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# The Role of Physical Exercise and Diet Modification on Lipid Profile and Lipid Peroxidation in Long Term Glycemic Control Type 2 Diabetics

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#### Abstract

**Objective:** Exercise has been considered a cornerstone of diabetes management, along with diet and medication. The aim of the present study was to assess the effect of yogic exercise along with diet management on glycemic control and lipid peroxidation.

**Methods:** The patients were clinically diagnosed Type 2 patients, controlling their glycemic from more than five years. Patients have been divided into two groups, Group-II, who controlled their glycemia by exercise and dietand Group-III, who controlled their glycemia by antidiabetic therapy (either oral hypoglycemic agent (OHA) or by insulin), and the Healthy control group is categorized as Group-I.The blood samples were analyzed for the estimation of blood sugars, HbA1c and EMDA (Erythrocyte Malondialdehyde) for lipid peroxidation.

**Results:** The levels of Fasting blood Sugar (FBS), Glycosylated hemoglobin (HbA1c) and Erythrocyte malondialdehyde (EMDA) was good controlled in Group-II patients as compared to Group III. So we have concluded that exercise and diet control have a beneficial effect on the metabolic control of diabetes management. HbA1c was significantly lower in group-II. These results would provide support for encouraging type 2 diabetic individuals who are already exercising at moderate intensity to consider increasing the intensity of their exercise to obtain additional benefits in both aerobic fitness and glycemic control.

**Conclusion:** Our study concluded that exercise and diet control have a beneficial effect on the metabolic control of diabetes management.

**Keywords:** Exercise; Glycemic control; Lipid peroxidation; Type 2 diabetes

# Introduction

India has the largest diabetic population in the world. Changes in eating habits, increasing weight and decreased physical activity are major factors leading to increased incidence of type 2 diabetes [1]. Epidemiological studies have shown that regular physical exercise associated with changes in dietary lifestyles, such as reduction in saturated fatty acid consumption and enhanced fiber intake, delay the onset of type 2 diabetes mellitus (DM2) [2-4]. Obesity and reduced levels of physical activity are directly associated with the insulin resistance that characterizes Type 2 diabetes. Exercise is widely perceived to be beneficial for glycemic control and weight loss in patients with type 2 diabetes. Yogic exercise (Kapalbhati), stimulates pancreas to release insulin, thereby helps to control diabetes mellitus.

Physical exercise has antiatherogenic effects because of its potential benefits on blood pressure levels, glucose tolerance, body weight and composition, plasma lipids, and lipoprotein metabolism [5-7]. Exercise reduces HbA1c by an amount that should decrease the risk of diabetic complications [8]. Yogic exercises to enhance the antioxidant defense mechanism in diabetics by reducing oxidative stress, which affects the lipid peroxidation [9].

The Aim of the present study was to assess the effect of exercise (physical activity) on glycemic control and lipid peoxidation in Type 2 Diabetes mellitus.

# Materials and Methods

# **Study Subjects**

Sixty-seven type 2 diabetic patients (without complication) who regularly attended medical out-patient department and/or diabetic clinic of J.A. Hospital, G.R. Medical College, Gwalior, M.P. India were enrolled in this study. The patients were clinically diagnosed type-2 diabetic subjects controlling their glycemia from more than five years. The patients were asked for mode of control of hyperglycemia, and type of exercise or yoga they were doing. Beside this other important information regarding diet, familyhistory of diabetes, type of medication was also asked to the patients to categorize them into appropriate group. The study was approved by the institutional ethical committee and written consent has been taken for the study before blood collection from the patients. These subjects were divided into three groups:

**Group I:** This group consisted of age-matched healthy control (n=50).

**Group II:** This group consisted of type-2 diabetic subjects (n=12) controlling their glycemia by exercise (30 minutes walking or yoga/day) and diet, no antidiabetic therapy was taken by these patients.

**Group III:** This group consisted of type-2 diabetic subjects (n=50) controlling their glycemia by antidiabetic therapy either oral hypoglycemic agents (OHA) or by insulin.

Age-matched healthy controls were defined as not having a major medical illness, no hospital admissions, no current medication, and a subjective perception of good health. None of the healthy subjects received any medication and trace element supplement in the previous

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2-3 months. The blood sample was taken from diabetic patients and healthy controls after an overnight fast under all aseptic precautions for analysis. All the samples were analyzed on the same day of collection.

