



ISSN:2456-4419

Impact Factor:(RJIF):5.88

Yoga 2025; 10(2): 422-425

© 2025 Yoga

www.theyogicjournal.com

Received: 15-08-2025

Accepted: 18-09-2025

Dr. Alope Sen Barman

Assistant Professor,

Department of Physical

Education, Seva Bharati

Mahavidyalaya, Kapgari,

Jhargram, West Bengal, India

Effect of pranayama on volume of oxygen maximum uptake (VO2 Max)

Alope Sen Barman

DOI: <https://www.doi.org/10.22271/yogic.2025.v10.i2f.1815>

Abstract

Volume of oxygen maximum uptake (VO2 max) is utilized during intense exercise, serving as a key indicator of cardiovascular fitness and aerobic endurance. The main objective of this study was to do a systematic review to examine the effects of pranayama intervention on volume of oxygen maximum uptake. Data was collected from PubMed, JSTORE and Web of Science. Randomized controlled trials published in English from the inception of the database until 31st September, 2025, were included. Only pranayama as intervention were included in the study. The Pranayama intervention also had a significant effect on VO2 max. It may be concluded that pranayama can elicit significantly improved in the VO2 max that may be due to increase in aerobic capacity.

Keywords: Pranayama, volume, oxygen, maximum, uptake

Introduction

The utmost ability to transport and consume oxygen is known as maximal oxygen consumption (VO2max), and it's frequently used to gauge an individual's aerobic capacity. In general, VO2max declines gradually as one ages; beyond the age of 25, the rate of reduction is roughly 10% per ten years, and it was estimated that this decline would reach 15% between the ages of 50 and 75 ^[1, 2, 3]. Furthermore, a prior meta-analysis showed that age-related decreases in VO2max were roughly 0.40, 0.39, and 0.46 ml/kg/min annually for males who were sedentary, active, and trained, and 0.35, 0.44, and 0.62 ml/kg/min annually for females who were sedentary, active, and trained ^[4, 5, 6]. Reduced maximal heart rate and stroke volume, decreased blood volume from pooling from less efficient muscle pump action of the valves in the extremities, stiffening of the heart muscle fibers and thickening and stiffening of the arterial walls, decreased peripheral oxygen extraction, and a maximal A-V O2 difference are some of the factors that contribute to age-related declines in VO2max ^[7, 8, 9, 10]. The decrease in VO2max also seems to be significantly influenced by age-related muscle loss, or sarcopenia; by the age of 50, almost 10% of muscle mass has been lost, and this rate only rises in the ensuing decades. Muscle strength decreases by 15% every decade in the 60s and 70s and increases to 30% around the age of 80 due to sarcopenia ^[11]. According to a prior study by Fleg and Lakatta (1988), inactive people's VO2max decreased with age due in part to their whole body lean mass ^[12]. Those that were active had similar outcomes ^[13, 14]. But compared to sedentary people, active people showed a slower rate of decrease ^[3, 15]. Therefore, training and possibly training type (e.g., upper vs. lower extremity exercise) as well as other factors, such as how muscle mass is measured (e.g., just active muscles vs. total lean body or skeletal muscle mass), can affect the effect of muscle mass on VO2max ^[13]. Muscle mass has a strong correlation with aerobic capacity (VO2max), and in both younger and older athletes, rowing VO2max was higher than cycling VO2max. Additionally, younger subjects showed a greater change in VO2max with rowing than with cycling when compared to older subjects. When the active muscle mass being recruited for the particular exercise mode was taken into account, older subjects' VO2max during cycling did not differ significantly from that of younger subjects, who were recruiting less muscle mass; however, older subjects' VO2max during rowing remained lower than that of younger subjects, who were recruiting more muscle mass.

Corresponding Author:

