

# Effect of Body Mass Index on Cardiorespiratory Fitness: A Review

Aissyawa Febilia Fertien<sup>1</sup>, I Ketut Ngurah Sajjana Kirthana Pamecut<sup>1</sup>, Hayuris Kinandita Setiawan<sup>2\*</sup>, Yetti Hernaningsih<sup>3</sup>, Misbakhul Munir<sup>2</sup>

<sup>1</sup>Medical Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia - 60131

<sup>2</sup>Department of Medical Physiology and Biochemistry, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia – 60131

<sup>3</sup>Department of Clinical Pathology, Faculty of Medicine, Dr. Soetomo Hospital, Surabaya, Indonesia – 60131

\*Corresponding Author: hayuris-k-s@fk.unair.ac.id

**Abstract**—Body Mass Index (BMI) is a widely used anthropometric indicator for classifying weight status, yet it does not distinguish between fat mass and lean mass. In contrast, cardiorespiratory fitness (CRF), typically assessed by maximal oxygen uptake ( $VO_{2max}$  or  $VO_{2peak}$ ), represents a key determinant of aerobic capacity and a robust predictor of cardiovascular and all-cause mortality. Evidence consistently demonstrates an inverse association between BMI and CRF, where higher BMI, particularly driven by excess adiposity, is associated with reduced aerobic capacity. This relationship is explained by several physiological mechanisms, including increased mechanical load during movement, unfavorable body-composition profiles, systemic low-grade inflammation, metabolic dysfunction, and ventilatory limitations that collectively impair oxygen delivery, utilization, and exercise tolerance. The BMI–CRF relationship is further shaped by important confounding factors such as habitual physical activity, age, sex, fat distribution, comorbidities, and methodological choices in  $VO_{2max}$  expression. These factors highlight that BMI alone is insufficient to characterize an individual's aerobic capacity. From a clinical and public-health perspective, CRF emerges as a powerful modifier of health risks, with evidence showing that individuals with elevated BMI but high CRF exhibit substantially lower mortality risk compared to unfit individuals regardless of BMI category. Overall, understanding how BMI influences CRF is essential for guiding prevention strategies and risk assessment. Integrating CRF evaluation alongside BMI in clinical settings and population surveillance provides a more comprehensive assessment of cardiometabolic health and supports dual-target interventions aimed at both reducing adiposity and enhancing fitness to optimize long-term health outcomes.

**Keywords**— Body Mass Index, Cardiorespiratory Fitness, Obesity,  $VO_{2max}$ , Health Risk Factors.

## I. INTRODUCTION

Body Mass Index (BMI) is a widely used anthropometric indicator for classifying weight status and assessing population-level health risks. BMI is calculated as body mass divided by the square of height ( $kg/m^2$ ) [6]. Meanwhile, cardiorespiratory fitness (CRF) that commonly measured via maximal oxygen uptake ( $VO_{2max}$ ) or  $VO_{2peak}$  is considered a key physiological parameter reflecting the integrated function of the cardiovascular and respiratory systems to supply oxygen during sustained aerobic activity [14].

A growing body of evidence demonstrates an inverse association between BMI (or adiposity) and CRF, indicating that higher BMI, particularly when due to excess fatness, tends

to be associated with lower aerobic capacity. Several physiological mechanisms have been proposed to explain this relationship can increased mechanical load during physical activity, unfavorable changes in body composition (higher fat mass, lower relative lean mass), and adiposity-related metabolic or inflammatory effects that may impair oxygen utilization, muscle function, or cardiopulmonary efficiency [15]. Cross-sectional and observational studies, including populations of healthy adults, confirm that elevated BMI is often accompanied by reduced CRF (lower  $VO_{2max}$  or  $VO_{2peak}$ ) compared to individuals with normal BMI [11].

Beyond this direct association, recent systematic reviews and meta-analyses highlight CRF as a powerful modifier of health risks associated with overweight and obesity. For example, a large meta-analysis combining CRF and BMI data found that overweight or obese individuals but with “fit” cardiorespiratory levels did not have statistically significantly higher risk of cardiovascular disease (CVD) or all-cause mortality compared to normal-weight, “fit” individuals; in contrast, “unfit” individuals (regardless of BMI) had a 2–3 times greater risk of mortality [12]. This suggests that high CRF can attenuate, though not necessarily eliminate, the elevated health risks commonly associated with high BMI.

From a public health perspective, this interplay between BMI and CRF is highly relevant. Given the rising global prevalence of overweight and obesity and their relation to non-communicable diseases (e.g., cardiovascular disease, metabolic syndrome), interventions focusing on improving CRF (e.g., structured exercise, increasing physical activity) may provide substantial health benefit — even in absence of dramatic weight loss. Indeed, improving CRF has been associated with better health outcomes across BMI categories [15].

