



# INFLUENCE OF AEROBIC TREADMILL EXERCISE ON BLOOD GLUCOSE HOMEOSTASIS AND LIPID PROFILE IN OBESE TYPE-II DIABETICS MELLITUS

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#### Abstract

The influence of aerobic treadmill exercise on blood glucose homeostasis and lipid profile in obese Type 2 diabetics over a period of six weeks was investigated. The experimental group consisted of 10 males with mean age ( $X=52\pm4$ ) and a control group of 10 males with mean age ( $X=53\pm3$ ), who were clinically and biochemical confirmed as obese diabetics. The results of both groups were compared after six weeks. The results were analyzed using an independent t- test. The experimental group showed a significant decrease in mean fasting blood sugar (FBS) of 39.4±8.315 and Post-prandial blood sugar (PPBS) of 44.4  $\pm$  8.617 as compared to the control group with mean FBS of 27.4 SD±9.720 and PPBS of 32.2  $SD \pm 6.972$  with a significant inter group difference (P<0.005). The Lipid Profile also showed a significant difference between two groups. The mean decrease in cholesterol for the experimental group was 44.23±7.34; the control group was 14.34 ±5.782. The mean decrease in Triglycerides for the experimental group was 27.12  $\pm$ 7.34 as compared to the control group with 16.45 ±4.34. Clinically, Body Mass Index (BMI) was found to be significantly decreased in the experimental group with 25.67 ±1.150 as compared to the control group with 29.56 ±2.007. Treadmill exercise was found to be a viable tool in addition to drug and diet in glycemic and Lipid profile control.





*Key words: Type 2 Diabetes Mellitus, obesity and aerobic treadmill exercise* 

## Introduction

Diabetes mellitus and obesity are metabolic disorders characterized by actual or relative insufficiency of insulin, an abnormal growth of adipose tissue with an enlargement of fat cell size (hypertrophy) or an increase in fat cell number (hyperplasic) or a combination of both. Both environmental and genetic factors are involved in the pathogenesis of diabetes and obesity. The diabetes mellitus and obesity are interrelated - one is the risk factor for the other and affects approximately 2%-3% of the Indian population. These include excess calorie intake, decreased physical activity, and metabolic as well as endocrine abnormalities<sup>1</sup>. These factors lead to an inability to utilize glucose, hence elevated blood glucose levels called hyperglycemia,<sup>2</sup> and an increase in cholesterol and triglycerides called hyperlipidemia<sup>3</sup>.

The diagnosis is confirmed by following blood parameters fasting blood sugar (FBS), post-prandial blood sugar (PPBS), oral glucose tolerance test (OGTT) and urine examination, Lipid profile examination and clinical examination (Body Mass Index, Waist-Hip Ratio and skin fold measurement).

The complications associated with diabetes and obesity proves to be a major public health problem. The distribution of adipose tissue in different anatomic depots has substantial implications or morbid mortality in obese patients. Intra abdominal and abdominal





subcutaneous fat are more substantive risk factors for diseases than subcutaneous fat in the buttocks and lower extremities. Based on the distribution of adipose tissue, obesity can be classified as android obesity and gynoid obesity<sup>4</sup>. In android obesity, there is storage of fat in the upper body-mainly in the abdomen giving the individual an "apple" shape and a waist hip ratio of more than one. It is seen predominantly in the male population. A waist hip ratio of more than one may be closely related to disease risk than Body Mass Index (BMI) alone. A waist hip ratio of more than one indicates an increased risk for cardiovascular disease and diabetes mellitus<sup>5-6</sup>, it is associated with a number of health risks including like an increased risk of hypertension, ischemic heart disease, cerebrovascular accident, diabetes mellitus, cancer, a decrease in lung function and capacities, psychological stress even with sudden death.

Treatment modalities of hyperglycemia and hyperlipidemia consist of a triad of drugs, diet and exercise. Each has a specific role in promoting glucose uptake and hence balancing blood glucose levels and lipid levels <sup>7</sup>. Regular exercise is a valuable supplement to diet. The brisk walking is the best form of exercise to any non-insulin dependent diabetes mellitus (NIDDM) patients with obesity. The natural walking involves large group of muscles of lower limb along with rhythmic upper body muscular work. The Treadmill aerobic exercise simulates all the natural walking features and has added advantage<sup>8</sup>

There is still controversy regarding the beneficial effect of exercise in NIDDM patients with obesity<sup>9-10</sup>.





