ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 09 | September - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CARDIOVASCULAR RESPONSES TO MODERATE-INTENSITY AEROBIC EXERCISE IN OBESE VERSUS NORMAL-WEIGHT YOUNG ADULT MALES: A CROSS-SECTIONAL STUDY

Physiology

Dr Tripathi Ashwin N. *
MBBS, MS, DNB. Senior Medical Officer, Dr SPM Civil Hospital, Lucknow
*Corresponding Author

ABSTRACT

Background: Obesity is an established risk factor for cardiovascular disease, and altered autonomic reactivity to exercise may be an early marker of this risk. Data on cardiovascular responses to exercise among obese Indian young adults remain limited. **Objectives:** To evaluate and compare heart rate, blood pressure and oxygen saturation responses to moderate-intensity aerobic exercise between obese and normal-weight young adult males. **Methods:** This cross-sectional study enrolled 45 healthy male college students (18–22 years) at a tertiary care teaching hospital: 20 obese cases (BMI >30 kg/m²) and 25 normal-weight controls (BMI <30 kg/m²). Anthropometric parameters were recorded, and each participant performed a standardized six-minute bicycle ergometer protocol at 25–50% of predicted VO2 max. Heart rate, blood pressure, electrocardiogram and peripheral oxygen saturation were recorded at baseline, immediately post-exercise, and at 2, 5 and 20 minutes of recovery. Between-group comparisons were made using the unpaired t-test, with $p<0.05$ considered significant. **Results:** Obese participants had significantly higher basal heart rate, systolic, diastolic and mean blood pressure than controls ($p<0.05$). Both groups showed a comparable rise in heart rate and blood pressure immediately after exercise, followed by a gradual decline over the recovery period; however, obese participants maintained significantly higher values than controls at every recovery time point ($p<0.05$). No significant inter-group difference was observed in oxygen saturation or in ECG findings at any stage. **Conclusion:** Obese young adult males demonstrate an exaggerated cardiovascular response to moderate exercise, with a similar but blunted recovery pattern compared with normal-weight peers, consistent with heightened sympathetic reactivity. These findings support early screening for cardiovascular risk in obese adolescents and young adults.

KEYWORDS

Obesity; Adolescents; Exercise Test; Blood Pressure; Heart Rate; Cardiovascular Fitness

INTRODUCTION

Obesity is an independent risk factor for several lifestyle-related disorders, particularly cardiovascular and endocrine disease, and its rising prevalence among children and young adults has become a major global public health concern.[1,2] While the proximate cause of obesity is an imbalance between energy intake and expenditure, its development is influenced by a complex interplay of genetic, environmental, neuroendocrine, metabolic and psychosocial factors.[3-5] Body mass index (BMI) remains the most widely used index for classifying nutritional status, with values above 30 kg/m² generally defining obesity.[6]

The transition from adolescence to young adulthood is associated with the steepest rise in obesity prevalence, driven by physical inactivity, poor dietary habits and increasing academic or occupational stress.[7] Obesity in this age group predisposes to insulin resistance, dyslipidaemia, elevated blood pressure and abdominal adiposity, laying the foundation for the metabolic syndrome later in life.[8,9] Despite this, few obese young adults engage in regular physical activity, commonly citing lack of time.[7]

Globally, the burden of overweight and obesity has continued to rise sharply over the past two decades, prompting some authors to describe it as the leading edge of a broader cluster of non-communicable diseases with substantial socioeconomic consequences, particularly in resource-limited settings.[11] In the Indian context, published estimates of overweight prevalence among adolescents range widely, from approximately 10% to 30%, reflecting differences in study populations, diagnostic criteria and geographic setting, but consistently point to an upward trend that parallels patterns already well described in Western populations.[17,18] Beyond its metabolic and cardiovascular consequences, adolescent and young-adult obesity also carries a substantial psychosocial burden, including social isolation and depressive symptoms, which may in turn reinforce sedentary behaviour and further weight gain.[13,14]