#### Sample collections

The blood samples collected under all aseptic conditions after an overnight fast between 8:00 AM to 9:00 AM into EDTA, Citrate and Plain vial.

#### **Assay of Biochemical Parameters**

FPG was estimated by the method of Glucose Oxidase-Peroxidase (GOD-POD) by Trinder, [10] and quantified spectrophotometrically at 500 nm, Glycosylated hemoglobin was measured by BeenaRai&Pattabiramans Method [11] Total Cholesterol (TC) by Ferric Chloride and Sulfuric Acid Method. (1953) [12], Triglyceride estimated by Van handle method [13], High density lipoprotein cholesterol (HDL-c)by Burnstein Method [14], LDL and VLDL cholesterol were calculated by Friedwal's formula[15].

Friedwal's formula for LDL Cholesterol: Total cholesterol minus high-density lipoprotein (HDL) cholesterol minus VLDL cholesterol (estimated as triglyceride multiplied by 0.46).

LDL cholesterol (mmol/L) = Total cholesterol - HDL Cholesterol -VLDL cholesterol

Friedwal's formula for VLDL Cholesterol:

VLDL cholesterol (mmol/L) =  $0.46 \times$  Triglyceride

### Assay of lipid peroxides

Lipid peroxidation was estimated by measuring the level of Erythrocyte malonyldialdehyde (E-MDA) through thiobarbituric acid reaction Bidder and Jaeger [16].

#### **Statistical Analysis**

Data were expressed as a Mean±Standard deviation (SD). The unpaired t - test was used for the statistical analysis. The level of significance was considered as P value <0.05. Statistical analysis was

carried out using Statistical Product and Service Solutions (SPSS) software version 7.

#### Results

The demographic data and biochemical characteristics of the study subjects are summarized in Table 1. Patients' group (group-II, III) were compared statistically with age matched healthy control group (group-I). There were significant increases in the levels of FPG, HbA1c and EMDA (P<0.001) in Group-III patients where asnon significantly elevated in Group-II as compared to group-I (Table 1).

Significant difference in TC, TG, HDL cholesterol, VLDL cholesterol and LDL cholesterol was observed in group-III when compared to healthy control (group-I) subjects. Among the lipid profile, the level of triglyceride was significantly higher, whereas the level of HDL was lower in diabetic patients as compared to healthy individuals (Table 2).

### Discussion

Rejuvenation/regeneration of cells of the pancreas due to abdominal stretching during yoga exercise, which may increase utilization and metabolism of glucose in peripheral tissues, liver, and adipose tissues through an enzymatic process [17,18]. Exercises have a direct influence on pancreatic secretion by rejuvenation of the pancreatic cells through alternate abdominal contractions and relaxation. Reduction in blood glucose levels due to muscular exercise involved in the asanas [19]. Muscular relaxation, development and improved blood supply to muscles might enhance insulin receptor expression on the muscles causing increased glucose uptake by muscles and thus reducing blood sugar [20]. HbA1c was significantly lower in the exercise group (Group-II) than drug control group (Group-III). These results would provide support for encouraging type 2 diabetic individuals who are already exercising at moderate intensity to consider increasing the intensity of their exercise to obtain additional benefits in both aerobic fitness and glycemic control [21]. The improvement in the lipid levels after yoga could be due to increased hepatic lipase and lipoprotein lipase at the cellular level, which affects the metabolism of lipoprotein and thus increase uptake of triglycerides by adipose tissues [22,23].

Parameters	Group-I	Group-II	Group-III
	Healthy Control Subjects	Glycemic control by Diet and Exercise	Glycemic control byantidiabetic Drugs
	( n=50)	( n=12)	( n=50)
Age(Yrs.)	42.02 ± 11.46	53.5 ± 6.15°	53.40 ± 8.07°
Duration of Diabetes (Yrs.)	-	5.33 ± 0.89	7.42±4.22
FBG (mmol/L)	4.27 ± 0.42	4.91 ± 0.83ª	5.50 ± 0.51°
HbA1c(MH/M Hb)	0.26 ± 0.009	0.551 ±0.12°	0.61 ± 0.21°
EMDA(nmol/gmHb/2 hrs.)	125.11 ± 12.48	134.50 ± 23.76 NS	155.94± 41.10°

The values are in Mean ± Standard deviation (SD); P values: \*p<0.05, \*p<0.001, p=Non significant (NS).Control vs. Group II and Group III. **Table 1:** Showing levels of FBG, HbA1c and E-MDA in Group-I, Group-II and Group-III subjects (Mean ± SD).

