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Original Article

# Peak $VO_2$ During Bicycle Ergometry Is Reduced Even in Early Diabetics

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## ABSTRACT

**Background:** Type 2 diabetes mellitus (T2DM) is associated with early cardiovascular and metabolic alterations that may precede overt complications. Peak oxygen uptake (Peak $VO_2$) is a key indicator of aerobic capacity and cardiovascular fitness.
**Objective:** To assess Peak $VO_2$ during bicycle ergometry in early diagnosed T2DM patients and compare it with healthy controls. And also to assess the correlation between peak VO2 and duration of diabetes
**Material and Methodology:** The Cardiopulonary Exercise Testing was performed on a USA based GASSYS3 BIOPAC gas analyzer machine on 64 diabetic and 64 healthy subjects. Oxygen consumption ($VO_2$), $CO_2$ production ($VCO_2$), Respiratory exchange ratio (RER) and Minute ventilation (VE) were recorded both at rest and after performing symptom limited CPET on the bicycle ergometer.
**Result:** Peak $VO_2$ was found to be significantly ($p<0.01$) reduced while RER was found to be significantly ($p=0.01$) increased in early diabetics. However, $VO_2$ at rest, was not significantly different among the groups ($p>0.05$). Peak $VO_2$ also showed a significantly negative correlation with duration of diabetes. ($p<0.01$; $r = -0.34$)
**Conclusion:** Peak $VO_2$ is reduced even in the early stages of T2DM, indicating early impairment of aerobic capacity. Bicycle ergometry is a useful non-invasive tool for early functional assessment in diabetic patients.

***Keywords****: Peak $VO_2$; Bicycle ergometry; Type 2 diabetes mellitus; Cardiopulmonary Exercise test(CPET).*

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease characterized by hyperglycemia resulting from insulin resistance and relative insulin deficiency.(1,2) Globally, T2DM prevalence is rising, affecting younger adults and increasing the burden of cardiovascular disease, particularly in developing countries like India(3). While the long-term complications of diabetes are well recognized, growing evidence suggests that cardiovascular and metabolic dysfunction begins early in the course of the disease(4,5).

Peak oxygen uptake (Peak $VO_2$) reflects the integrated response of the cardiovascular, pulmonary, and muscular systems during exercise(6,7) and is considered the gold standard measure of aerobic fitness(8). Reduced Peak $VO_2$ is associated with increased cardiovascular risk and mortality.(9)

Exercise testing using bicycle ergometry is a safe and reproducible method to evaluate exercise capacity(10). Previous studies predominantly focused on long-standing diabetes or patients with complications. There is limited data evaluating $VO_2$ peak in early-stage diabetes (<5 years duration). Identifying reduced functional capacity early could guide preventive strategies, including tailored aerobic exercise programs and lifestyle interventions.

This study aimed to assess $VO_2$ peak during bicycle ergometry in early diabetics and compare it with healthy controls to determine early impairment in aerobic capacity. And also aimed to assess the correlation between peak VO2 and duration of diabetes.

## MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Physiology, G. R. Medical College, Gwalior. A total of 128 subjects were enrolled, including 64 patients with type 2 diabetes mellitus of less than five years' duration and 64 age- and sex-matched healthy controls. Diabetic subjects were recruited from the Medicine Outpatient Department of Jaya Arogya Hospital, Gwalior.

The study group comprised patients aged 30–60 years with type 2 diabetes mellitus and no history of cardiovascular disease. The control group included healthy individuals without any chronic illness. Exclusion criteria were refusal to participate, body mass index (BMI) >30 kg/m², resting blood pressure >140/90 mmHg, abnormal electrocardiographic findings, history of cardiovascular, respiratory, hepatic, or renal disease, and presence of endocrine disorders other than diabetes mellitus.

A detailed medical history was obtained from all participants, followed by general physical examination and anthropometric measurements. Written informed consent was obtained prior to participation

Cardiopulmonary exercise testing (CPET) was performed using a GASSYS3 BIOPAC gas analyzer. After application of a tight-fitting face mask, resting oxygen consumption ($VO_2$), carbon dioxide production ($VCO_2$), respiratory exchange ratio (RER), and minute ventilation (VE) were recorded. Participants performed a 2-minute warm-up on a cycle ergometer, followed by rest until heart rate returned to baseline.

Submaximal, symptom-limited CPET was conducted using a constant work-rate protocol as described by **Mezzani et al**(11)**.** Exercise was continued until approximately 75% of age-predicted maximum heart rate was achieved or till volitional fatigue.

