Effect of Duration of Starvation on Lipid Profile in Albino Rats

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ABSTRACT: This study was carried out using forty Wistar rats of both sexes and the test groups were differently starved according to time duration. Blood samples were collected from the rats through ocular puncture at intervals and was used for the analysis of lipid profile. There was no significant increase ($p>0.05$) in the body weights of the test animals compared with the body weights of the animals in control group at 0 hour of the experiment. The concentrations of total cholesterol, triacylglycerol, HDL and LDL of animals of the test groups were not significant ($p<0.05$) compared with the control at 0 hour of the experiment. There was no significant alteration ($p>0.05$) in the concentrations of total cholesterol of the test group animals compared with the control at 6 and 12 hours duration, while groups 3 (animals starved and received water) and 4 (animals starved and received fruits only) had elevated concentrations of total cholesterol ($p<0.05$). In triacylglycerol, a trend of results not significant ($p>0.05$) was observed at starvation intervals of 6 to 48 hours when a comparison was made between the test groups and control. When the high density lipoprotein and low density levels of the test groups were compared with the control, there was no significant difference ($p>0.05$).

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INTRODUCTION

Malnutrition and starvation can be caused by diseases, injuries, the range the animal lives on or the environmental conditions it lives in. Starvation and malnutrition occur in several wild life species and routinely eliminate the young, old, weak and sick animals. Malnutrition involves deficiency of not only the macronutrients –fats, proteins and carbohydrates, but also results in subphysiological concentration of most micronutrients. Many antioxidant defense systems depend on micronutrients or are micronutrients themselves (Evans and Halliwell, 2001). Therefore, one would expect a gross derangement of the antioxidant defense mechanisms in malnutrition. The role of oxidative stress is clear and well known in the pathogenesis of acquired malnutrition (Tatli *et al.*, 2000).

Hypoxia, immobilization and starvation are among the stressful physical stimuli applied to experimental animals. Stress is an adaptive response that prepares the organisms towards threat. It induces strain upon both emotional and physical endurance which has been considered a basic factor in aetiology of a number of diseases e.g. cardiovascular diseases, cancer, diabetes mellitus, etc (Halliwell and Gutteridge, 1984). Most investigations concerning the influence of prolonged starvation on the metabolic responses in mammals report that the activity of glucose-degrading enzymes and those of lipogenesis was depressed, whereas the fatty acids derived from triacylglycerols hydrolysis were preferentially used as fuels through the corresponding oxidative pathways (Shimeno *et al.*, 1997; Dou *et al.*, 2002; Guderley *et al.*, 2003).

Present research on starvation in vertebrates is connected with studying leptin that serves as a mediator of the adaptation to fasting. In humans, serum leptin concentrations as well as plasma levels of metabolic parameters (glucose, cholesterol, lipids) change rapidly after short term starvation (Boden *et al.*, 1996).

It is known that deprivation of energy supply induces a delay in the development of some vital functions in mammals: puberty starts later, the reproductive age prolongs, and ageing starts later and deterioration of immunity and health is delayed (Banks and Lebel, 2002).

Oxidative metabolism of cells is a continuous source of reactive oxygen species (ROS), resulting from univalent reduction of O_2, which can damage most cellular components leading to cell death. Under severe conditions, the rate of generation of ROS exceeds that of their removal and oxidative stress occurs (Sies, 1986; Di Giulio *et al.*, 1995; Halliwell and Gutteridge, 2000; Livingstone, 2001). In this sense, starvation has been reported to have pro-oxidant effects in mammals.It is during starvation that increased ROS generation is not adequately neutralized by antioxidant systems (Robinson *et al.*, 1997; Domenicali *et al.*, 2001).