Dr. Alope Sen Barman

Assistant Professor,

Department of Physical

Education, Seva Bharati

Mahavidyalaya, Kapgari,

Jhargram, West Bengal, India

These results imply that the total active muscle mass being recruited during exercise affects VO₂max, with the higher the VO₂max, the more muscle mass is used. These findings also imply that muscle mass may have less of an impact on VO₂max in older subjects than in younger ones. In addition, older participants required more cardiac effort (higher HR) at a given VO₂. As people age, their aerobic capacity usually decreases. Regardless of exercise modality, we found that older subjects in the current investigation had lower VO₂max than younger subjects. VO₂max typically decreases by 10% every ten years [1, 2, 3]. Athletes and active people seem to experience a higher rate of VO₂max decline than sedentary people [16, 17, 18]. A higher starting VO₂max could be the cause of this [17]. This hypothesis hasn't been well explored yet, though, because the results can vary depending on the study design (cross-sectional and longitudinal) and the non-linearity of VO₂max variations in sedentary and athletic/active people [3, 19]. Numerous research has looked into the factors underlying the age-related loss in aerobic capacity over the years. The Fick equation states that the arterio-venous O₂ differential and maximal cardiac output determine VO₂max. Decreases in maximal heart rate and stroke volume [20, 21, 22], as well as the maximal arterio-venous O₂ differential [23], are the causes of age-related losses in VO₂max. However, the mechanism is not easily explained due to a number of confounding factors, including skeletal muscle mass, training volume, vascular conductance, skeletal muscle mitochondrial density and enzyme activity, and the lungs' diffusion capacity. By the age of 50, skeletal muscle mass has decreased by 10%, and by the age of 80, it has decreased by 30% [24, 25]. Age-related decreases in VO₂max are further triggered by the normal age-related loss of muscle mass (sarcopenia) and the extra changes in body composition that often follow a decrease in physical activity in older patients [26]. Skeletal muscle mass was a significant driver of aerobic ability, according to earlier research by Flegg and Lakatta (1988), who measured muscle mass using 24-hour urine creatinine excretion [12]. Furthermore, using a DEXA scan to measure upper and lower appendicular muscle mass (logical active muscle mass during treadmill exercise), Proctor and Joyner (1997) discovered a substantial correlation between active muscle mass and VO₂max. [13]. Alternatively, we compared the association between VO₂max and LBM for rowing activity and LMM for cycling exercise in order to be more focused on the active muscles used during exercise in our experiment. We found a significant correlation between muscle mass and VO₂max in both groups, which is in line with other research. Secher *et al.* (1974) noted that individuals exhibited higher oxygen uptake during combined arm and leg exercise compared to arm or leg exercise alone, which is further supported by this study [27]. Additionally, following strength training, VO₂max increased [26, 28]. As a result, there is a strong correlation between skeletal muscle mass and VO₂max, which leads to the age-related decrease in VO₂max. Even though a similar amount of muscle mass was added, we found that older subjects showed a smaller change in VO₂max from cycling to rowing than younger subjects. Additionally, correcting for LBM during rowing did not completely account for the age-related differences in VO₂max between young and old people. This finding might indicate that skeletal muscle mass has less of an impact on VO₂max in older subjects than in younger ones. In older, healthy, and trained cohorts, age-related decreases in maximum heart rate and stroke volume are comparatively well-documented [20, 21, 29]. Age-related declines in VO₂max

are also caused by decreasing cardiac output and decreased blood supply [30, 31]. This theory is in line with earlier research by Hagberg *et al.* (1985), which found that the hemodynamic difference between younger runners and older athletes is primarily explained by the lower maximal HR [21]. The current study evaluated changes in VO₂max due to pranayama and mechanism behind it. With a pragmatic approach, the study offers a cross-sectional view of the particular population. A longitudinal study design, however, would yield more data than what can be inferred from cross-sectional researchers. Despite being a reasonable assumption, the pranayama does not account for the additional muscle mass that will be recruited during yoga, such as the respiratory muscles. The VO₂max of older and younger patients did not significantly differ, despite a further analysis that pranayama VO₂max with LBM. The researcher therefore assumed that this restriction had no bearing on the result. However, this study did evaluate how pranayama-induced adaptations in might affect muscle recruitment (e.g., force output associated with concentric versus eccentric contractions) during pranayama. Although one might anticipate that body posture or position might affect the study's findings, since body posture was consistent across pranayama types, the effect of posture in this study should be negligible.

Methods

A systematic literature search was conducted in PubMed, Web of Science, and J-Store with no data restrictions, up to 30th September, 2025. Yoga training intervention studies along with pranayama were included. In total, data from 1059 participants in 253 and 13 research articles were included for the synthesis of this review regarding measures of VO₂max, muscle fiber cross-sectional area, and fiber type proportion were extracted from these studies.