Heart rate and blood pressure were recorded at rest and immediately after exercise. Peak $VO_2$, peak $VCO_2$, peak RER, and VE were documented.

## OBSERVATION TABLES

**Table 1: Anthropometric Parameters of Diabetic and Healthy Group**

| S. No. | Parameters | Diabetics Group | HealthyGroup | p-value |
|---|---|---|---|---|
| 1 | Age (years) | 44.61±7.243 | 43.70±7.206 | 0.4793 |
| 2 | Height (cm) | 165± 6.08 | 164±4.34 | 0.3551 |
| 3 | Weight (Kg) | 67.92±6.84 | 66.17±5.851 | 0.1227 |
| 4 | BMI ($kg/m^2$) | 24.91±1.707 | 24.54±1.670 | 0.2162 |

*=p<0.05 = Statistically Significant**=p<0.01 = Statistically Highly Significant
***=p<0.001 = Statistically Very Highly Significant

**Table 2: Comparison of investigations between Diabetic Group and Healthy Group**

| S.No. | Parameters | Diabetics Group | HealthyGroup | p-value |
|---|---|---|---|---|
| 1. | Triglyceride (mg/dl) | 137±23.1 | 130±52.0 | 0.380 |
| 2. | Cholesterol (mg/dl) | 146±21.0 | 142±29.0 | 0.4818 |
| 3. | FBS (mg/dl) | 118.4 ± 16.75 | 83.89±9.71 | **<0.001*** ** |
| 4. | RBS (mg/dl) | 154±20.8 | 119±19.0 | **<0.001*** ** |

**Table 3: Comparison of haemodynamic parameters of Diabetic Group & Healthy Group at rest**

| S.No. | Parameters | Diabetics Group | HealthyGroup | p-value |
|---|---|---|---|---|
| 1 | SBP (mm Hg) | 126.9±6.872 | 125±4.605 | 0.0671 |
| 2 | DBP (mm Hg) | 81.2 ±3.98 | 80.9 ±4.66 | 0.7141 |
| 3 | Mean BP (mm Hg) | 96.4 ± 3.57 | 95.6 ±3.72 | 0.2040 |
| 4 | Pulse rate/min | 73.38 ±5.86 | 74.34 ±8.16 | 0.4423 |

**Table 4: Comparison of haemodynamic parameters of Diabetic and Healthy group at end of exercise**

| S.No. | Parameters | Diabetics Group | Healthy Group | p-value |
|---|---|---|---|---|
| 1 | SBP mm Hg | 153 ±7.87 | 151 ±6.29 | 0.076 |
| 2 | DBP mm Hg | 84.00 ± 4.00 | 83.44 ± 4.238 | 0.4415 |
| 3 | Mean BP mm Hg | 107.3±3.60 | 106.4±3.57 | 0.067 |
| 4 | Pulse rate/min | 116 ± 5.25 | 118±8.54 | 0.1219 |

**5: Comparison of Cardiopulmonary Exercise Test Parameters between Diabetics and Healthy groups at rest**

| S.No. | Parameters | Diabetics Group | Healthy Group | p-value |
|---|---|---|---|---|
| 1. | VO2(ml/kg/min) | 3.66 ±0.610 | 3.59 ±0.599 | 0.56 |
| 2. | VCO2(l/min) | 0.2831±0.058 | 0.2736±0.051 | 0.2864 |
| 3. | RER | 0.874±0.081 | 0.876±0.083 | 0.91 |
| 4. | VE (l/min) | 6.40±2.33 | 5.85±1.68 | 0.1239 |

