Role of Body Mass Index in Cardiovascular Efficiency: A Narrative Review

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Abstract

Background: Body mass index (BMI) is the most widely used proxy of adiposity in clinical and epidemiological practice, yet its relationship with cardiovascular efficiency—the capacity of the heart and circulation to deliver and utilise oxygen per unit of physiological cost—remains incompletely characterised.

Objective: This narrative review synthesises evidence on how BMI relates to cardiorespiratory fitness (VO_2max), cardiac structure and function, and cardiovascular outcomes.

Methods: Peer-reviewed cohort studies, cross-sectional analyses, meta-analyses and reviews published between 1989 and 2025 were retrieved from PubMed/MEDLINE, Scopus and Web of Science and synthesised narratively.

Findings: Higher BMI is consistently associated with lower VO_2max, the association being stronger in women and for central-adiposity measures. Elevated BMI drives volume overload, left ventricular hypertrophy and diastolic dysfunction, while higher fitness substantially attenuates mortality risk and frequently outperforms BMI as a prognostic marker. After established cardiovascular disease, an "obesity paradox" of improved survival is observed.

Conclusion: BMI is a useful but imperfect index of cardiovascular efficiency; integrating cardiorespiratory fitness and body-composition measures refines risk stratification.

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Keywords: Body mass index, Cardiorespiratory fitness, VO_2max, Obesity paradox, Left ventricular function, Cardiovascular efficiency

1. Introduction

Cardiovascular efficiency describes the ability of the cardiovascular system to supply oxygenated blood to working tissues at the lowest possible energetic and mechanical cost, and is operationally captured by maximal oxygen uptake (VO_2max) together with measures of myocardial mechanical performance [3, 7]. Body mass index, defined as weight in kilograms divided by height in metres squared, is the dominant population-level measure of adiposity because it is inexpensive, reproducible and strongly correlated with body fatness [1].

With more than a third of the world's adult population now overweight or obese, the interaction between body mass and cardiovascular performance has acquired major public-health relevance [1, 14]. Obesity is an established independent risk factor for hypertension, coronary artery disease and heart failure; a follow-up of the Framingham cohort showed roughly twice the heart-failure risk in obese versus normal-weight individuals [2]. However, the link between BMI and cardiovascular efficiency is neither simple nor monotonic. BMI cannot distinguish fat from lean mass or describe fat distribution, and after the onset of cardiovascular disease a higher BMI is paradoxically associated with better survival [4, 10, 12]. This review examines the strength, direction and clinical meaning of the relationship between BMI and cardiovascular efficiency, and considers where complementary measures are required.

2. Methodology

This is a narrative (non-systematic) review; no primary human or animal data were generated. PubMed/MEDLINE, Scopus and Web of Science were searched for English-language articles published between January 1989 and May 2025 using combinations of the terms "body mass index", "adiposity", "cardiorespiratory fitness", "VO_2max", "left ventricular function", "obesity paradox" and "cardiovascular mortality". Prospective cohort studies, cross-sectional analyses, meta-analyses and authoritative reviews reporting quantitative associations between BMI or related adiposity indices and a marker of cardiovascular efficiency or outcome were eligible. Reference lists of retrieved articles were screened for additional sources. Findings were extracted and synthesised thematically across three domains: (i) BMI and cardiorespiratory fitness; (ii) BMI and cardiac structure and function; and (iii) BMI, fitness and cardiovascular outcomes. Reported effect estimates are presented as published; no pooled re-analysis was performed.

Table 1: World Health Organization BMI classification and associated cardiovascular features.

BMI category	BMI (kg/m²)	Typical cardiovascular / haemodynamic features
Underweight	< 18.5	Reduced lean and cardiac mass; lower stroke volume reserve
Normal weight	18.5–24.9	Reference range; lowest cardiovascular burden
Overweight	25.0–29.9	Rising blood volume and cardiac output; early eccentric remodelling
Obesity class I	30.0–34.9	Left ventricular hypertrophy; diastolic dysfunction emerges
Obesity class II	35.0–39.9	Concentric remodelling; impaired relaxation and filling pressures
Obesity class III	≥ 40.0	Marked volume load; reduced VO_2max; heightened heart-failure risk

Source: classification thresholds per WHO [1]; haemodynamic correlates summarised from [4, 5].