Complete fasting is accompanied by substantial lipolysis (Samra *et al.,* 1996, Vaisman *et al.*, 1990). The report of (Savendahl *and Underwood,* 1999) shows that in normal weight subjects, increased serum cholesterol associated with the amount of weight loss was observed between 2days to 1wk fasting. Food deprivation can cause a shift from lipogenesis to lipolysis increased fatty acid turnover and reduction in protein anabolism (Buyse *et al.*, 2002).

The present study is aimed at evaluating the effect of short starving periods (6, 12, 24, 48 hours) on the lipid profile of rats.

MATERIALS AND METHODS

Animals

The experimental animals used for this study were Wistar albino rats of both sexes. The average age of the rats was 12 weeks old. The rats were obtained from the animal house of the Faculty of Biological Sciences, University of Nigeria, Nsukka (UNN). The animals were acclimatized for two weeks under standard laboratory conditions. They were housed in wire-meshed cages at ambient temperature with 12 hour day–night cycle and fed with commercial rat chow (pelletised growers feed) and water *ad libitum.*

Chemicals/Reagents/Samples

All chemicals used in this study were of analytical grade and products of May and Baker, England; BDH, England and Merck, Darmstadt, Germany. Reagents used for all the assays were commercial kits and products of Randox, USA; QCA, Spain; Teco (TC), USA; Biosystem Reagents and Instruments, Spain. Blood samples were collected at intervals through ocular puncture. The blood was allowed to clot and serum separated, which was then used for assaying of some parameters.

Experimental Design

Forty male Wistar rats of both sexes were housed in separate cages, acclimatized for fourteen days and then divided into Control group of four rats and three groups of three rats each.

The first, being control (n = 4) was maintained on normal rat chow and water *ad libitum*. The experimental animals formed the second, third and fourth groups. Each group contained 3 rats and had 4 types of time period associated with it. So, each group contained 12 rats (n = 12).

Group 1 animals were the Control, fed with the normal rat diet and water *ad libitum*.

Group 2 animals were starved of feed and water.

Group 3 animals were starved but received water.

Group 4 animals were starved but received fruits (carrots).

The parameters assayed were:

- Total serum cholesterol.
- Low density lipoprotein (LDL).
- High density lipoprotein (HDL).
- Triacyglycerol (TAG).

Total Cholesterol Determination [Using QCA Commercial Kit; Allain *et al*. (1976)]

Principle

The total cholesterol determination using QCA Commercial Enzyme kit is based on the assay principle that total cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator, coloured quinonic derivative is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of p-hydroxybenzoic acid and peroxidase.

High Density Lipoproteins (HDL) –Cholesterol Determination [Using QCA Commercial Kit; Albers *et al*. (1978)]

Principle

Low density lipoprotein (LDL) and Very low density lipoprotein (VLDL) are lipoproteins precipitated from serum by the action of a polysaccharide, in the presence of divalent cations. Then, the high density lipoprotein–cholesterol (HDL–Cholesterol) present in the supernatant, is determined.

Low Density Lipoprotein-Cholesterol Determination [Using QCA Commercial Kit; Assmann *et al*. (1984)]

Principle

Low density lipoprotein–Cholesterol (LDL–Cholesterol) can be determined as the difference between total cholesterol and cholesterol content of the supernatant after precipitation of the LDL fraction by polyvinyl sulphate (PVS) in the presence of polyethyleneglycol monomethyl ether.

Determination of Serum Triacylglycerols (Colorimetric Method of Tietz, 1990).

Principle

This method is based on the fact that triacylglycerols undergo enzymatic hydrolysis to yield H_2O_2. This hydrogen peroxide produces a quinoneimine which when reacted with 4-aminophenazone which absorbs light at 500 nm.

Statistical Analysis

The results were expressed as mean ± SD and tests of statistical significance were carried out using student t-test and both one-way and two-way analysis of variance (ANOVA). The means were separated using Duncan Multiple Test. The statistical package used was Statistical Package for Social Sciences (SPSS); version 17.