Exercise stress testing offers a simple, reproducible means of evaluating cardiovascular reactivity and autonomic function, but such studies remain scarce among Indian obese youth.[10] Previous work has linked obesity with reduced vagal tone and heightened sympathetic drive, manifesting as elevated resting heart rate and blood pressure as well as an exaggerated pressor response to exertion.[21,22] However, comparative data on the pattern of cardiovascular recovery after moderate exercise in obese versus normal-weight Indian young adults are limited. This study was therefore undertaken to characterize the heart rate, blood pressure and oxygen saturation responses to a standardized bout of moderate aerobic exercise in obese adolescents and young adults, in comparison with normal-weight controls.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional comparative study was conducted in the Department of Physiology at a tertiary care teaching hospital in New Delhi, India, following approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment.

Study Participants

Forty-five healthy male college students aged 18–22 years were recruited and screened using a structured questionnaire covering personal and medical history. Participants were allocated to two groups on the basis of BMI: Group I comprised 20 obese cases (BMI >30 kg/m²), and Group II comprised 25 normal-weight controls (BMI <30 kg/m²).

Individuals with obstructive airway disease, asthma, musculoskeletal abnormality, cardiac disease, anaemia, a history of smoking or substance use, or a diagnosed organic or psychiatric illness were excluded from participation.

Anthropometric Assessment

Height, weight and waist circumference were recorded using standard techniques. BMI was calculated as weight (kg) divided by height squared (m²), and body surface area (BSA) was derived using the DuBois nomogram.

Exercise Protocol

Following a brief clinical examination to exclude acute abnormalities, participants were seated comfortably on a bicycle ergometer (Proline Fitness Systems). Each test began with a two-minute warm-up at a light workload, after which the workload was progressively increased to achieve 25–50% of the individual's predicted maximal oxygen uptake (VO2 max), corresponding to moderate-intensity exercise. The exercise phase was sustained for six minutes, followed by a supervised cool-down.

Cardiovascular Monitoring

Heart rate, blood pressure, continuous electrocardiogram, and peripheral oxygen saturation (SpO2, measured by pulse oximetry) were recorded using the ML870B80 Exercise Physiology System (AD Instruments) at baseline (basal), immediately after cessation of exercise, and at 2, 5 and 20 minutes of recovery.

Statistical Analysis

Data were analysed using SPSS version 17.0. Continuous variables are expressed as mean ± standard deviation. Between-group comparisons at each time point were performed using the unpaired t-test. A two-

tailed p value <0.05 was considered statistically significant.

RESULTS

Anthropometric Characteristics

Obese participants had significantly higher body weight, waist circumference and BMI than controls (p<0.05). Height and BSA did not differ significantly between the groups (Table 1).

Group	Height (cm)	Weight (kg)	Waist circumference (cm)	BMI (kg/m²)	BSA (m²)
Control (n=25)	170.16 ± 7.28	63.48 ± 7.55	96.48 ± 7.18	21.75 ± 1.58	1.73 ± 0.13
Obese (n=20)	167.45 ± 7.10	98.85 ± 11.38*	125.7 ± 7.63*	35.22 ± 2.74*	2.142 ± 0.16

Heart Rate Response

Basal heart rate was significantly higher in obese participants than in controls (p<0.05). Both groups showed a comparable rise in heart rate immediately after exercise (p<0.05 for the increment within each group), followed by a progressive decline toward baseline at 2, 5 and 20 minutes of recovery. At every recovery time point, heart rate remained significantly higher in the obese group compared with controls (p<0.05) (Table 2).