Therefore, understanding the effect of BMI on CRF is important for shaping effective prevention strategies, mitigating CVD risk, and improving long-term health outcomes.

## II. METHODS

This study was a focused narrative literature review that aimed to bring jointly the most recent research about how body mass index can affect cardiorespiratory fitness.

### III. RESULT

#### 3.1 Physiological Mechanisms Linking BMI and Cardiorespiratory Fitness

Body Mass Index (BMI) reflects total body mass relative to height but does not discriminate between fat mass and lean body mass, which has important physiological implications for CRF because adipose tissue and muscle mass influence oxygen delivery and utilization differently. For example, higher adiposity increases the metabolic cost of locomotion and elevates oxygen demand at submaximal workloads, which can  $VO_{2max}$  when expressed relative to body weight. [7].

Obesity drives hemodynamic changes that increase cardiac workload, such as elevated resting heart rate and cardiac output, thereby promoting cardiac remodeling and reduced stroke volume reserve during exercise, which ultimately limits exercise capacity [7].

Excess adipose tissue, particularly visceral fat, secretes pro-inflammatory adipokines and cytokines that contribute to systemic low-grade inflammation, endothelial dysfunction, and impaired vasodilation, all of which reduce skeletal muscle perfusion and oxygen extraction during exertion. Excess adipose tissue also increases arterial stiffness and atherogenic risk, further compromising cardiovascular compliance; this stiffening of large and small vessels increases left ventricular afterload and reduces stroke volume augmentation during dynamic exercise, thereby restricting maximal cardiac output and aerobic capacity [5].

Obesity is also associated with metabolic disruptions, including insulin resistance and altered lipid metabolism, which adversely affect mitochondrial function and reduce oxidative phosphorylation capacity in skeletal muscle, key determinants of  $VO_{2max}$ . [10].

Moreover, mechanical effects of excess fat mass such as reduced chest wall and diaphragmatic compliance and increased work of breathing can further constrain ventilatory efficiency during exercise, lowering exercise tolerance especially at high intensities [3].

#### 3.2 Confounding Factors Influencing the BMI–CRF Relationship

Confounding factors substantially influence the observed relationship between BMI and cardiorespiratory fitness (CRF), and careful methodological consideration is essential to avoid biased inferences. BMI itself is an imperfect surrogate of adiposity and body composition; the failure to account for differences in fat distribution or lean mass often leads to misclassification, particularly in individuals with higher muscularity, thereby distorting associations with CRF (2)

Physical activity and habitual exercise represent strong, independent determinants of  $VO_{2max}$ , and cross-sectional assessments of BMI–CRF relationships are therefore highly susceptible to confounding when activity level is not rigorously measured and appropriately adjusted for [1,8].

Age and sex systematically influence both body composition and peak aerobic capacity, older adults and females typically show different trajectories of lean mass loss and  $VO_{2max}$  decline, so stratified or interaction analyses are often necessary. [12]

Comorbid conditions that commonly co-occur with elevated BMI (for example, hypertension, dyslipidemia, and insulin resistance) independently lower CRF and may confound the apparent effect of BMI unless multivariable adjustment is applied [1,12].

Broader socioeconomic, behavioral, and environmental determinants, including diet quality, smoking habits, and access to safe recreational spaces, also correlate strongly with both adiposity and fitness, acting as contextual confounders that shape population-level estimates of the BMI–CRF association (2).

Measurement choices, whether  $VO_{2max}$  is reported as absolute, per kg total mass, or per kg fat-free mass, substantially change observed associations and should be pre-specified and justified to avoid scaling artefacts [8].

#### 3.3 Clinical and Public Health Implications

A growing body of high-quality evidence demonstrates that CRF is a strong and independent predictor of both cardiovascular and all-cause mortality, with higher levels of fitness markedly attenuating the excess mortality risk typically associated with overweight and obesity (2). Because BMI alone does not capture fitness, fat distribution, or lean mass, clinical risk stratification is improved when CRF (measured directly or estimated reliably) is integrated with anthropometry in routine assessment [12].

The predictive utility of CRF persists across clinical subpopulations. In adults with overweight/obesity and type 2 diabetes, higher baseline CRF, as quantified by maximal exercise treadmill testing, was associated with significantly lower rates of both all-cause and cardiovascular disease (CVD) mortality over long-term follow-up, independent of BMI and other conventional risk factors [13]. Similarly, among men with metabolic syndrome, inclusion of CRF in multivariate models attenuated the association between metabolic syndrome and mortality to non-significance, while unfit individuals consistently demonstrated double the risk of all-cause mortality and more than triple the risk of CVD death compared to their fit counterparts [9].

