al. Gender-and age-related differences in heart rate dynamics: are women more complex than men? J Am Coll Cardiol. 1994;24(7):1700–7.
56. Umetani K, et al. Twenty-four hour time domain heart rate variability and heart rate: relations to age and gender over nine decades. J Am Coll Cardiol. 1998;31(3):593–601.
57. Uranga AP, Levine J, Jensen M. Isotope tracer measures of meal fatty acid metabolism: reproducibility and effects of the menstrual cycle. Am J Physiol Endocrinol Metab. 2005;288(3):E547-55.
58. Waddell TK, et al. Women exhibit a greater age-related increase in proximal aortic stiffness than men. J Hypertens. 2001;19(12):2205–12.
59. Gao Q, et al. Association between mean arterial pressure and clinical outcomes among patients with heart failure. ESC Heart Fail. 2023;10(4):2362–74.
60. Freed LA, et al. Prevalence and clinical outcome of mitral-valve prolapse. N Engl J Med. 1999;341(1):1–7.
61. Sonaglioni A, et al. Echocardiographic assessment of mitral valve prolapse prevalence before and after the year 1999: a systematic review. J Clin Med. 2024;13(20).

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