Time point	Basal	Immediate	2 min	5 min	20 min
Control	80 ± 5	122 ± 6	93 ± 7	88 ± 6	84 ± 5
Obese	93 ± 8*	127 ± 7*	104 ± 9*	95 ± 7*	92 ± 7*

Blood Pressure Response

Obese participants demonstrated significantly higher basal systolic, diastolic and mean blood pressure than controls (p<0.05). Systolic blood pressure rose significantly in both groups immediately after exercise and declined progressively thereafter, remaining significantly higher in obese participants at all recovery points (p<0.05) (Table 3). Diastolic blood pressure fell modestly immediately after exercise and at 2 minutes in both groups, without significant inter-group difference at these points (p>0.05); however, a rise in diastolic pressure at 5 and 20 minutes was significantly greater in obese participants (p<0.05 at 20 minutes). Mean arterial pressure followed a pattern similar to systolic pressure, with obese participants showing consistently higher values at every time point (p<0.05).

Time point	Basal	Immediate	2 min	5 min	20 min
Control	112 ± 15	149 ± 10	135 ± 8	124 ± 8	113 ± 9
Obese	134 ± 12*	172 ± 11*	155 ± 12*	141 ± 11*	127 ± 10*

Oxygen Saturation and ECG

No significant difference in SpO2 was observed between groups at baseline. Both groups showed a mild, statistically non-significant fall in oxygen saturation immediately after exercise, with recovery to near-baseline values by 2, 5 and 20 minutes and no significant inter-group difference at any stage. No ST-segment changes were observed on continuous ECG monitoring in either group at any point during or after exercise.

DISCUSSION

This study demonstrates that obese young adult males exhibit significantly higher basal heart rate and blood pressure than their normal-weight counterparts, and that this difference persists throughout the immediate post-exercise period and into recovery, despite a broadly similar pattern and time-course of recovery in both groups. These findings are consistent with previous reports linking obesity to reduced vagal tone and heightened sympathetic cardiovascular drive.[21,22]

The exaggerated rise in heart rate and blood pressure observed in obese participants during exercise may be explained by several interrelated mechanisms. Increased body mass imposes a greater cardiovascular workload for any given level of external exertion, requiring higher cardiac output and oxygen delivery to peripheral tissues.[24] Expanded blood volume and adipose tissue vascularity in obesity may further contribute to this haemodynamic burden. In addition, obesity is associated with heightened central sympathetic outflow, which becomes particularly prominent as metabolic and mechanical afferent feedback from exercising muscle intensifies during exertion.[23] The greater waist circumference observed in the obese group in this study is consistent with central adiposity, which has been linked to the autonomic and metabolic derangements that often precede the development of sustained arterial hypertension.[5]

The differential behaviour of systolic and diastolic blood pressure in this study merits comment. Systolic pressure, which is known to respond briskly to changes in cardiac output and exertion, showed a marked and consistent divergence between groups throughout exercise and recovery.[25] Diastolic pressure, being more dependent on peripheral vascular resistance, changed more gradually and did not differ significantly between groups in the immediate post-exercise phase, becoming significant only later in recovery. This pattern is in keeping with previous observations in obese populations.[25]

Notably, oxygen saturation and ECG findings did not differ between groups at any stage, suggesting that respiratory compensatory mechanisms and myocardial electrical stability were preserved in obese participants despite their heightened haemodynamic reactivity, at least over the short duration and moderate intensity of exercise studied.[26] This indicates that the cardiovascular abnormality identified in this study is primarily autonomic and haemodynamic rather than reflecting overt myocardial ischaemia or respiratory compromise at this stage of obesity.

These findings have practical relevance for preventive medicine. Medical students, the population studied here, might be expected to have greater awareness of the health hazards of obesity than the general population; the persistence of an adverse cardiovascular profile in this group is therefore a matter of particular concern and underscores the pervasiveness of the problem. Given the well-documented genetic predisposition of the Indian population to metabolic syndrome, early identification of exaggerated cardiovascular reactivity to exercise in obese adolescents and young adults may help target preventive counselling and lifestyle intervention before the establishment of overt cardiovascular disease.[2]