Parameters	Group-l	Group-II	Group-III
	Healthy Control Subjects	Glycemic control by Diet and Exercise	Glycemic control byantidiabetic Drugs
	( n=50)	(n=12)	(n=50)
Triglycerides (mmol/L)	1.44 ±0.28	2.30 ±0.59ª	2.37±0.73°
Total cholesterol (mmol/L)	4.79 ±0.57	6.06 ±1.88°	6.08 ±1.77°
HDL cholesterol (mmol/L)	1.01 ±0.17	0.683 ±0.15°	0.75± 0.15°
VLDL cholesterol(mmol/L)	0.65 ±0.13	1.05 ±0.27°	1.10 ± 0.35°
LDL cholesterol (mmol/L)	3.13 ±0.60	4.36 ±1.76ª	4.19 ± 1.63°

The values are in Mean ± Standard deviation (SD); *P* values:<sup>a</sup>p<0.05, <sup>c</sup>p<0.001 ,p=Non significant (NS).Control vs. Group II and Group III. **Table 2:** Showing status of Lipid Profile in Group-I, Group-II andGroup-IIIsubjects (Mean ± SD).

Most clinical trials on the effects of physical activity interventions in type 2 diabetes have had small sample sizes and therefore inadequate statistical power to determine the effects of exercise on glycemic control [24]. Yoga postures can lead to improvement in the sensitivity of the beta cells ( $\beta$ -cells) of the pancreas to the glucose signal and also the improvement in insulin sensitivity [25]. Direct stimulation of the pancreas by the yoga postures can rejuvenate its capacity to produce insulin [26]. Regeneration of pancreatic β-cells promotes blood circulation in the region of the pancreas [27]. Literature reviews revealed that exercises help to enhance antioxidant enzyme systems in diabetics by reducing oxidative stress. Reactive oxygen species (ROS) are highly reactive and readily oxidize membrane phospholipids containing unsaturated fatty acids. The lipid hydroperoxides generated by this process yield cytotoxic products such as malondialdehyde (MDA) [28]. Some studies supported our results [21] that yogic exercises have enhanced the antioxidant defense mechanism in diabetics by reducing oxidative stress [29]. Whereas some studies reported that Lipid peroxidation (LPO) is increased during exercise and this increase is inversely related to oxygen consumption. Oxygen consumption initiates enhanced formation of (ROS) [30,31].

A further research is needed on a large sample size to enhance our present understanding statistically, especially on Group- II of type 2 diabetic patients.

# Conclusion

Our study concluded that diet control and physical activity by yoga or exercises have a protective effect against oxidative stress and hyperglycemia in long term glycemic control Type 2 diabetics.

#### References

- Sahay BK, Sahay RK (2002) Lifestyle modification in management of diabetes mellitus. J Indian Med Assoc 100: 178-180.
- 2. Diabetes Prevention Program (DPP) Research Group (2002) The Diabetes Prevention Program (DPP): description of lifestyle intervention. Diabetes Care 25: 2165-2171.
- Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C (2004) Physical activity/exercise and type 2 diabetes. Diabetes Care 27: 2518-2539.
- American Diabetes Association (2002): Diabetes mellitus and exercise (Position Statement). Diabetes Care 25 (Suppl. 1):S64–S68.
- 5. Goodyear LJ, Kahn BB (1998) Exercise, glucose transport, and insulin sensitivity. Annu Rev Med 49: 235-261.
- Gupta AK, Ross EA, Myers JN, Kashyap ML (1993) Increased reverse cholesterol transport in athletes. Metabolism 42: 684-690.
- Ratner R, Goldberg R, Haffner S, Marcovina S, Orchard T, et al. (2005) Impact of intensive lifestyle and metformin therapy on cardiovascular disease risk factors in the diabetes prevention program. Diabetes Care 28: 888-894.
- Normand G Boulé, Elizabeth Haddad, Glen P. Kenny, George A Wells, Ronald J. Sigal (2001) Effects of Exercise on Glycemic Control and Body Mass in Type 2 Diabetes Mellitus: A Meta-analysis of Controlled Clinical Trials. JAMA 286:1218-1227.
- Singh S, Malhotra V, Singh KP, Madhu SV, Tandon OP (2004) Role of yoga in modifying certain cardiovascular functions in type 2 diabetic patients. J Assoc Physicians India 52: 203-206.
- Veiga F, Fernandes C, Teixeira F (2000) Oral bioavailability and hypoglycaemic activity of tolbutamide/cyclodextrin inclusion complexes. Int J Pharm 202: 165-171.
- BeenaRai K, Krishna Sharma K, Pattabiraman TN (1984) A short-duration colorimetric method based on phenol-sulfuric acid reaction for the estimation of glucosylhemoglobin. Biochem Med 31: 65-72.
- Zlatkis A, Zak B, Boyle AJ (1953) A new method for the direct determination of serum cholesterol. J Lab Clin Med 41: 486-492.