RESULTS

Effect of Starvation on Mean Serum Total Cholesterol Concentrations of Wistar Albino Rats at Various Intervals

There was no significant ($p>0.05$) decrease in the concentration of serum total cholesterol of the control animals at 12, 24 and 48 hours compared with the serum total cholesterol concentration of the control animals at 0 hour (Fig. 1). Generally, there were no significant differences ($p>0.05$) in the concentrations of serum total cholesterol of animals in the test groups compared with the animals in the control group at 0 hours as shown in Fig. 1.

Fig. 1 shows no significant difference ($p>0.05$) in the concentrations of serum total cholesterol between the control and all the test groups at the 6^{th}-hour interval of starvation. However, the serum total cholesterol concentrations of all test experimental groups at the 12^{th}-hour interval of starvation increased significantly ($p<0.05$) compared with the control. There was no significant difference ($p>0.05$) in the concentrations of serum total cholesterol between the test groups (groups 2, 3 and 4) within the duration of 12 hours.

When considering starvation at the 24-hour interval, significant ($p<0.05$) elevated concentrations of serum total cholesterol was observed (Fig. 1) in all the test groups in comparison with the control. There was also significant difference ($p<0.05$) between group 2 (starved of feed and water) and group 4 (starved and received fruits only) at the interval of 24 hours of starvation (Fig. 1). In the same vein, significant increase ($p<0.05$) in total cholesterol concentrations was observed in all the test groups compared with the control at the 48^{th} hour interval of starvation (Fig. 1).

Considering differences between the different time intervals of starvation in each experimental group, no significant differences ($p>0.05$) were observed as recorded in Fig. 1 between the different time intervals of starvation in group 1. Significant difference ($p<0.05$) exists in the cholesterol concentrations between 6^{th} hour interval of starvation and other time intervals (12, 24 and 48 hours of interval of starvation) in the group 2 (starved of feed and water). Similar trend was observed in the same group 2 when comparing 48 hours and other hours (6, 12 and 24 hours) of starvation. There was a significant difference ($p<0.05$) in serum total cholesterol concentrations between 6 hours of starvation and other intervals (12, 24 and hours) of starvation under group 3. In group 3, significant difference ($p<0.05$) in the concentrations of total cholesterol was observed between 48 hours of starvation and that of 6 and 12 hours of starvation ($p<0.05$; Fig. 1). No significant difference ($p>0.05$) was observed in group 4 serum total cholesterol concentrations between all the intervals of starvation with exception of 6 hours of starvation. However, Fig. 1 shows significant difference ($p<0.05$) in the concentrations of serum total cholesterol between 6 hours of starvation and other intervals (12, 24 and 48 hours) of starvation as observed in group 4 (starved but received fruits only).

Effect of Starvation on Mean Triacyglycerol Concentrations of Wistar Albino Rats at Various Intervals

The concentrations of triacylglycerol (TAG) of animals in the test groups did not alter significantly ($p>0.05$) compared with the TAG concentrations of the animals in the control group at 0 hour of the experiment (Fig. 2). Fig. 2 shows no significant difference ($p>0.05$) in the concentrations of triacylglycerol (TAG) between the control and the test groups at 6 hours interval of starvation. Similar pattern of result was obtained at starvation intervals of 12, 24 and 48 hours.

On the aspect of time-dependent effects in individual groups, there was no significant difference ($p>0.05$) in the concentrations of TAG between 6 hours of starvation and other hours (12, 24 and 48 hours) of starvation and the control (Fig. 2) . On the contrary, Fig. 2 shows that significant difference ($p<0.05$) exists in group 2 (starved of feed and water) between 48 hours of starvation and other intervals of starvation (6, 12 and 24 hours). In group 2, there was significant difference ($p<0.05$) in TAG concentrations between 6 hours of interval and 48 hours of interval of starvation but no significant difference ($p>0.05$) between other intervals of starvation (Fig. 2). No significant difference ($p>0.05$) was observed in the concentrations of TAG between all the time intervals of starvation as found in group 4 (Fig. 2).