There are studies, which support the view that aerobic exercises decrease blood glucose levels, plasma triglyceride concentrations and there are studies, which are of the view, that aerobic exercises alone are not effective in altering the lipid profile. Therefore, in order to study the efficacy of aerobic treadmill exercise on glucose homeostasis and lipid profile this investigation has been conducted.

## Method

The study was conducted in Kasturba Hospital, Manipal, India (a University Teaching Hospital) after clearance obtained from the Ethical committee.

*Experimental group*: Ten males between the ages of 45 and 60 years (X-52±4), who were clinically and biochemically confirmed cases of obese Type 2 diabetics were selected. Only those willing to participate in the experimental study were included.

*Control group*: It consisted of 10 males between the ages of 45 and 60 (X-53±3) years, who were clinically and biochemically confirmed cases of obese Type 2 diabetics and who were not willing to participating in exercise were selected for the control group.

A physician examined all participants. They were screened for cardiovascular problems such as ischemic heart disease and peripheral vascular disease, respiratory diseases such as chronic obstructive pulmonary diseases and musculoskeletal problems such as osteo-arthritis, cervical spondylosis and low backache, peri-arthritis and diabetic neuropathy before commencing the study. Both groups continued with standard diet and medication as







prescribed. The experimental group was given graded Treadmill exercise for period of six weeks.

Both groups were clinically assessed for weight, height, body mass index (BMI), waist-hip ratio and skin fold measurement. Muscular flexibility and strength in lower limbs were assessed and, in case of tightness or weakness, an appropriate physical therapy program was instituted before the study. At the beginning of the study, blood glucose levels were investigated for PPBS and FBS. A Lipid Profile for cholesterol and Triglycerides were ascertained on both groups.

A motor-driven treadmill was used for the study. It provided computerized programming parameters for distance traveled (Kilometers), calories used (Kilocalories) and speed (RPM). Treadmill calibration was done before starting treatment. A clear explanation involving treadmill protocol was given to each patient. Each experimental session lasted for 40 minutes including 5 minutes of warm-up, 30 minutes of aerobic treadmill walking and 5 minutes of cool-down. The vital parameters for HR, BP, and RR were recorded before, during and after treatment. Intensity of exercise was regulated by maintaining constant speed of 3.4 km/hr, fixed inclination of 4.2 angle and Borg's score of 13-14 for rate of perceived exertion. All 10 patients completed five sessions per week, for six weeks, The Clinical Examination and blood uneventfully. investigations were repeated for both the groups after a six-week period. Results were compared between both groups and statistically analyzed with an independent ttest.



## **Result Analysis**

All patients in the experimental group completed the planned six weeks of treadmill endurance exercise program without any complications. The BMI of the experimental group was 25-29; the control group was 27-31. Blood glucose levels were analyzed to determine the inter-groups decrease in FBS and PPBS. The mean decrease in FBS was  $39.4\pm8.315$  for the experimental group and  $27.4\pm9.720$  for the control group; the intergroup decrease in FBS was significant with p value <0.005. The decrease in PPBS was  $44.4\pm8.617$  for the experimental group and  $32.2\pm6.972$  for the controls; the inter-group decrease in PPBS was significant with p<0.005. (Table I & Figures 1 & 2).

The Lipid Profile was analyzed to determine the intergroup decrease in cholesterol and triglycerides. The mean decrease in cholesterol was  $44.23\pm27.34$  for the experimental group; the control group was  $14.34\pm5.782$  and was highly significant at p<0.0001. The mean decrease in triglycerides for the experimental group was 27.12  $\pm7.34$ ; the control group was 16.45 $\pm4.3$ ; it was significant at p<0.005. (Table II & Figures 3 & 4).

The mean body mass index (BMI) of the experimental group was  $25.67\pm1.150$ . The mean body mass index (BMI) of the control group was  $29.56\pm2.007$ . After exercise, there was a significant difference between the groups (p <0.0001). (Table III).