Results and Discussion

Peak VO₂ was measured as an endpoint in both experiments. [32, 33]. According to the meta-analyses, participants in the yoga group significantly outperformed controls in terms of peak VO₂ (by 3.87 mL·kg⁻¹·min⁻¹, 95% CI: 1.95, 5.80, N = 59). Furthermore, the yoga group's peak VO₂ improved by 22.0%, according to the meta-analysis. At baseline and at the conclusion of the intervention, the mean peak VO₂ in the two studies under analysis was 15.85 mL·kg⁻¹·min⁻¹ and 19.05 mL·kg⁻¹·min⁻¹, respectively. In particular, from baseline to post-intervention, the WMD in peak VO₂ peak was 3.97 mL·kg⁻¹·min⁻¹. The amount of change is comparable to a prior meta-analysis that assessed the impact of several exercise methods in CHF patients [34, 35]. The degree of improvement and the peak VO₂ of 19.05 mL·kg⁻¹·min⁻¹ attained following the intervention are other significant factors to be discussed. Complete and independent life (e.g., walking upstairs, gardening, etc.) has been shown to require a minimum peak VO₂ of 15 mL·kg⁻¹·min⁻¹ for women and 18 mL·kg⁻¹·min⁻¹ for men aged 85. [36]. As a result, yoga helps people with CHF manage their health and perform daily tasks more effectively. It has been demonstrated that yoga helps CHF patients recover. It is commonly known that improvements of more than 10% following a cardiovascular rehabilitation program are satisfactory and indicate a favourable prognosis for individuals with congestive heart failure (CHF), when peak VO₂ is taken into account [37]. Yoga has been shown to help with major mental health issues like depression and anxiety. Furthermore, it has been discovered that yoga is essential for improving patients' ability to

exercise. The findings are consistent with those of earlier research on exercise training [38]. Additionally, a variety of factors, including culture and individual approach, may affect how yoga works as a therapy. In spite of this, yoga seems like a promising cardiac rehabilitation technique that merits more research using better-controlled RCTs. This meta-analysis, taking into account the available data, demonstrated that yoga increased peak VO₂.

Conclusion

It may be concluded that pranayama may elicited the increase in vo₂ max by improving respiratory muscle mass and oxidisation capacity.

