



## Systematic Review Article

# ASSOCIATION BETWEEN OBESITY AND HYPERTENSION: A SYSTEMATIC REVIEW

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## ABSTRACT

**Background:** Obesity and hypertension are major public health challenges worldwide and are key contributors to cardiovascular morbidity and mortality. Growing evidence suggests a strong relationship between excess adiposity and elevated blood pressure; however, findings vary across populations and obesity indices. The objective is to systematically review published literature evaluating the association between obesity and hypertension in adult populations.

**Materials and Methods:** This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Electronic databases including PubMed, Scopus, Web of Science, and Google Scholar were searched for studies published. Eligible studies included observational designs (cross-sectional, cohort, or case-control studies), review articles, case-report and randomized controlled trials that reported associations between obesity measures and hypertension and articles published in English were considered for inclusion. Non-English papers without extractable data, Lacked clear outcome measures or evaluable results were excluded. Data extraction and quality assessment were performed.

**Results:** A total of 21 studies met the inclusion criteria. Obesity, measured by BMI, waist circumference, or waist-hip ratio, showed a consistent and significant association with hypertension. Prospective studies demonstrated a clear dose-response relationship, with hypertension risk increasing across BMI categories, even within the normal range. Central obesity was independently linked to higher blood pressure and greater cardiometabolic risk. Evidence from trials and meta-analyses confirmed that weight reduction lowers blood pressure, identifying both general and central obesity as key modifiable risk factors for hypertension.

**Conclusion:** Increasing adiposity markedly elevates hypertension risk, while weight reduction results in significant blood pressure improvement. Early identification and effective management of obesity are essential for preventing and controlling hypertension.

**Keywords:** Obesity, overweight, body mass index, waist circumference, hypertension, blood pressure.

## INTRODUCTION

Hypertension remains one of the leading causes of premature death worldwide and is a major risk factor for cardiovascular diseases, stroke, and renal disorders. According to global estimates, suggest that in 2010, 31.1% of adults (1.39 billion) worldwide had hypertension (Mills KT et al.,2020).<sup>[1]</sup> Parallel to this trend, the prevalence of obesity has increased dramatically over recent decades, making it a major public health concern.

Obesity, particularly visceral adiposity, accounts for nearly 65–75% of primary hypertension risk. It increases blood pressure by enhancing renal sodium reabsorption and impairing pressure natriuresis. Key mechanisms include renal compression by fat, activation of the renin-angiotensin-aldosterone system, and increased sympathetic nervous system activity, partly mediated by leptin. With prolonged obesity and renal damage, hypertension becomes resistant and requires multidrug therapy with

management of associated metabolic risks (Hall JE et al., 2015).<sup>[2]</sup>

The mechanisms linking obesity to hypertension remain incompletely understood, though sympathetic nervous system activation plays a central role. In obesity, pressure-natriuresis is shifted to higher arterial pressures due to early renal sodium retention, leading to extracellular fluid expansion and volume-dependent hypertension. Increased activity of the renin-angiotensin-aldosterone system, along with insulin resistance and inflammation, further contributes to vascular dysfunction and elevated blood pressure. Leptin and other neuropeptides may mediate these effects, highlighting obesity as a chronic condition requiring long-term, mechanism-based management (Kotsis V et al., 2010).<sup>[3]</sup>

Despite extensive research, variations in study design, population characteristics, and obesity measurements necessitate a comprehensive synthesis of existing evidence. This systematic review aims to evaluate and summarize the association between obesity and hypertension in adult populations.

### Objectives

1. To evaluate the association between obesity and hypertension in adults.
2. To determine the risk of hypertension among obese individuals compared with non-obese individuals.
3. To assess the role of different obesity indicators in predicting hypertension.

## MATERIALS AND METHODS

This systematic review was conducted following the PRISMA guidelines. A systematic literature search was performed using PubMed, Scopus, Web of Science, and Google Scholar. The search strategy included combinations of the following terms: obesity, overweight, body mass index, waist circumference, hypertension, and blood pressure. Reference lists of relevant articles were also screened to identify additional studies. After removal of duplicate records, titles and abstracts were screened for relevance, and potentially eligible studies underwent full-text assessment for inclusion.