#### Discussion

The deficiency of insulin and the failure of glucose uptake lead to a backup of glucose in the blood resulting in hyperglycemia. The treatment of diabetes consists of education, exercise, diet, oral hypoglycemic drugs and subcutaneous insulin therapy<sup>11</sup>. The triad of drugs, diet and exercise has been the basis for treatment of diabetes Each done individually or in for the past 60 years<sup>12</sup>. combination has a place in treatment regimen. An exercise program in conjunction with diet and oral medication can create glycemic control, weight reduction, and reduction in cardiovascular risk factors as well as improvement in the mental well being of the patient <sup>13-14</sup>. Wahren and colleagues<sup>15</sup> reported that exercise in poorly controlled diabetes cases, leads to increased blood glucose and ketoacids. Hermansen and co-workers<sup>16</sup> reported that after six weeks of treadmill walking, insulin binding to monocyte receptor sites increased and allowed greater insulin mediated glucose uptake within promoted oxidation of glucose and its glycolytic products. There is increased glycogen utilization and storage during and after physical activity influencing glucose metabolism, so that there is enhanced glucose uptake associated with endurance exercise.

Despite significant progress in recent years, Lipid and lipoprotein abnormalities play a major role in the development and progression of coronary artery disease and diabetes mellitus, and coronary artery disease (CAD) is still the leading cause of death due to Obesity<sup>17</sup>. Abnormally high levels of low-density





lipoprotein cholesterol (LDL-C) and low levels of highdensity lipoprotein cholesterol (HDL-C) have been identified as independent coronary risk factors<sup>18-20</sup>. A significant reduction in coronary events is noted when plasma LDL-C levels are decreased and/or HDL-C levels are increased<sup>21-22</sup>.

A sedentary lifestyle is considered a risk factor for the development of coronary artery disease, <sup>23-25</sup> whereas regular exercise is associated with reduced Coronary Artery Disease mortality<sup>26</sup>. Increased HDL-C levels observed with regular exercise <sup>27</sup> might be partially responsible for this protection. Regular exercise, along with other lifestyle changes (smoking cessation, fat weight reduction, and a low fat diet) are now recommended as an adjunct to medical therapy in an effort to combat Coronary Artery Disease.

Although the favorable effects of regular exercise on HDL-C metabolism have been reported previously, <sup>28-29</sup> the amount of exercise necessary to increase HDL-C levels has not been well defined<sup>30</sup>. Previous studies suggest that an exercise threshold for the amount and intensity must be met or exceeded before favorable changes in HDL-C levels can occur. This exercise threshold varies considerably among studies, ranging from 8 to 18miles per week. Others suggest a doseresponse relationship. If exercise is to be used as a therapeutic approach in the treatment of dyslipidemia, these variables must be well defined to provide physicians and health professionals with the guidelines for prescribing a safe and effective exercise regime for their patients<sup>31-33</sup>.







Prolonged exercise can result in lowering of fasting plasma triglyceride (TG) concentration<sup>34</sup>. Repeated exercise on successive days can bring about a progressive decline in elevated TG levels. It is not known whether the lowering of TG is attributable to a specific effect of exercise on lipid metabolism, or to a decreased availability of substrate for TG synthesis secondary to the increased energy expenditure<sup>35</sup>.

Therefore, controversy still exists as to whether or not exercise has beneficial effects in obese Type 2 diabetics. Therefore, we studied efficacy of the treadmill walking exercise as a supplement to diet and drug in controlling the diabetic mellitus with obesity. Naturally, the patients don't like dietary restriction or exercise because the diet is self-denial and exercise means an effort on his part. The oral drug intake is the best treatment from the patients' point of view, but it has certain disadvantages and side effects. The regular endurance treadmill walking exercise can decrease BP, increase oxygen consumption and transport, and increases the collateral circulation.

In this study, it was found that six-week of aerobic treadmill exercise can facilitate the glycemic control by increased insulin sensitivity, improved fuel for oxidation, and increased storage of muscle glycogen as evidenced by the previous study<sup>36</sup>. The result of this showed that there is significant decrease in the blood glucose levels (FBS and PPBS) of the experimental group as compared to control group with p<0.0005 (Table I). The mechanism by which exercise facilitates the glycemic control was because working muscle is more







sensitive to insulin action than the muscle at rest, resulting in greater assimilation of glucose per unit of insulin during the exercise. Increased sensitivity of a muscle to insulin occurs with mild to moderate exercise. Exercise increases the blood flow to working muscles so that increases in size of per fused capillary bed and available number of insulin receptors, counterbalancing this increased sensitivity to insulin, ultimately leads to a concomitant decrease in the production of pancreatic insulin in NIDDM during exercise. As endurance exercises begin, muscle glycogen is available for a short As exercise is prolonged, glycogenolysis period. becomes the source of increased glucose availability. After 15 minutes of exercise, hepatic gluconeogenesis begins. When exercise is prolonged over 30 minutes, free fatty acid is generated by adipocyte lipolysis. In a diabetic, conversion from resting to working metabolism occurs more rapidly due to higher basal circulating levels of free fatty acid and circulating gluconeogenic substrates. Exercise facilitates blood lipid profile favorably, i.e., increases HDL-C and decreases total cholesterol, triglycerides as evidenced by our study (Table II).

