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Research Article

BENEFIT OF REGULAR EXERCISE IN TYPE 2 DIABETIC SUBJECTS

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ABSTRACT

Adipose tissue functions as an endocrine organ, and also a role in fuel storage, thermal insulation, and mechanical protection, releasing biologically active and diverse cytokines. Exercise is known to increase the insulin sensitivity in muscles. Most of the type 2 diabetes occurs due to life style changes and increased fat accumulation resulting in insulin insensitivity. An increase in physical activity is known to influence the adiponectin, a cytokine that is released from the adipose tissue. Hence, this study was undertaken to understand the effect of regular exercise, on the levels of adiponectin of diabetic subjects. Forty six diabetic subjects who have not exercised regularly and 40 diabetic subjects who exercised regularly were selected. They were matched with the normal healthy 40 subjects who exercised regularly for 1 hour daily and 72 subjects who had not exercised regularly. Adiponectin concentration was estimated using ELISA kit. The results showed better increase in adiponectin levels in diabetic subjects than the normal subjects. Mean value between the normal subjects showed no significant change (p=0.291) within the group but the level was significantly increased in the diabetic group (p=0.0001). Hence with the present study, it is concluded that long term exercising in diabetic subjects was much beneficial in increasing the adiponectin level, hence a decrease in risk factors of cardiovascular disease.

Keywords: Adiponectin, cytokine, exercise, type 2 diabetes.

INTRODUCTION

Adipose tissue was considered as a major site for energy storage, important for energy homeostasis, and also an important endocrine organ that secrets a number of biologically active "adipokines"^{1, 2.}

It is known via the secretion of adipocytokines that adipose tissue regulates energy balance, glucose and lipid metabolism. The levels of adipokines like leptin, adiponectin and tumor necrosis factor- α (TNF- α) are different in diabetic and/or obese subjects compared with nondiabetic, non-obese individuals. Secreted from white adipose tissue, concentrations of adiponectin are reduced in the presence of metabolic and cardiovascular disease such as type 2 diabetes and obesity. Together with the weight loss and exercise are considered effective in the prevention and treatment of arteriosclerosis and skeletal muscle insulin resistance³⁻⁵ while weight loss and/or energy intake restriction have been connected with elevated plasma adiponectin concentrations, studies investigating the effects of exercise on adiponectin release have given inconsistent findings⁶⁻¹¹. Kraemer *et al*⁶ investigated the acute effects of

short-term, running exercise on plasma adiponectin secretion in healthy subjects but observed no changes. Other studies, exploring the more long-term benefits of exercise training, also reported no changes in basal plasma adiponectin concentration^{7, 11-13}. In contrast, Kriketos *et al.*⁸ recently reported substantially elevated fasting plasma adiponectin levels (260% above baseline values) after 2–3 bouts of low to moderate intensity exercise. Studies investigating the effects of prolonged, moderate intensity exercise in diabetic subjects are presently lacking. Therefore, the first aim of the present study was to determine the effect of prolonged moderate exercise on the adipokines of both diabetic and non-diabetic groups.

MATERIALS AND METHODS

This was a cross sectional study of 229 cases of type 2 diabetics and 205 healthy individuals of Dakshina Kannada district. Out of 229 subjects, we had only 40 persons who were diabetic and had exercised regularly for 1 hour. These were matched with 46 diabetic subjects who have not exercised regularly. Further in the normal healthy subjects we had 40 subjects who exercised regularly for 1 hour daily and 72 subjects who had not exercised regularly. The study population was aged between 30-70 years. Persons with history of type 2 diabetes for at least one year, without any micro and macro vascular complications and on oral or on diet control were selected as the study group. A similar age matched healthy individual was selected as the control group. Criteria for inclusion of study subjects -should have been recognized as diabetic for at least one year, should be on oral hypoglycemic drugs or diet control, free from diabetic neuropathy, nephropathy and retinopathy, free from any pre-existing cardio vascular disease, non-pregnant in case of females and free from usage of oral contraceptives. Relevant examination was done to establish the inclusion and exclusion criteria. Written informed consent was obtained from the selected subjects. Ten ml of blood was drawn and separated blood samples were stored at -30°C for marker estimation. Adiponectin was estimated using Ravbiotech Kits using ELISA reader. The results were analyzed using SPSS 10.0. Student's unpaired't' test was used to compare between the two groups and Pearson correlation was used to find the correlation between the subjects. P value <0.01 was taken as the level of significance. The Inter assay CV% and intra assay CV% for adiponectin estimation was <10% in the analysis of biochemical parameters. Ethical clearance for the study was obtained from Yenepoya University Ethics Committee, Mangalore.

RESULTS

The demographic data of the study population is shown in the table below. Table 1 shows the adiponectin concentration among the normal exercising group and the normal subjects with no exercise. Table 2 shows the adiponectin concentration among the diabetic exercising group and the diabetic subjects with no exercise.

DISCUSSION

Adipose tissue has recently been revealed to secrete a variety of bioactive peptides, called adipocytokines, that is able to potentially impact on glucose and lipid metabolism¹⁴⁻¹⁶.

The result of our study in diabetic individual indicates the effects of long term exercise on adiponectin levels. Exercise is often associated with reduction of body fat, which could elucidate the effect of increased adiponectin and improvement in the insulin sensitivity. Intense exercise increases the capillary density among muscle fibers, a shift of muscle fiber type to type IIa and improvement in intracellular signaling of insulin. The increased aerobic capacity after exercise training might be the possible mechanism behind it. Further research has also shown that exercise improves insulin sensitivity at least in part through AMP kinase pathway activation¹⁷. It has recently been shown that adiponectin also increases muscular insulin sensitivity through the same pathway¹⁷.

Studies have shown that adiponectin levels were increased after 7-month aerobic exercise programme in eight young obese female subjects (BMI>25 kg/m2)¹⁸. The subjects' exercise training decreased BMI, percentage body fat, leptin and TNF-a. Thus, this exercise-training programme was of a sufficient caloric expenditure and duration to reduce body fat and increase adiponectin concentrations in young obese female subjects¹⁸. Since our study was a cross sectional study, we failed to show any such correlation.

But our study showed some affirmative findings in the level of adiponectin in diabetic subjects who exercised regularly than those who did not exercise and also among the normal healthy subjects. We conclude that the reason for this positive statement is that the intense long term exercise training induces an increase in insulin sensitivity, which will be reflected on the glycemic index and an increase in adiponectin. These changes are more prominent in type 2 diabetic subjects than in healthy individuals.

Table 1: Mean, SD of adiponectin in the normal subjects with exercise and without exercise

Adiponectin(mg/l)	Normal with exercise (n=40)	Normal with no exercise (n=72)	Significance
Mean ± SD	19.063 ± 9.996	17.303 ± 7.424	0.2919 ^{ns}

Table 2: Mean, SD of adiponectin in diabetic subjects with exercise and without exercise

Adiponectin(mg/l)	Diabetic with exercise (n=40)	Diabetic with no exercise (n=46)	Significance
Mean ± SD	34.143 ± 8.519	12.261 ± 4.292	0.0001***

*** Significant at the level of 0.0001



Fig. 1: Level of adiponectin among the diabetic and normal subjects with and without exercise for comparison

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