**INTRODUCTION**

Adipose tissue was considered as a major site for energy storage, important for energy homeostasis, and also an important endocrine organ that secretes 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-\(\alpha\) (TNF-\(\alpha\)) are different in diabetic and/or obese subjects compared with non-diabetic, 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 resistance\(^3\)\(^-\)^\(^5\). 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 findings\(^6\)\(^-\)^\(^11\).

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.\(^8\)
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 adipokines, that is able to potentially impact on glucose and lipid metabolism\textsuperscript{14-16}. 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 activation\textsuperscript{17}. It has recently been shown that adiponectin also increases muscular insulin sensitivity through the same pathway\textsuperscript{17}.

Studies have shown that adiponectin levels were increased after 7-month aerobic exercise programme in eight young obese female subjects (BMI>25 kg/m\textsuperscript{2})\textsuperscript{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 subjects\textsuperscript{18}. 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

<table>
<thead>
<tr>
<th>Adiponectin(mg/l)</th>
<th>Normal with exercise (n=40)</th>
<th>Normal with no exercise (n=72)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>19.063 ± 9.996</td>
<td>17.303 ± 7.424</td>
<td>0.2919**</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Adiponectin(mg/l)</th>
<th>Diabetic with exercise (n=40)</th>
<th>Diabetic with no exercise (n=46)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>34.143 ± 8.519</td>
<td>12.261 ± 4.292</td>
<td>0.0001***</td>
</tr>
</tbody>
</table>

** Significant at the level of 0.0001

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

ACKNOWLEDGEMENT
We would like to express our gratitude to Dr. G.S. Chandrashekar, Senior Physician & Cardiologist of Adarsha Hospital & Institute of Cardio Diabetes, Trauma & Joint Replacement, Udupi, Karnataka for his support in conducting this study.

REFERENCES