Exercise increases the levels of HDL-C more systematically than dietary change or weight loss. The mechanism by which it increases the HDL-C content and decreases the LDL content is due to the stimulation of lipoprotein lipase activity in adipose tissue during the aerobic exercise<sup>37</sup>.

Endurance exercise can be prescribed for obese diabetic patients in the same manner as diet and insulin.





Like medication and diet, the intensity of exercise that a patient needs to perform can be quantified by mode of exercise, duration of prescribing the proper exercise, frequency of exercise and intensity of exercise. The initial exercise program should include moderate duration of 15 minutes with warm up and cool down phase. If the patient does not have musculo-skeletal injury or adverse symptoms, the duration may be gradually increased by 5 minutes every week. The optimal frequency is 5 days per week. The intensity of the exercise prescribed should be demanding to meet both the glycemic control, maintaining the Lipid profile and cardiovascular conditioning. The intensity of the exercise can be prescribed by, Target HR = 0.6 - 0.7(HRmax - HRrest) + HRrest and Borg's scale of RPE between 13-14 (some what hard) which corresponds to a Vo2 Max between 70-80%, which would give an intensity of exercise within the aerobic target zone as described by American College of Sports Medicine (ACSM 1990).

In this investigation, there was a significant decrease in cholesterol level of the experimental group compared to the control group (p < as 0.0001.Triglyceride content was decreased the in experimental group (p < 0.0005). However, there was no significant difference in Triglyceride content of the control group (Table II).

In addition to glycemic control and Lipid profile control, the experimental group showed significant decreases in BMI (p < 0.0001) as compared to the control group. (Table III).







The cardiorespiratory parameters like heart rate, respiratory rate, were recorded before and after the experimental period for both groups. In the experimental group at the end of six weeks, all experimental subjects showed a decrease in heart rate, respiratory rate as compared to the control group. This decrease in parameters shows conditioning effects of exercise. (Table IV & V).

## Conclusion

The Graded Aerobic Treadmill Exercise is a substantive tool in addition to drugs and diet in glycemic and Lipid Profile Control. Moderate exercise has an additional advantage in cardiorespiratory conditioning.

#### References

- 1. National Institutes of Health Consensus Development Conference Statement. Annals of Inter Med 1985:103; 147-151.
- 2. Mishra S, Wasir HS, Obesity as A Risk Factor for Coronary Artery Disease, JAPI 1997; 45: 555-558.
- C Haslett, ER Chilvers, JAA Hunter, NA Boon. Davidson's Principles and Practice of Medicine, 18<sup>th</sup> edition: 1999: Churchill Livingstone
- 4. Eugene Braunwald. Harrison's. Principles of Internal Medicine, 15th edition: 2001;McGraw-Hill Medical Publishing Division
- 5. Richard H Strauss. Sports Medicine, second edition: 1991; WB Saunders Company.
- 6. Karim Khan, Peter Brunkner.Clinical Sports Medicine, 1st edition: 1993; Mc Graw-Hill Book Company.
- 7. Richter EA, and Ruder man, NR, (1981) Diabetes and exercise. American Journal of Medicine. 70, 201-209
- 8. National Institute of Health (1987). Consensus development conference on diet and exercise in NIDDM. Diabetes care. 10, 639-644
- 9. Golderberg L, Elliot DL, Shutz rw, Kloster FE, Changes in Lipid and Lipoprotein Levels after Weight Training, JAMA 1984, 252: 504-506