Effect of Starvation on Mean High Density Lipoprotein Concentrations of Wistar Albino Rats at Various Intervals

The concentrations of high density lipoprotein (HDL), as observed in Fig. 3, showed no significant ($p>0.05$) differences in the test groups (groups 2, 3 and 4) compared with the HDL concentrations of animals in the control group at 0 hour of the experiment. Results (Fig. 3) show that the high density lipoprotein (HDL) concentrations of the test groups (Groups 2, 3 and 4) were not significant ($p>0.05$) when compared with the control after the duration of the starvation (6 to 48 hours). There were no time-dependent HDL differences in the rats after the duration of 6 to 48 hours. Also, no significant differences in the HDL concentrations of the rats in control group as well as groups 2 to 4 under the 6 to 48 hours of experiment was seen (Fig. 3).

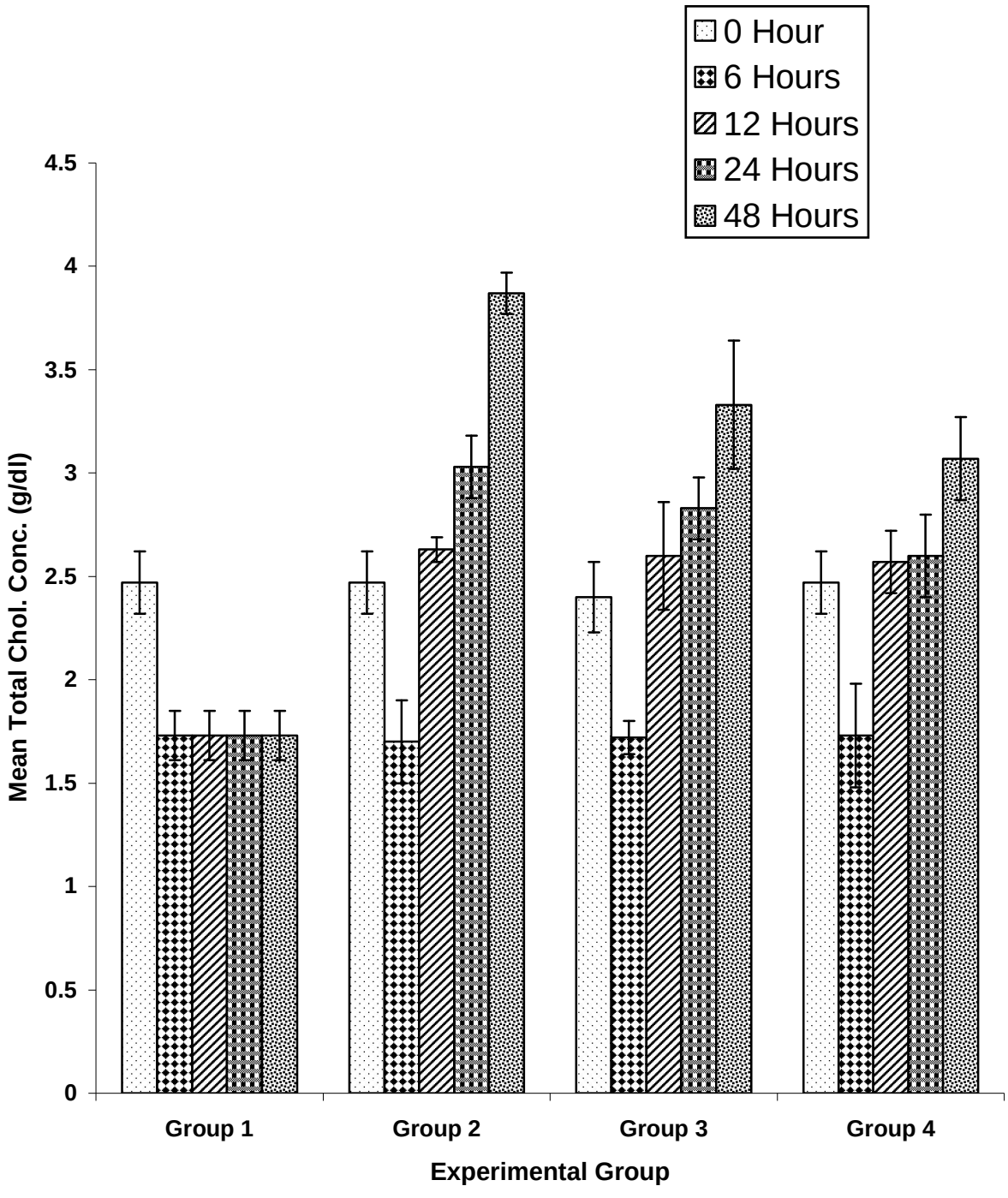

Fig. 1: Effect of starvation on the serum total cholesterol concentration of rats at various time intervals

Group 1: Control (Normal feed and water
Group 2: Starved of feed and water
Group 3: Starved but given water
Group 4: Starved but fed with fruit