3. Results

3.1. BMI and cardiorespiratory fitness

Across studies, higher BMI is inversely related to VO_2max. In a cross-sectional analysis of 403 healthy adults, BMI correlated negatively with calculated VO_2max in both sexes, with a markedly stronger association in women ($r = -0.51$) than in men ($r = -0.28$); waist circumference showed comparable, slightly stronger relationships (Table 2) [6]. These data indicate that adiposity accounts for a meaningful but partial share of the variance in fitness, and that central adiposity tracks fitness at least as closely as BMI. Importantly, evidence consistently shows that fat mass, rather than total body mass, drives the decline in fitness, so that a high BMI does not invariably signal low fitness [9, 15].

3.2. BMI, cardiac structure and function

Chronically elevated body mass increases circulating blood volume and cardiac output, imposing a sustained volume load that promotes eccentric and later concentric left ventricular hypertrophy, subclinical diastolic dysfunction and reductions in myocardial strain [4, 5]. These changes raise the metabolic cost of each cardiac cycle and lower mechanical efficiency. Their partial reversibility is instructive: in long-term follow-up of obese individuals, those achieving sustained weight loss displayed superior left ventricular systolic and diastolic function compared with weight-stable counterparts, underscoring a causal contribution of adiposity to impaired cardiac performance [13].

3.3. BMI, fitness and cardiovascular outcomes

At the population level, rising BMI predicts incident heart failure and increased cardiovascular mortality in primary prevention [2, 10]. Yet cardiorespiratory fitness exerts a powerful, partly independent influence: low fitness is a strong predictor of all-cause and cardiovascular mortality, and improvements in fitness are associated with reduced mortality irrespective of weight change [3, 7, 8]. A 2025 meta-analysis reported that being fit substantially attenuated the excess mortality associated with higher BMI, supporting the "fit-but-fat" concept [9]. Paradoxically, once cardiovascular disease or heart failure is established, overweight and mildly obese patients often show better survival than lean patients—the obesity paradox (Table 3) [10, 11, 12].

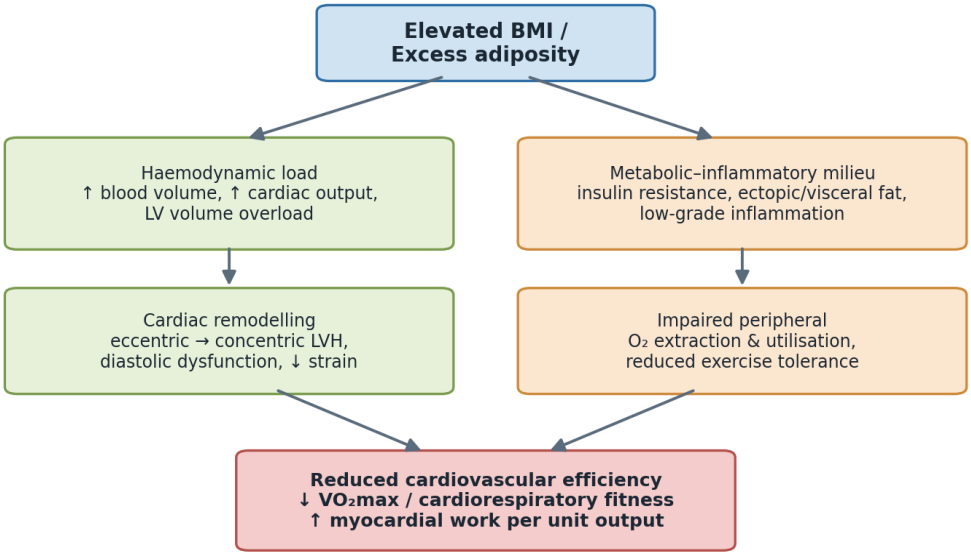

Fig 1: Principal haemodynamic and metabolic pathways through which elevated BMI reduces cardiovascular efficiency (synthesised from [4, 5, 13]).

Table 2: Reported correlations between adiposity indices and VO_2max, by sex (n = 403).

Sub-group	Adiposity index	Correlation with VO_2max (r)	Direction
Men (n = 222)	Body mass index	−0.28	Inverse
Men (n = 222)	Waist circumference	−0.38	Inverse
Women (n = 181)	Body mass index	−0.51	Inverse
Women (n = 181)	Waist circumference	−0.49	Inverse

Source: Dankner *et al*. [6]. All correlations negative and statistically significant.