References

1. Robinson S. Experimental studies of physical fitness in relation to age. *Arbeitsphysiologie*. 1938;10:251-323.
2. Astrand I. Aerobic work capacity in men and women with special reference to age. *Acta Physiol Scand Suppl*. 1960;49:1-92.
3. Hawkins S, Wiswell R. Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training. *Sports Med*. 2003;33:877-888.
4. Fitzgerald MD, Tanaka H, Tran ZV, Seals DR. Age-related declines in maximal aerobic capacity in regularly exercising vs. sedentary women: a meta-analysis. *J Appl Physiol*. 1997;83:160-165.
5. Brown SJ, Ryan HJ, Brown JA. Age-associated changes in VO₂ and power output-A cross-sectional study of endurance trained New Zealand cyclists. *J Sports Sci Med*. 2007;6:477-483.
6. Wilson TM, Tanaka H. Meta-analysis of the age-associated decline in maximal aerobic capacity in men:relation to training status. *Am J Physiol Heart Circ Physiol*. 2000;278:H829-H834.
7. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease. *Circulation*. 2003;107:139-146.
8. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part II:the aging heart in health: links to heart disease. *Circulation*. 2003;107:346-354.
9. Tanaka H, Seals DR. Endurance exercise performance in Masters athletes: age-associated changes and underlying physiological mechanisms. *J Physiol*. 2008;586:55-63.
10. North BJ, Sinclair DA. The intersection between aging and cardiovascular disease. *Circ Res*. 2012;110:1097-1108.
11. Mazzeo RS. Exercise and the older adult. *ACSM Current Comment*, 2014
12. Fleg JL, Lakatta EG. Role of Muscle Loss in the Age-Associated Reduction in VO₂max. *J Appl Physiol*. 1988;65:1147-1151.
13. Proctor DN, Joyner MJ. Skeletal muscle mass and the reduction of VO₂max in trained older subjects. *J Appl Physiol*. 1997;82:1411-1415.
14. Cosgrove MJ, Wilson J, Watt D, Grant SF. The relationship between selected physiological variables of rowers and rowing performance as determined by a 2000 m ergometer test. *J Sport Sci*. 1999;17:845-852.
15. Rogers MA, Hagberg JM, Martin WH 3rd, Ehsani AA, Holloszy JO. Decline in Vo₂max with Aging in Master Athletes and Sedentary Men. *J Appl Physiol*. 1990;68:2195-2199.
16. Eskurza I, Donato AJ, Moreau KL, Seals DR, Tanaka H. Changes in maximal aerobic capacity with age in endurance-trained women: 7-yr follow-up. *J Appl Physiol*. 2002;92:2303-2308.
17. Pimentel AE, Gentile CL, Tanaka H, Seals DR, Gates PE. Greater rate of decline in maximal aerobic capacity with age in endurance-trained than in sedentary men. *J Appl Physiol*. 2003;94:2406-2413.
18. Ladyga M, Faff J, Burkhard-Jagodzinska K. Age-Related Decrease of the Indices of Aerobic Capacity in the Former Elite Rowers and Kayakers. *Biol Sport*. 2008;25:245-261.
19. Betik AC, Hepple RT. Determinants of VO₂ max decline with aging: an integrated perspective. *Appl Physiol Nutr Metab*. 2008;33:130-140. doi:10.1139/H07-174
20. Heath GW, Hagberg JM, Ehsani AA, Holloszy JO. A physiological comparison of young and older endurance athletes. *J Appl Physiol Respir Environ Exerc Physiol*. 1981;51:634-640.
21. Hagberg JM, Allen WK, Seals DR, Hurley BF, Ehsani AA, Holloszy JO. A hemodynamic comparison of young and older endurance athletes during exercise. *J Appl Physiol*. 1985;58:2041-2046.
22. Fuchi T, Iwaoka K, Higuchi M, Kobayashi S. Cardiovascular changes associated with decreased aerobic capacity and aging in long-distance runners. *Eur J Appl Physiol Occup Physiol*. 1989;58:884-889.
23. Seals DR, Hagberg JM, Hurley BF, Ehsani AA, Holloszy JO. Endurance Training in Older Men and Women.1. Cardiovascular-Responses to Exercise. *J Appl Physiol*. 1984;57:1024-1029.
24. Lexell J, Taylor CC, Sjostrom M. What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15-to 83-year-old men. *J Neurol Sci*. 1988;84:275-294
25. Deschenes MR. Effects of aging on muscle fibre type and size. *Sports Med*. 2004;34:809-824.
26. Evans WJ, Campbell WW. Sarcopenia and age-related changes in body composition and functional capacity. *The Journal of nutrition*. 1993;123:465-468.
27. Secher NH, Ruberg-Larsen N, Binkhorst RA, Bonde-Petersen F. Maximal oxygen uptake during arm cranking and combined arm plus leg exercise. *J Appl Physiol*. 1974;36:515-518.
28. Frontera WR, Meredith CN, O'Reilly KP, Evans WJ. Strength training and determinants of VO₂max in older men. *J Appl Physiol*. 1990;68:329-333.
29. McElvaney GN, Blackie SP, Morrison NJ, Fairbairn MS, Wilcox PG, Pardy RL. Cardiac output at rest and in exercise in elderly subjects. *Med Sci Sport Exerc*. 1989;21:293-298.
30. Rivera AM, Pels AE 3rd, Sady SP, Sady MA, Cullinane EM, Thompson PD. Physiological factors associated with the lower maximal oxygen consumption of master runners. *J Appl Physiol*. 1989;66:949-954.
31. Ogawa T, Spina RJ, Martin WH 3rd, Kohrt WM, Schechtman KB, Holloszy JO *et al*. Effects of aging, sex, and physical training on cardiovascular responses to exercise. *Circulation*. 1992;86:494-503.
32. Pullen PR, Nagamia SH, Mehta PK, Thompson WR, Benardon D, Hammoud R, *et al*. Effects of yoga on inflammation and exercise capacity in patients with chronic heart failure. *J Cardiac Fail*. 2008;14(5):407-413.
33. Pullen PP, Thompson WR, Benardon D, Brandon J,

- Mehta PK, Rifai L, *et al.* Benefits of yoga for African American Heart Failure Patients. *Med Sci Sports Exerc.* 2010;42(4):651-657.
34. Haykowsky MJ, Liang Y, Pechter D, Jones LW, McAlister FA, Clark AM. A meta-analysis of the effect of exercise training on left ventricular remodeling in heart failure patients: the benefit depends on the type of training performed. *J Am Coll Cardiol.* 2007;49(24):2329-2336.
35. Haykowsky MJ, Timmons MP, Kruger C, McNeely M, Taylor DA, Clark AM. Meta-analysis of aerobic interval training on exercise capacity and systolic function in patients with heart failure and reduced ejection fractions. *Am J Cardiol.* 2013;111(10):1466-1469.
36. Paterson DH, Cunningham DA, Koval JJ, St Croix CM. Aerobic fitness in a population of independently living men and women aged 55-86 years. *Med Sci Sports Exerc.* 1999;31(12):1813-1820.
37. Frankenstein L, Nelles M, Hallerbach M, Dukic D, Fluegel A, Schellberg D, *et al.* Prognostic impact of peakVO₂-changes in stable CHF on chronic beta-blocker treatment. *Int J Cardiol.* 2007;122(2):125-130.
38. Davies EJ, Moxham T, Rees K, Singh S, Coats AJS, Ebrahim S. Exercise training for systolic heart failure: Cochrane systematic review and meta-analysis. *Eur J Heart Fail.* 2010;12(7):706-715.