14. Burnstein M, Scholnick HR, Morfin R (1991) Clinical practical Biochemistry by Harvoldvarley. (5th edn); CBS Publisher, New Delhi, India Vol-1, 665-667.

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- Friedewald WT, Levy RI, Fredrickson DS (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. ClinChem 18: 499-502.
- Bidder TG, Jaeger PD (1982) Malondialdehyde production by erythrocytes from alcoholic and non-alcoholic subjects. Life Sci 30: 1021-1027.
- Dang KK, Sahay BK. (1999) Yoga and Meditation, Medicine update. In: Singh MM, editor. The Association of Physicians of India. Vol 9. New Delhi: APICON, The Association of Physicians of India conference; 502–512. Part 1, chapters 57 and 58.
- Sahay BK, Murthy KJR. (1988) Long term follow up studies on effect of yoga in diabetes. Diab Res ClinPract 5(suppl.1):S655.
- Mourier A, Gautier JF, De Kerviler E, Bigard AX, Villette JM, et al. (1997) Mobilization of visceral adipose tissue related to the improvement in insulin sensitivity in response to physical training in NIDDM. Effects of branched-chain amino acid supplements. Diabetes Care 20: 385-391.
- 20. Chandratreya S. (2012) Diabetes & Yoga.
- Boulé NG, Haddad E, Kenny GP, Wells GA, Sigal RJ (2001) Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA 286: 1218-1227.
- 22. Delmonte MM (1985) Biochemical indices associated with meditation practice: a literature review. NeurosciBiobehav Rev 9: 557-561.
- Tulpule TH, Shah HM, Shah SJ, Haveliwala HK (1971) Yogic exercises in the management of ischaemic heart disease. Indian Heart J 23: 259-264.
- Mahapure HH, Shete SU, Bera TK (2008) Effect of yogic exercise on super oxide dismutase levels in diabetics. Int J Yoga 1: 21-26.
- 25. Manjunatha S, Vempati RP, Ghosh D, Bijlani RL (2005) An investigation into the acute and long-term effects of selected yogic postures on fasting and postprandial glycemia and insulinemia in healthy young subjects. Indian J PhysiolPharmacol 49: 319-324.
- Ramaiah SA. Yoga Therapy for Diabetes: Washington, D.C. (1986) Study, International Conference on Traditional Medicine. Madras, India: Published by Siddha Medical Board, Govt. of Tamil Nadu.
- 27. Yogalink (2012) A community service donated by samyama yoga.
- Leeuwenburgh C, Heinecke JW (2001) Oxidative stress and antioxidants in exercise. Curr Med Chem 8: 829-838.
- Sayar N, Terzi S, Yilmaz HY, Tangurek B, Bilsel T, et al. (2007) Exercise-induced increase in lipid peroxidation in patients with chronic heart failure: relation to exercise intolerance. Cardiology 108: 307-313.
- Roberts CK, Vaziri ND, Barnard RJ (2002) Effect of diet and exercise intervention on blood pressure, insulin, oxidative stress, and nitric oxide availability. Circulation 106: 2530-2532.
- 31. Stadtman ER, Berlett BS (1998) Reactive oxygen-mediated protein oxidation in aging and disease. Drug Metab Rev 30: 225-243.