Exercise stress testing, of the type employed in this study, offers a practical and low-cost screening tool that could plausibly be incorporated into routine health assessments in schools, colleges and primary care settings, particularly in populations where access to more sophisticated autonomic function testing is limited. Identifying an exaggerated pressor response to submaximal exercise at a young age may allow clinicians to flag individuals at elevated future cardiovascular risk well before the onset of sustained hypertension or overt disease, creating a window of opportunity for lifestyle counselling, structured physical activity programmes and dietary modification. Given that the recovery pattern observed in obese participants, though numerically higher throughout, broadly paralleled that of controls, it is plausible that timely intervention through weight reduction and regular aerobic training could normalize this exaggerated reactivity, as has been suggested by studies demonstrating restoration of vasodilatory and blood pressure responses following structured diet and exercise programmes in obese children.[14] This underscores the importance of not only identifying at-risk individuals but also linking screening findings to actionable, sustainable interventions.

Limitations

This study was limited by its relatively small sample size, single-sex population, cross-sectional design, and reliance on a single moderate-intensity exercise protocol. Autonomic function was not assessed directly through heart rate variability or baroreflex sensitivity testing, and long-term cardiovascular outcomes could not be evaluated. Larger, longitudinal studies incorporating both sexes and direct autonomic measures are needed to confirm and extend these findings.

CONCLUSION

Obese young adult males show significantly higher basal heart rate and blood pressure, and an exaggerated cardiovascular response to moderate aerobic exercise, compared with normal-weight peers, although their recovery pattern remains broadly similar. These findings reinforce the need for early cardiovascular risk screening and lifestyle intervention among obese adolescents and young adults, particularly in populations with a known predisposition to metabolic syndrome.

Conflict of Interest

None declared.

Source of Funding

This work was supported by the Indian Council of Medical Research (ICMR) under the Short Term Studentship (STS) Programme, 2012 (Reference ID: 201200242).

REFERENCES

1. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. Int J Obes Relat Metab Disord. 1999;23(Suppl 2):S2-S11.

2. Raj M, Kumar RK. Obesity in children & adolescents. Indian J Med Res. 2010;132(5):598-607.
3. Swinburn B, Egger G. Preventive strategies against weight gain and obesity. Obes Rev. 2002;3:289-301.
4. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. Science. 1998;280:1371-4.
5. Grundy SM. Multifactorial causation of obesity: implications for prevention. Am J Clin Nutr. 1998;67:563S-72S.
6. Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: a validation study. J Pediatr. 1998;132(2):204-10.
7. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. Pediatrics. 1998;101(Suppl 3):497-504.
8. Daniels SR, Arnett DK, Eckel RH, Gidding SS, Hayman LL, Kumanyika S, et al. Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. Circulation. 2005;111:1999-2012.
9. Eckel RH, Krauss RM. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. Circulation. 1998;97:2099-100.
10. Sowmya R, Maruthy KN, Gupta R. Cardiovascular autonomic responses to whole body isotonic exercise in normotensive healthy young adult males with parental history of hypertension. Indian J Physiol Pharmacol. 2010;54(1):37-44.
21. Salvadori P, et al. Oxygen uptake and cardiac performance in obese and normal subjects during exercise. Respiration. 1999;66:25-33.
22. Ibhazehiebo K, Dimkpa U, Iyawe VI. Hypertension, and blood pressure response to graded exercise in young obese and non-athletic Nigerian university students. Niger J Physiol Sci. 2007;22(1-2):37-42.
23. Carletti L, Rodrigues AN, Perez AJ, Vassallo DV. Blood pressure response to physical exertion in adolescents: influence of overweight and obesity. Arq Bras Cardiol. 2008;91(1):24-8.
24. Tulio S, Eglé S, Greily B. Blood pressure response to exercise of obese and lean hypertensive and normotensive male adolescents. J Hum Hypertens. 1995;9:953-8.
25. Nishina M, Kikuchi T, Yamazaki H, Kameda K, Hiura M, Uchiyama M. Relationship among systolic blood pressure, serum insulin and leptin, and visceral fat accumulation in obese children. Hypertens Res. 2003;26(4):281-8.
26. Tschakovsky ME, Hughson RL. Interaction of factors determining oxygen uptake at the onset of exercise. J Appl Physiol. 1999;86:1101-13.