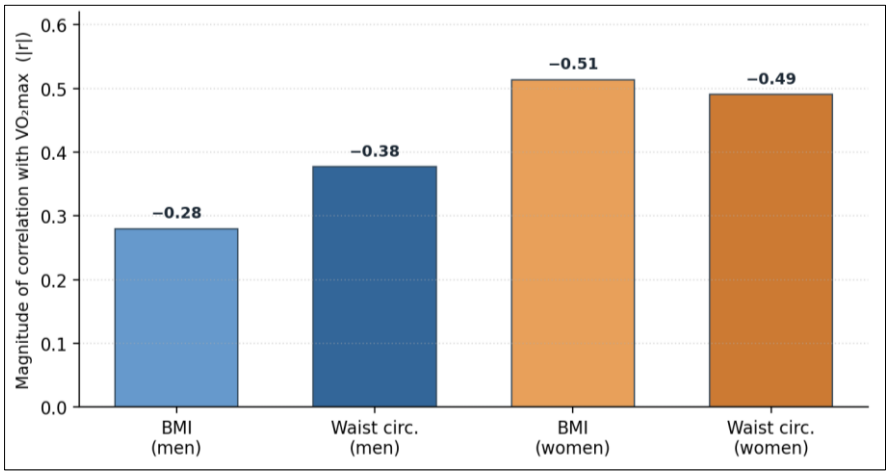

Fig 2: Magnitude of the inverse association (|r|) between adiposity indices and VO_2max, illustrating stronger coupling in women [6].

Table 3: BMI, cardiorespiratory fitness and cardiovascular outcomes: principal patterns.

Relationship / outcome	Observed direction	Representative evidence
Incident heart failure with rising BMI	↑ (≈ doubled in obesity)	Framingham cohort [2]
All-cause / CVD mortality, high BMI (primary prevention)	↑	Pooled cohorts [3,4]
Mortality after established CVD or heart failure	↓ in overweight / mild obesity ('paradox')	HF & CAD cohorts [10,11]
Cardiorespiratory fitness vs BMI as predictor	CRF often the stronger predictor	ACLS; meta-analysis [7,9]

CVD, cardiovascular disease; CRF, cardiorespiratory fitness; ACLS, Aerobics Center Longitudinal Study.

4. Discussion

The evidence indicates a real but modest inverse relationship between BMI and cardiovascular efficiency that is mediated by adiposity-driven cardiac and metabolic changes rather than by body mass per se. Three features qualify any simple interpretation. First, the association is sex-dependent and is captured at least as well by central-adiposity measures, implying that fat distribution carries information BMI omits [6, 15]. Second, because BMI conflates fat and lean mass, muscular individuals may be misclassified as high-risk despite preserved fitness, while “normal-weight” individuals with high visceral fat may be falsely reassured [9,14,15]. Third, cardiorespiratory fitness frequently outperforms BMI as a prognostic marker, and the survival of fit individuals across BMI strata supports prioritising fitness in clinical counselling [3, 7, 8].

The obesity paradox illustrates the limits of BMI as an efficiency index. The apparent protective effect of higher BMI after established disease is at least partly explained by reverse causation—illness-related weight loss and cachexia—and by the confounding influence of fitness and muscle mass, several studies reporting that the paradox weakens after adjustment for cardiorespiratory fitness [11, 12]. Clinically, these findings argue for interpreting BMI alongside waist circumference and an objective measure of fitness rather than in isolation. The reversibility of obesity-related cardiac dysfunction with sustained weight loss reinforces the value of combined weight-management and exercise strategies that improve cardiovascular efficiency independently of the number on the scale [9, 13].

5. Conclusion

BMI is inversely but only moderately related to cardiovascular efficiency, acting largely through adiposity-induced increases in cardiac workload and reductions in cardiorespiratory fitness. It remains a valuable, accessible screening tool, but it is an incomplete surrogate: it neither distinguishes body-composition compartments nor captures the prognostic weight of fitness, and it behaves paradoxically in established disease. Cardiovascular risk stratification is best served by combining BMI with measures of central adiposity and cardiorespiratory fitness, with fitness improvement a primary therapeutic target alongside weight control.

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