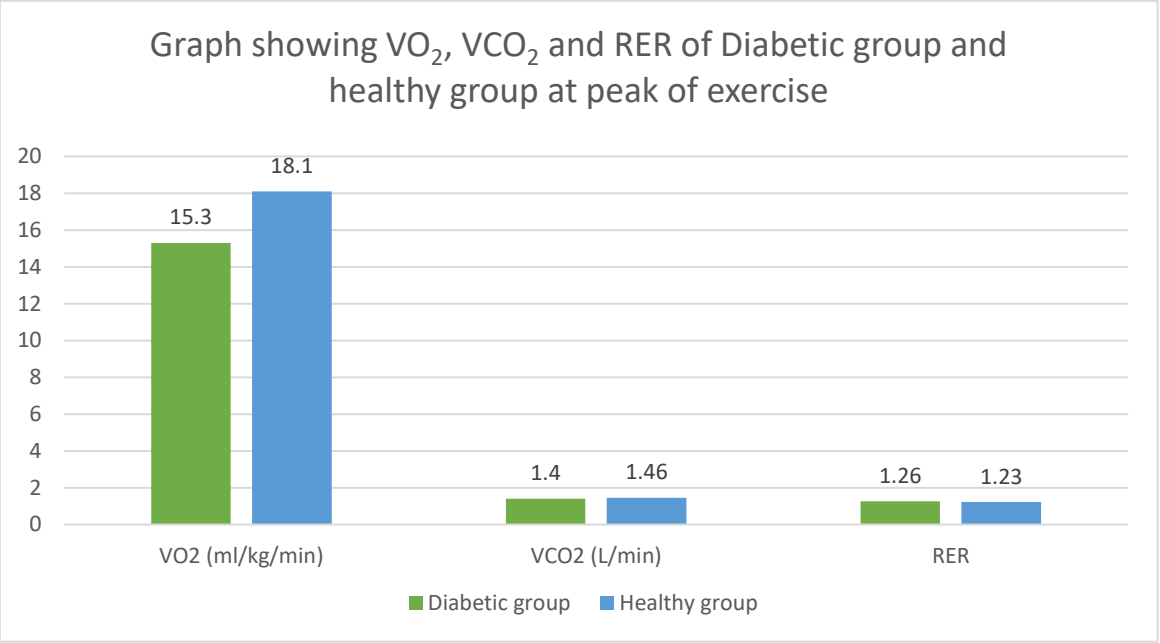
**Table 6: Comparison of Cardiopulmonary Exercise Test Parameters between Diabetics and Healthy groups at end of exercise**

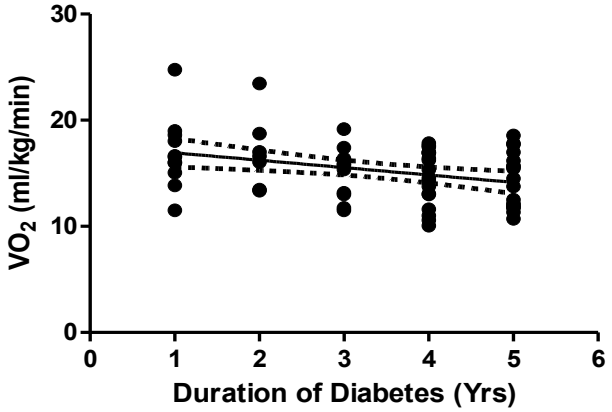
| S.No. | Parameters | Diabetics Group | Healthy Group | p-value |
|---|---|---|---|---|
| 1. | $VO_2$(ml/kg/min) | 15.3± 2.92 | 18.1±2.52 | **<0.0001** |
| 2. | $VCO_2$(l/min) | 1.405±0.2436 | 1.461±0.1897 | 0.1512 |
| 3. | RER | 1.262±0.0633 | 1.230±0.0787 | **0.0126*** |
| 4. | VE (L/min) | 37.86 ± 8.388 | 37.23 ± 6.544 | 0.6379 |

**Table7: Correlation of Cardiopulmonary exercise test with Duration of Diabetes Mellitus**

| S.No. | Parameter | p-value | Pearson r |
|---|---|---|---|
| 01 | VO2 peak (ml/kg/min) | **0.0058*** | -0.3414 |
| 02 | VCO2 (L/min) | 0.2497 | -0.1460 |
| 03 | RER | 0.8077 | -0.0310 |
| 04 | VE (L/min) | 0.9349 | 0.0104 |

*- signifies significant difference having p-value<0.05.



**Statistical Analysis**
Data were expressed as mean ± standard deviation. Student's unpaired t-test was used for comparison between groups. A p-value <0.05 was considered statistically significant.

## RESULTS
Anthropometric measurements and hemodynamic parameters at rest were comparable between the two groups (p>0.05). Even early(less than 5 years) diabetic subjects demonstrated significantly lower Peak $VO_2$ values, along with reduced exercise duration and early onset of fatigue.

## DISCUSSION
This study evaluated cardiovascular fitness in normotensive patients with early type 2 diabetes mellitus using cardiopulmonary exercise testing (CPET) and compared the findings with healthy controls. Anthropometric parameters were comparable between the two groups, thereby minimizing the potential confounding effects of body composition on exercise capacity. As expected, fasting and random blood glucose levels were significantly higher in diabetic subjects, reflecting suboptimal glycemic control, while lipid profiles did not differ significantly between groups. Similar observations have been reported by **DeFronzo and Ferrannini et al**(12), who highlighted that metabolic abnormalities often precede overt cardiovascular manifestations in early diabetes.