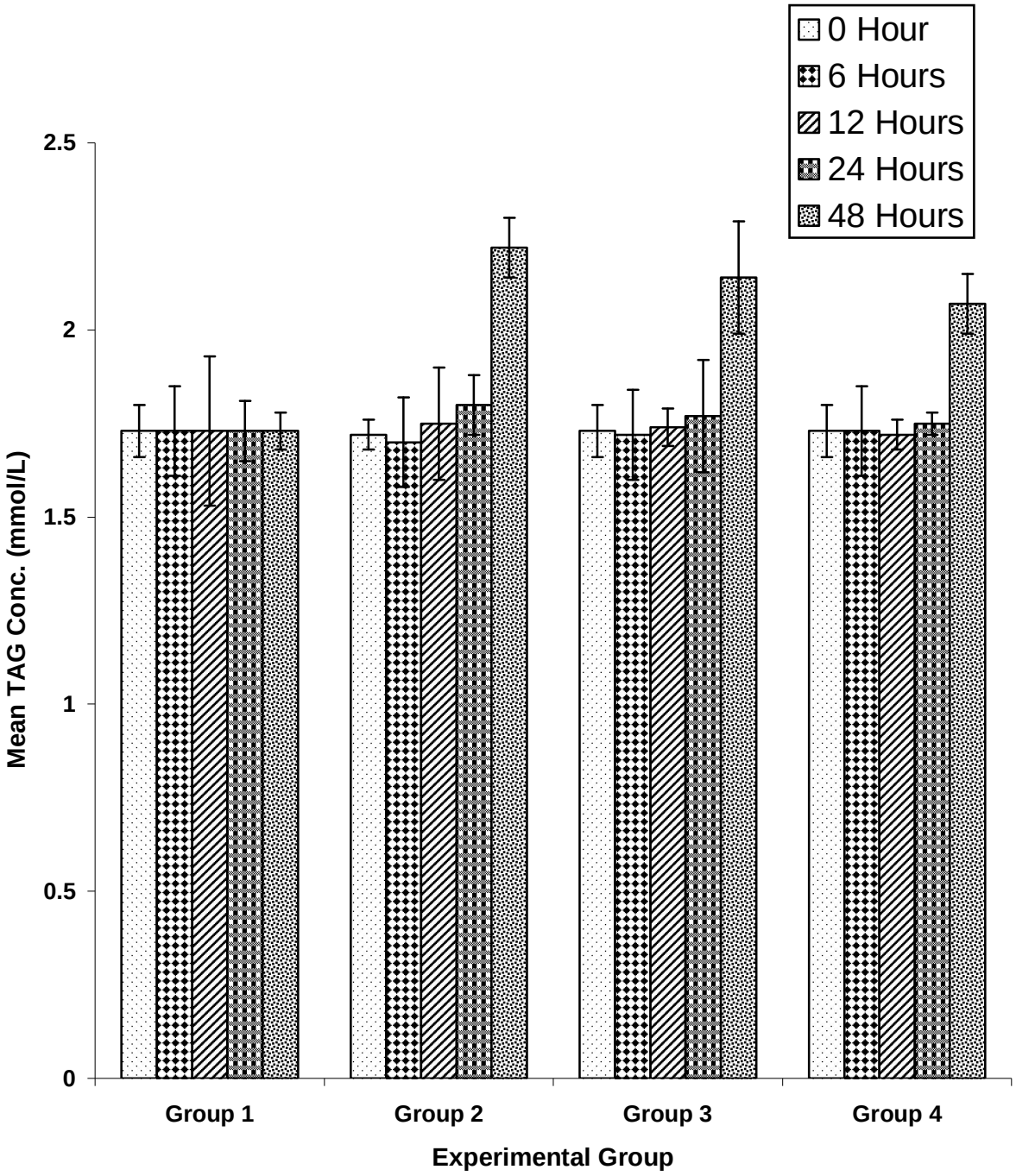

Fig. 2: Effect of starvation on the serum triacylglycerol concentration of rats at various time intervals

Group 1: Control (Normal feed and water
Group 2: Starved of feed and water
Group 3: Starved but given water
Group 4: Starved but fed with fruit