Randomized and nonrandomized intervention studies demonstrate that structured aerobic and resistance training consistently increase  $VO_{2max}$  and vascular function even when weight loss is modest or absent, supporting the clinical value of fitness-focused therapies (1).

Although lifestyle strategies aimed at reducing adiposity, including behavioral modification, pharmacotherapy, and bariatric surgery, can improve traditional cardiometabolic risk markers (e.g., glycemic control, lipids, blood pressure), converging evidence suggests that the combination of adiposity reduction and fitness enhancement yields the most favorable long-term cardiovascular outcomes. Improving CRF augments energy expenditure capacity, improves insulin sensitivity, and favorably modulates vascular and inflammatory pathways, while also enhancing resilience to noncardiovascular stressors such as severe infectious disease [4].

Weight-reduction strategies (behavioral, pharmacologic, surgical) can improve many cardiometabolic risk markers, but the combination of interventions that target both adiposity

reduction and fitness enhancement is likely to produce the most favorable long-term cardiovascular outcomes. At the population level, public-health policies that increase opportunities for physical activity, improve food environments, and monitor both BMI and CRF will better address the twin burdens of low fitness and excess adiposity than BMI-centric approaches alone (2).

#### IV. CONCLUSION

The present review demonstrates that the relationship between BMI and cardiorespiratory fitness is multifactorial, physiologically interconnected, and influenced by a range of contextual factors. Consistent with the evidence summarized in the introduction, the findings reinforce that higher BMI, particularly when driven by excess adiposity, is commonly associated with lower CRF due to an interplay of unfavorable alterations in cardiovascular load, systemic inflammation, metabolic dysfunction, ventilatory mechanics, and reduced relative lean mass.

These mechanisms collectively impair oxygen delivery, utilization, and exercise tolerance, thereby explaining the inverse association observed across numerous adult populations. At the same time, the review highlights that BMI alone cannot fully characterize the BMI-CRF relationship because several confounders (including body composition, habitual physical activity, age, sex, comorbidities, and methodological variations in  $VO_2$ max measurement) substantially modify the association. Such factors may either attenuate or amplify the effect of BMI on aerobic capacity, underscoring the importance of interpreting CRF within a broader physiological and behavioral context rather than relying on BMI as a standalone indicator.

Importantly, the clinical and public-health implications converge with the introduction's emphasis on CRF as a strong modifier of health outcomes across BMI categories. The evidence reviewed supports that high CRF can mitigate, though not completely eliminate, the elevated cardiometabolic and mortality risks associated with overweight and obesity.

This reinforces the growing consensus that interventions aimed at improving CRF, through structured aerobic and resistance training, increased physical activity, and supportive environmental policies, offer substantial health benefits even in the absence of significant weight reduction. Overall, understanding the effect of BMI on CRF is crucial for guiding prevention strategies, clinical risk stratification, and policy development. Integrating CRF assessment alongside BMI in both clinical practice and population surveillance provides a more accurate appraisal of cardiometabolic health and supports targeted interventions that address both excess adiposity and low fitness. This dual-focus approach is likely to be more effective in reducing cardiovascular disease burden and improving long-term health outcomes globally.

#### ACKNOWLEDGMENT

The authors gratefully acknowledge the Faculty of Medicine, Universitas Airlangga, and Dr. Soetomo General Hospital, Surabaya, Indonesia, for their support in facilitating the development of this review.