Resting systolic and diastolic blood pressure, mean arterial pressure, and heart rate were comparable between diabetic and control subjects, confirming preserved resting hemodynamic status and normotension. Post-exercise increases in hemodynamic parameters were observed in both groups without significant intergroup differences. These findings are consistent with earlier studies by **Regensteiner et al.** (13)and **Fang et al.**(14), who demonstrated preserved resting cardiovascular function in patients with early type 2 diabetes prior to the onset of overt cardiovascular disease.

Resting oxygen consumption ($VO_2$) did not differ significantly between groups, suggesting similar basal metabolic requirements. However, peak oxygen consumption ($VO_2$ peak) was significantly lower in diabetic subjects, indicating impaired cardiorespiratory fitness. $VO_2$ peak is a well-established indicator of aerobic capacity and cardiovascular health, and its reduction reflects early limitations in oxygen delivery and utilization. Similar reductions in $VO_2$ peak among patients with type 2 diabetes have been consistently reported by **Regensteiner et al.** (1) and **Baldi and Snowling et al**(15).

Several mechanisms may underlie the observed reduction in $VO_2$ peak. Chronic hyperglycemia contributes to endothelial dysfunction, impaired nitric oxide–mediated vasodilation, and microvascular remodeling, resulting in reduced skeletal muscle perfusion during exercise (**Hamburg et al.,**(16)**; Ceriello et al**(17). In addition, mitochondrial dysfunction, decreased oxidative enzyme activity, and reduced skeletal muscle oxidative capacity have been demonstrated in patients with type 2 diabetes **Kelley et al.,**(18)**; Phielix and Mensink et al,** (19). The accumulation of advanced glycation end products further compromises vascular compliance and oxygen diffusion **Brownlee et al,** (20).

The present findings align with previous investigations suggesting that diminished aerobic capacity may represent an early marker of subclinical cardiovascular dysfunction in type 2 diabetes. Studies by **Poole et al.**(21) and **Ratchford et al.** (22)have shown that impaired cardiac output, reduced peripheral oxygen extraction, and a diminished arteriovenous oxygen difference contribute to reduced exercise tolerance in diabetic individuals.

Peak respiratory exchange ratio (RER) was significantly higher in diabetic subjects, indicating an increased reliance on anaerobic metabolism during maximal exercise. This finding suggests reduced metabolic flexibility and an early shift toward glycolytic pathways. Impaired mitochondrial oxidative phosphorylation, altered substrate utilization, and suboptimal blood flow distribution to exercising muscles may account for this response **Mogensen et al.,**(23)**; Schrauwen and Hesselink et al,** (24).

A significant negative correlation between diabetes duration and $VO_2$ peak was observed, indicating a progressive decline in cardiovascular fitness with increasing disease duration. Prolonged exposure to hyperglycemia promotes cumulative oxidative stress, endothelial injury, inflammation, and microvascular damage, which collectively impair cardiovascular and metabolic function (**Laakso et al,** (25)**; Stratton et al.,** (26)). These findings support earlier evidence linking longer duration of diabetes with increased cardiovascular risk and reduced functional capacity.

## CONCLUSION
In conclusion, normotensive patients with early type 2 diabetes demonstrate significantly reduced aerobic capacity despite preserved resting hemodynamics and comparable anthropometric characteristics. CPET emerges as a sensitive tool for detecting early impairments in cardiovascular fitness in this population. Early identification of reduced $VO_2$ peak may facilitate timely lifestyle and therapeutic interventions aimed at improving glycemic control and cardiorespiratory fitness, thereby reducing future cardiovascular risk.