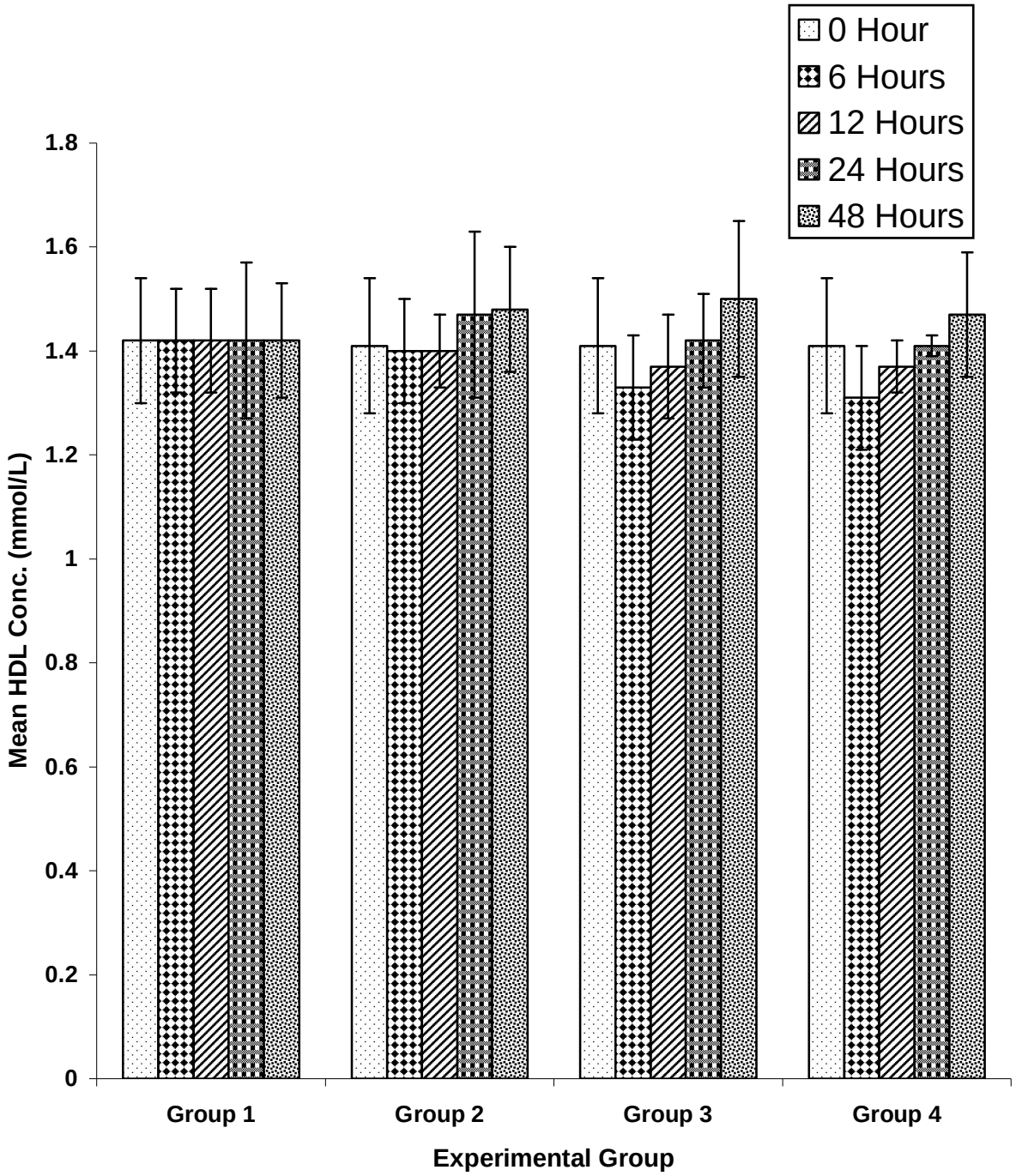

Fig. 3: Effect of starvation on the serum high density lipoprotein concentration of rats at various time intervals

Group 1: Control (Normal feed and water
Group 2: Starved of feed and water
Group 3: Starved but given water
Group 4: Starved but fed with fruit

Effect of Starvation on Mean Low Density Lipoprotein Concentrations of Wistar Albino Rats at Various Intervals

Fig. 4 shows no significant ($p>0.05$) concentrations of low density lipoprotein (LDL), of the test groups (groups 2, 3 and 4) compared with the concentrations of LDL of the animals in the control group at 0 hour of the experiment. In Fig 4, a non- significant difference ($p>0.05$) in the low density lipoprotein (LDL) concentrations between the test groups and the control after 6 to 48 hours duration was observed. Then, under 6 to 48 hours time intervals, the control did not show statistical difference ($p>0.05$; Appendix a, b, c, d). Low density lipoprotein (LDL) concentrations of groups 2 to 4 under 6 hours of starvation were found to decrease significantly ($p<0.05$) as compared with group 2 of 12 to 48 hours and likewise to other time intervals (12, 24 and 48 hours time intervals respectively).