### Eligibility Criteria

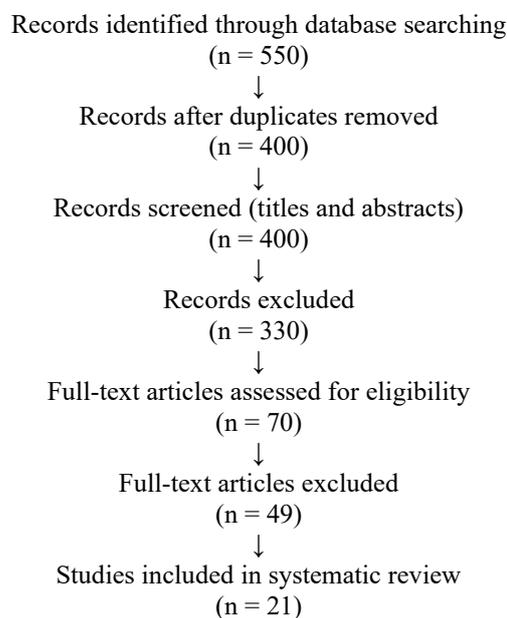
#### Inclusion criteria:

- Observational studies (cross-sectional, cohort, or case-control)
- Review articles, randomized control trials, case-report
- Studies reporting associations between obesity measures and hypertension
- Articles published in English

#### Exclusion criteria:

- Non-English papers without extractable data, lacked clear outcome measures or evaluable results.

Titles, abstracts, and full texts were screened. Data extraction and quality assessment were performed independently using standardized tools appropriate to study design. Due to heterogeneity among studies, a qualitative synthesis was conducted.



## RESULTS

A WHO Expert Consultation held in Geneva in December 2008 reviewed the use of waist circumference and waist-hip ratio as measures of body fat distribution to complement BMI-based obesity assessment (Nishida C et al., 2009).<sup>[4]</sup> Hypertension was generally defined according to international guidelines, with systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg, (Unger T, et al., 2020) or the use of antihypertensive medication.<sup>[5]</sup>

### Association Between General Obesity and Hypertension

In a prospective cohort of 13,563 initially normotensive men from the Physicians' Health Study, higher body mass index (BMI) was independently associated with an increased risk of developing hypertension over a median follow-up of 14.5 years. A total of 4,920 participants developed hypertension, with risk rising progressively across BMI categories, including values within the normal range. Compared with the lowest BMI group, men with higher BMI showed a stepwise increase in hypertension risk, which remained significant after adjustment for baseline blood pressure, diabetes, and hypercholesterolemia. These findings demonstrate a strong dose-response relationship between BMI and incident hypertension and underscore the importance of preventing overweight and obesity to reduce hypertension risk (Gelber RP et al., 2007).<sup>[6]</sup>

In the Framingham Offspring Study of 4,294 adults aged 20–49 years followed for 8 years, hypertension

prevalence was higher in men than women, though treatment rates increased over time, particularly among women. Hypertension incidence rose markedly with age, with men at greater risk before age 40 and similar rates in both sexes thereafter. Adiposity emerged as the strongest independent predictor of hypertension in both men and women, along with heart rate and metabolic factors. Changes in body fat over follow-up were directly associated with increases in systolic and diastolic blood pressure, highlighting adiposity as a key modifiable risk factor (Garrison RJ et al., 1987).<sup>[7]</sup>

A meta-analysis of 25 randomized controlled trials involving 4,874 participants demonstrated that weight reduction significantly lowers blood pressure. An average weight loss of about 5 kg was associated with reductions of approximately 4.4 mm Hg in systolic and 3.6 mm Hg in diastolic blood pressure, with greater effects seen in individuals achieving larger weight loss and in those receiving antihypertensive therapy. These findings confirm weight loss as an effective strategy for both prevention and management of hypertension (Neter JE et al., 2003).<sup>[8]</sup>