- Jack H Willmore, David L Costill. Physiology of Sport and Exercise, 2<sup>nd</sup> edition: 1999
- Krall LP, and RS. Beaser, Joslin, (1989) Diabetes Manual. 12<sup>th</sup> edn. Lea and Febiger, Philadelphia. 1-19, 81-91.
- 12. Eriksson KF, and Lindgarde, F, (1991) Prevention of NIDDM by diet and exercise. Diabetologia. 34, 891-898.
- 13. Franz, MJ, (1997) Lifestyle modification for Diabetes management Endocrinology
- 14. and Metabolism clinics of North America 26, 499-509.
- 15. Giacca, A, and Elane, Y, (1998) Glucose production utilization and cycling in response to moderate exercise in obese subjects with NIDDM. Diabetes. 47, 1763-1770.
- 16. Wahren, J, and Felig, P, (1971) Glucose metabolism during leg exercises. J clinical investigation 50, 2715-2725.
- 17. Hermansen, L, and Saltin, B, (1969) Oxygen update during maximal Treadmill and Bicycle exercise. J. Applied Physiology 26, 31-37.
- Kokkinos PF, Holland JC, Narayan P, etal, Miles Run per Week and High Density Lipoprotein levels in Healthy Middle- aged Men, Arch Inter Med 1995: 155: 415-420.
- 19. Goldbourt U, Holtzman E, Neufeld HN. Total and high-density lipoprotein cholesterol in the serum and risk of mortality: evidence of a threshold effect. BMJ 1985: 290; 1239-1243
- 19.The Expert Panel. Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. Arch Intern Med 1988: 148; 36-69.
- 21. Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial, II: the relationship of reduction in incidence of coronary heart disease to cholesterol lowering. JAMA 1984: 251; 365-374
- Stamler J, Wentworth D, Neaton JD. Is the relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screens of the Multiple Risk Factor Intervention Trial (MRFIT). JAMA 1986: 256; 2823-2828.
- Morris JN, Clayton DG, Everitt MG, Semmence AM, Burgess EH. Exercise in leisure time: coronary attack and death rates. Br Heart J 1990: 63; 325-334.
- 24. Blair SN, Kohl HW III, Paffenberger RS Jr, Clark DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality: a prospective study of healthy men and women.JAMA 1989: 262; 2395-2401.







- 25. Fletcher GF, Blair SN, Bluementhal J, et al. Benefits and recommendations for physical activity programs for all Americans: a statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology. American Heart Association. Circulation. 1992: 86; 340-344.
- 26. Chandrashekhar Y, Anand IS. Exercise as a coronary protective factor. Am Heart J. 1991: 122; 1723-1739.
- 27. Paffenbarger RS Jr, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. The association of changes in physical activity level and other life style characteristics with mortality among men. N Engl J Med. 1993: 328; 538-545.
- Wood PD, Sefanic ML, Williams PT, Haskell WL. The effects on plasma lipoproteins of a prudent weight -reduction diet with or without exercise, in overweight men and women. N Engl J Med 1991: 325; 461-466.
- 29. Superko HR, Haskell WH. The role of exercising training in the therapy of hyperlipoprotenemia. Cardiol Clin. 1987: 5; 285-310.
- 30. Superko RH. Exercise training, serum lipids, and lipoprotein particles: is there a change threshold? Med Sci Sports Exerc. 1991: 23; 667-685.
- 31. Wood PD, Haskell WL, Blair SN, et al. Increased exercise exercise level and plasma lipoprotein concentrations: a one-year, randomized study in sedentary, middle-aged men. Metabolism 1983: 32; 31-38.
- 32. Lakka TA, Salonen JT. Physical activity and serum lipids: a cross sectional study in eastern Finnish men. Am J Epidemiol. 1992: 136; 806-818.
- 33. Leclerc S, Allard C, Talbot J, et al. High density lipoprotein cholesterol, habitual activity and physical fitness. Arteriosclerosis. 1985: 57; 43-51.
- 34. Gotto AM, Diet and Cholesterol guidelines and coronary heart disease, AM j Cardiol 1989: 13:503-507.
- 35. Gyntelberg F, Brenen R, Holloszy J, etal Plasma triglyceride lowering by exercise despite food intake in patients with type IV hyperlipoprotenemia, Am J Clin Nutr 1977; 30: 716-720.
- 36. Alexander Marble, Leo P Krall, Robert F Bradley.Joslin' Diabetes Mellitus, 12<sup>th</sup> edition:1985; K M Vargheese Company
- 37. Vranic, M, and Berger, M, (1979) Exercise and diabetes mellitus. Diabetes. 28, 147-162.
- 38. Oscal Lb, Patterson JA, Bogard DL, Beck RJ, Rothermel BL. Normalization of serum triglycerides and lipoprotein electrophoretic patterns by exercise. Am J Cardiol. 1972: 30; 775.