#### REFERENCES

- 1) Babu, A. F., Csader, S., Lok, J., Gómez-Gallego, C., Hanhineva, K., El-Nezami, H., & Schwab, U. (2021). Positive effects of exercise intervention without weight loss and dietary changes in nafld-related clinical parameters: A systematic review and meta-analysis. *In Nutrients* (Vol. 13, Issue 9). MDPI. <https://doi.org/10.3390/nu1309135>
- 2) Badrić, M., Roca, L., Pelemiš, V., Branković, D., & Živanović, V. (2024). Indicators of Obesity and Cardiorespiratory Fitness in Croatian Children. *Journal of Functional Morphology and Kinesiology*, 9(4). <https://doi.org/10.3390/jfink9040250>
- 3) Battista, F., Neunhaeuserer, D., Centanini, A., Gasperetti, A., Quinto, G., Vecchiato, M., Bianchi, E., Frigo, A. C., Bettini, S., Vettor, R., Busetto, L., & Ermolao, A. (2023). The "Aging Effect" of BMI on Cardiorespiratory Fitness: A New Insight on Functional Evaluation in Obesity. *Journal of Clinical Medicine*, 12(22). <https://doi.org/10.3390/jcm12227183>
- 4) Ekblom-Bak, E., Väisänen, D., Ekblom, B., Blom, V., Kallings, L. V., Hemmingsson, E., Andersson, G., Wallin, P., Salier Eriksson, J., Holmlund, T., Lindwall, M., Stenling, A., & Lönn, A. (2021). Cardiorespiratory fitness and lifestyle on severe COVID-19 risk in 279,455 adults: a case control study. *International Journal of Behavioral Nutrition and Physical Activity*, 18(1). <https://doi.org/10.1186/s12966-021-01198-5>
- 5) Fico, B. G., Maharaj, A., Pena, G. S., & Huang, C. J. (2023). The Effects of Obesity on the Inflammatory, Cardiovascular, and Neurobiological Responses to Exercise in Older Adults. *In Biology* (Vol. 12, Issue 6). MDPI. <https://doi.org/10.3390/biology12060865>
- 6) Frank Q. Nuttall, M. P. (2015). Obesity, BMI, and Health: A Critical Review. *Nutrition Research*.
- 7) Haidar, A., & Horwich, T. (2023). Obesity, Cardiorespiratory Fitness, and Cardiovascular Disease. *In Current Cardiology Reports* (Vol. 25, Issue 11, pp. 1565–1571). Springer. <https://doi.org/10.1007/s11886-023-01975-7>
- 8) Han, M., Qie, R., Shi, X., Yang, Y., Lu, J., Hu, F., Zhang, M., Zhang, Z., Hu, D., & Zhao, Y. (2022). Cardiorespiratory fitness and mortality from all causes, cardiovascular disease and cancer: dose-response meta-analysis of cohort studies. *British Journal of Sports Medicine*, 56(13), 733–739. <https://doi.org/10.1136/bjsports-2021-104876>
- 9) Katzmarzyk, P. T., Church, T. S., & Blair, S. N. (2016). Cardiorespiratory Fitness Attenuates the Effects of the Metabolic Syndrome on All-Cause and Cardiovascular Disease Mortality in Men. <http://archinte.jamanetwork.com/>
- 10) Men, J., Yu, Z., An, W., Wang, P., Hou, X., Zhang, Y., Wu, S., Zhu, G., Wang, P., Cui, C., Zhang, Y., Wang, J., Ding, J., & Wang, Y. (2025). Effects of exercise on cardiorespiratory fitness in children and adolescents with overweight and obesity: a systematic review and meta-analysis of 72 randomized controlled trials. *In BMC Public Health* (Vol. 25, Issue 1). BioMed Central Ltd. <https://doi.org/10.1186/s12889-025-25254-y>
- 11) Takken, T., Hulzebos, H. J., Schmitz, M., van Ooi, P. J., van Beek, G., van Galen, L., Molinger, J., Rozenberg, R., van den Oord, M., Hartman, Y., Verbaarschot, N., Snoek, A., Stomphorst, J., & van Kesteren, J. (2022). Is BMI Associated with Cardiorespiratory Fitness? A Cross-Sectional Analysis Among 8470 Apparently Healthy Subjects Aged 18–94 Years from the Low-Lands Fitness Registry. *Journal of Science in Sport and Exercise*, 4(3), 283–289. <https://doi.org/10.1007/s42978-021-00143-z>
- 12) Weeldreyer, N. R., De Guzman, J. C., Paterson, C., Allen, J. D., Gaesser, G. A., & Angadi, S. S. (2025). Cardiorespiratory fitness, body mass index and mortality: a systematic review and meta-analysis. *British Journal of Sports Medicine*, 59(5), 339–346. <https://doi.org/10.1136/bjsports-2024-108748>
- 13) Wills, A. C., Vazquez Arreola, E., Olaiya, M. T., Curtis, J. M., Hellgren, M. I., Hanson, R. L., & Knowler, W. C. (2022). Cardiorespiratory Fitness, BMI, Mortality, and Cardiovascular Disease in Adults with Overweight/Obesity and Type 2 Diabetes. *Medicine and Science in Sports and Exercise*, 54(6), 994–1001. <https://doi.org/10.1249/MSS.0000000000002873>
- 14) Wirdati, I. E., Muhibbi, M., Muzaqi, L., & Hasan, C. R. (2025). Cardiorespiratory Fitness Level of College Students in terms of Abdominal Circumference, Body Mass Index, and Gender. *Journal of Physical Education, Sport, Health and Recreation*, 14(2), 708–714. <https://journal.unnes.ac.id/journals/peshr>



- 15) Zeiher, J., Ombrellaro, K. J., Perumal, N., Keil, T., Mensink, G. B. M., & Finger, J. D. (2019). Correlates and Determinants of Cardiorespiratory Fitness in Adults: a Systematic Review. In *Sports Medicine - Open* (Vol. 5, Issue 1). Springer. <https://doi.org/10.1186/s40798-019-0211-2>