#### Central Obesity and Hypertension

Beyond visceral and hepatic fat, other ectopic fat depots may also contribute to the cardiometabolic risk associated with overweight and obesity. At a given BMI, increased waist circumference reflects greater abdominal and visceral fat, particularly when accompanied by elevated triglyceride levels. Simple measures such as waist circumference and serum triglycerides can help clinicians identify overweight or obese individuals with excess visceral or ectopic fat who are at higher cardiovascular risk (Després JP, 2012).<sup>[9]</sup>

Studies consistently show that higher BMI is associated with increased risk and progression of kidney disease. A significant proportion of cases are attributable to overweight and obesity, with stronger associations observed in women. Obesity also worsens outcomes in patients with pre-existing kidney disease (Wang Y et al., 2008).<sup>[10]</sup>

The prevalence of hypertension, diabetes, dyslipidemia, and metabolic syndrome rises progressively with increasing BMI. Compared with normal-weight individuals, those with class 3 obesity have markedly higher odds of hypertension, diabetes,

dyslipidemia, and metabolic syndrome. These findings underscore the significant public health impact of obesity and the need for effective preventive and therapeutic strategies (Nguyen NT et al., 2008).<sup>[11]</sup>

Individuals with abdominal obesity exhibited higher systolic and diastolic blood pressure levels compared with those without central fat accumulation. These findings suggest that visceral adiposity may play a particularly important role in the development of hypertension.

#### Sex and Age Differences

In large cohort study by Gelber RP et al., 2007, increasing BMI was associated with a progressively higher risk of developing hypertension, including among men with normal or mildly elevated BMI. These findings suggest that preventing excess weight gain may play an important role in reducing the future risk of hypertension.<sup>[6]</sup>

In a cohort of 82,473 U.S. female nurses followed biennially since 1976, higher BMI in early adulthood and midlife was independently associated with an increased risk of hypertension. Weight gain markedly elevated hypertension risk, while long-term weight loss significantly reduced risk, particularly among women with higher baseline BMI and younger age. These findings indicate that excess body weight and even modest adult weight gain substantially increase hypertension risk, whereas weight reduction is protective (Huang Z et al., 1998).<sup>[12]</sup>

Multivariate analysis showed no significant association between blood pressure and duration of menopause, while age and body mass index were key determinants of systolic and diastolic blood pressure. A temporary increase in blood pressure and BMI was observed after surgical, but not natural, menopause. Overall, postmenopausal blood pressure increases appear to be driven more by aging and weight gain than by ovarian failure itself (Portaluppi F et al., 1997).<sup>[13]</sup>

Sex differences significantly influence blood pressure, with premenopausal women exhibiting lower levels than age-matched men, while blood pressure increases after menopause. This pattern suggests a modulatory role of ovarian hormones, although their precise contribution remains uncertain (Dubey RK et al., 2002).<sup>[14]</sup>

**Table 1: Overview of included studies and main findings**

Study / Author (Year)	Main Findings Related to obesity and hypertension
Gelber RP et al., 2007	Higher BMI was independently associated with a progressively increased risk of incident hypertension, even within the normal BMI range.
Garrison RJ et al., 1987	Adiposity was the strongest independent predictor of hypertension in both men and women; increases in body fat were associated with rises in systolic and diastolic blood pressure.
Neter JE et al., 2003	Weight loss significantly reduced systolic and diastolic blood pressure, with greater reductions seen with larger weight loss and antihypertensive therapy.
Després JP, 2012	Increased waist circumference and triglycerides identified excess visceral fat associated with higher cardiometabolic and hypertension risk.
Wang Y et al., 2008	Higher BMI increased the risk and progression of kidney disease, indirectly contributing to hypertension, with stronger effects observed in women.
Nguyen NT et al., 2008	The prevalence and odds of hypertension increased progressively with higher BMI, particularly in individuals with class III obesity.