**REFERENCES**


1. Regensteiner JG. Type 2 diabetes mellitus and cardiovascular exercise performance. Rev Endocr Metab Disord. 2004 Aug;5(3):269–76.
2. Kanaley JA, Colberg SR, Corcoran MH, Malin SK, Rodriguez NR, Crespo CJ, et al. Exercise/Physical Activity in Individuals with Type 2 Diabetes: A Consensus Statement from the American College of Sports Medicine. Med Sci Sports Exerc. 2022 Feb 1;54(2):353–68.
3. Tabish SA. Is Diabetes Becoming the Biggest Epidemic of the Twenty-first Century? Int J Health Sci. 2007 July;1(2):V–VIII.
4. Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). Lancet Diabetes Endocrinol. 2023 July;11(7):474–89.
5. Alvin C. Powers, Kevin D. Niswender, Carmella Evans-Molina.Diabetes Mellitus: Diagnosis, Classification, and Pathophysiology | Harrison's Principles of Internal Medicine, 21e; chapter 403: 3094-3103 - Google Search [Internet]. [cited 2026 Jan 13].
6. Green S, Egaña M, Baldi JC, Lamberts R, Regensteiner JG. Cardiovascular Control during Exercise in Type 2 Diabetes Mellitus. J Diabetes Res. 2015;2015:654204.
7. Malhotra R, Bakken K, D'Elia E, Lewis GD. Cardiopulmonary Exercise Testing in Heart Failure. JACC Heart Fail. 2016 Aug;4(8):607–16.
8. Gonzales TI, Westgate K, Strain T, Hollidge S, Jeon J, Christensen DL, et al. Cardiorespiratory fitness assessment using risk-stratified exercise testing and dose-response relationships with disease outcomes. Sci Rep. 2021 July 28;11(1):15315.
9. Seyoum B, Estacio RO, Berhanu P, Schrier RW. Exercise capacity is a predictor of cardiovascular events in patients with type 2 diabetes mellitus. Diab Vasc Dis Res. 2006 Dec;3(3):197–201.
10. Eckstein ML, Farinha JB, McCarthy O, West DJ, Yardley JE, Bally L, et al. Differences in Physiological Responses to Cardiopulmonary Exercise Testing in Adults With and Without Type 1 Diabetes: A Pooled Analysis. Diabetes Care. 2021 Jan;44(1):240–7.
11. Mezzani A. Cardiopulmonary Exercise Testing: Basics of Methodology and Measurements. Ann Am Thorac Soc. 2017 July;14(Supplement_1):S3–11.
12. DeFronzo RA. Pathogenesis of type 2 diabetes mellitus. Med Clin North Am. 2004 July;88(4):787–835, ix.
13. Regensteiner JG, Sippel J, McFarling ET, Wolfel EE, Hiatt WR. Effects of non-insulin-dependent diabetes on oxygen consumption during treadmill exercise. Med Sci Sports Exerc. 1995 June;27(6):875–81.
14. Fang ZY, Prins JB, Marwick TH. Diabetic cardiomyopathy: evidence, mechanisms, and therapeutic implications. Endocr Rev. 2004 Aug;25(4):543–67.
15. Baldi JC, Snowling N. Resistance training improves glycaemic control in obese type 2 diabetic men. Int J Sports Med. 2003 Aug;24(6):419–23.
16. Hamburg NM, McMackin CJ, Huang AL, Shenouda SM, Widlansky ME, Schulz E, et al. Physical inactivity rapidly induces insulin resistance and microvascular dysfunction in healthy volunteers. Arterioscler Thromb Vasc Biol. 2007 Dec;27(12):2650–6.
17. Ceriello A. Oxidative Stress and Diabetes-Associated Complications. Endocr Pract. 2006 Jan 1;12:60–2.
18. Kelley DE, He J, Menshikova EV, Ritov VB. Dysfunction of mitochondria in human skeletal muscle in type 2 diabetes. Diabetes. 2002 Oct;51(10):2944–50.
19. Phielix E, Mensink M. Type 2 diabetes mellitus and skeletal muscle metabolic function. Physiol Behav. 2008 May 23;94(2):252–8.
20. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001 Dec 13;414(6865):813–20.
21. Poole DC, Hirai DM, Copp SW, Musch TI. Muscle oxygen transport and utilization in heart failure: implications for exercise (in)tolerance. Am J Physiol Heart Circ Physiol. 2012 Mar 1;302(5):H1050-1063.
22. 12. Ratchford SM, Kargl J, Healey CM, et al. Vascular dysfunction and reduced aerobic capacity in individuals with type 2 diabetes. J Appl Physiol. 2016;121(2):527–35. - Google Search [Internet]. [cited 2026 Jan 15].
23. Mogensen M, Sahlin K, Fernström M, Glintborg D, Vind BF, Beck-Nielsen H, et al. Mitochondrial respiration is decreased in skeletal muscle of patients with type 2 diabetes. Diabetes. 2007 June;56(6):1592–9.
24. Schrauwen P, Hesselink MKC. Oxidative capacity, lipotoxicity, and mitochondrial damage in type 2 diabetes. Diabetes. 2004 June;53(6):1412–7.
25. Laakso M. Cardiovascular disease in type 2 diabetes: challenge for treatment and prevention. J Intern Med. 2001 Mar;249(3):225–35.
26. Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. BMJ. 2000 Aug 12;321(7258):405–12.