### **Metabolic and Clinical Correlates**

Many studies highlighted that obesity-related hypertension frequently coexisted with other metabolic abnormalities, including insulin resistance, dyslipidemia, and impaired glucose tolerance. Multivariate analysis showed that waist circumference and BMI were the strongest correlates of sympathetic activation. Baroreflex sensitivity was reduced in both heart failure groups and was more severely impaired in those with metabolic syndrome. These findings indicate that obesity and metabolic syndrome amplify sympathetic overactivity in heart failure, largely through metabolic and baroreflex dysfunction (Grassi G et al., 2007).<sup>[15]</sup>

Insulin resistance is associated with elevated circulating inflammatory markers and a cluster of established cardiovascular risk factors driven by compensatory hyperinsulinemia. Because these abnormalities are highly interrelated, identifying a single factor responsible for accelerated atherosclerosis is difficult, and reliance on multivariable epidemiologic analyses may be misleading (Reaven GM, 2005).<sup>[16]</sup>

### **Biological Mechanisms**

Multiple biological mechanisms explain the link between obesity and hypertension.

Obesity contributes to hypertension through sympathetic and renin–angiotensin–aldosterone system activation, along with endothelial and renal dysfunction. Emerging insights into appetite and metabolic regulators, including leptin resistance, ghrelin, and peptide YY, may enable novel therapeutic strategies for obesity-related hypertension (Rahmouni K et al., 2005).<sup>[17]</sup>

Obesity markedly increases the risk of cardiovascular disease and early mortality. Dysfunctional adipose tissue secretes bioactive mediators that promote insulin resistance, dyslipidemia, elevated blood pressure, prothrombotic states, and inflammation, ultimately contributing to endothelial dysfunction and atherosclerosis (Van Gaal LF et al., 2006).<sup>[18]</sup>

### **Clinical and Public Health Implications**

Obesity-related hypertension represents a growing public health challenge, as rising obesity rates are accompanied by increased cardiovascular risk. Although prevention remains the long-term goal, effective management of both obesity and hypertension in at-risk individuals is the current priority. Treatment of hypertension in obese patients must include weight management through lifestyle modification, supplemented when necessary with pharmacologic therapy, structured dietary and behavioral interventions, and, in selected cases, bariatric surgery (Landsberg L et al., 2013).<sup>[19]</sup>

The global rise in obesity is largely driven by technological advances that promote abundant low-cost food and sedentary lifestyles. Because obesity occurs within societies that emphasize personal choice, restrictive public health measures may be unacceptable, placing greater responsibility on individuals to adopt healthier behaviors (Grundy SM, 2004).<sup>[20]</sup>

Findings by Neter JE et al., 2003, provide strong evidence that weight reduction through lifestyle modification is a key component of hypertension management. Given the rising prevalence of overweight, population-level weight control is essential to reduce hypertension-related cardiovascular morbidity and mortality.<sup>[8]</sup>

The findings support targeted partnerships and strategic initiatives to translate evidence into action, while also highlighting the need to strengthen surveillance systems to better capture lifestyle behaviors and their policy, environmental, and industry-related determinants (Mozaffarian D et al., 2012).<sup>[21]</sup>

Routine assessment of obesity indices, particularly waist circumference, should be incorporated into hypertension risk assessment. Early identification and management of obesity may prevent or delay the onset of hypertension and reduce the need for long-term pharmacological treatment.

### **Limitations**

Future research should prioritize prospective cohort studies with standardized definitions to strengthen causal inference between obesity and hypertension. Use of precise adiposity measures, including visceral fat and body composition, may improve risk prediction. Mechanistic studies are needed to elucidate inflammatory, neurohormonal, and metabolic pathways. Long-term randomized trials should assess sustained effects of weight-loss interventions on blood pressure. Population- and sex-specific analyses will support targeted prevention and management strategies.

## **CONCLUSION**

This systematic review demonstrates a strong and consistent association between both general and central obesity and hypertension across diverse populations and study designs. Increasing body mass index and abdominal adiposity are associated with a progressively higher risk of developing hypertension, while weight reduction leads to clinically meaningful improvements in blood pressure. These findings highlight obesity as a key modifiable risk factor in the prevention and management of hypertension. Routine assessment of obesity indices, particularly waist circumference, and early implementation of effective weight management strategies should be integral components of hypertension risk reduction and public health interventions.

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