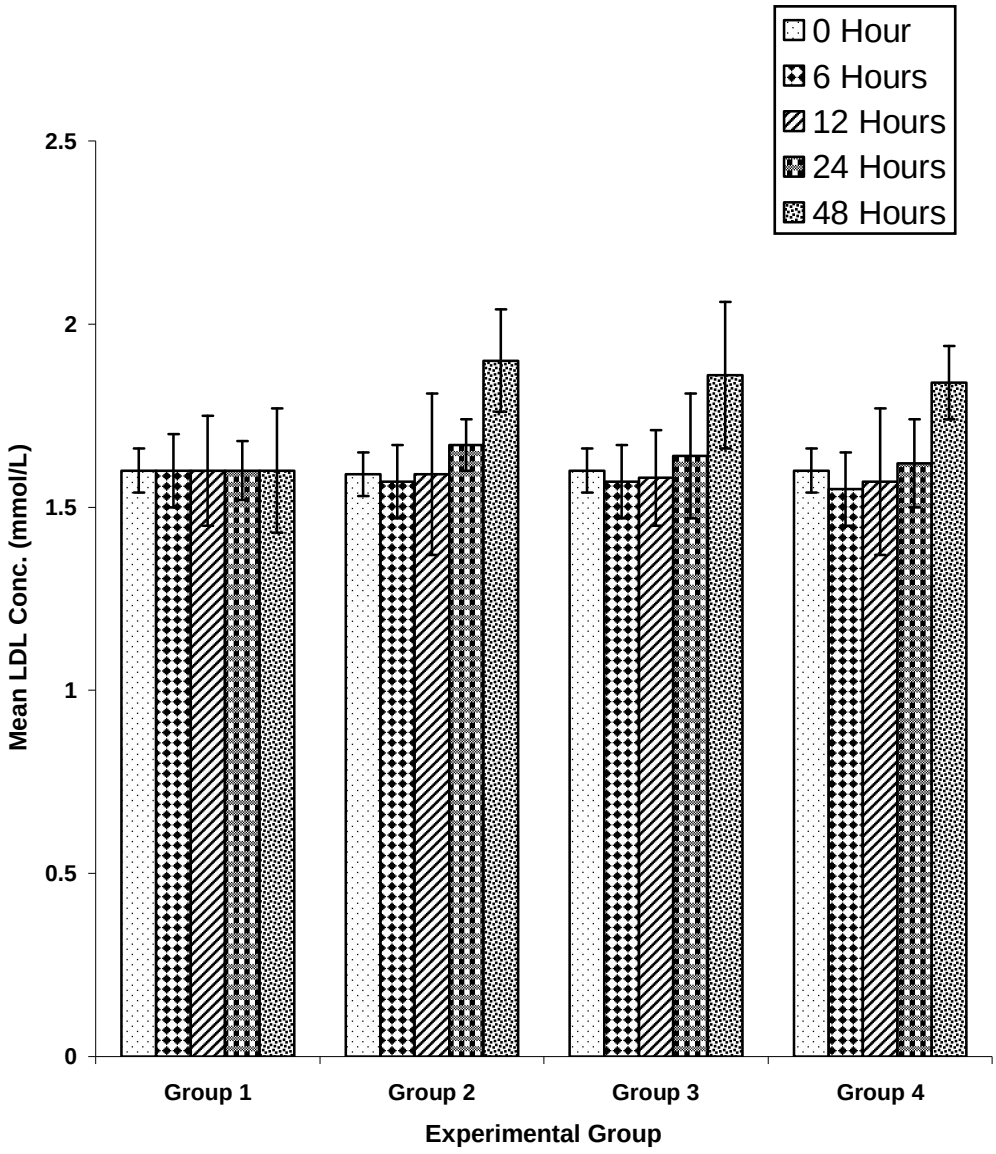

Fig. 4: Effect of starvation on the serum low density lipoprotein concentration of rats at various time intervals

Group 1: Control (Normal feed and water
Group 2: Starved of feed and water
Group 3: Starved but given water
Group 4: Starved but fed with fruit

DISCUSSION

Furthermore, the findings of this study reveal significant increase ($p<0.05$) in serum total cholesterol, low lipoprotein and triacylglycerols after starvation of different durations in rats. The increase in total serum cholesterol concentration and LDL is in agreement with previous studies of Hem Lata *et al.* (2002) and Bijlani *et al.* (1985). These results support the hypothesis that cholesterol stored in the adipose tissue cells is released into plasma and is the chief source of the hypercholesterolemia observed during starvation. Another cause of hypercholesterolemia during starvation may be attributed to the continued biosynthesis with concomitant decreased or complete absence of intestinal excretion (Swaner and Connor, 1975).

The main cellular components in the body susceptible to damage by free radicals are lipids (unsaturated fatty acids in membranes), proteins, carbohydrates and nucleic acids (Blokhina *et al.*, 2002). The interest for oxidative stress in relation to the development of disease has gained large attention during the last decade. Animals use their lipid stores to compensate for deficit of energy and loss of body weight induced by periods of food shortage (Boswell *et al.*, 2002).

The increase in LDL levels is time-dependent in this study. Between 24 and 48 hours, animals starved of feed and water, animals starved of water and animals starved of fruit increased; thus indicating that there may be an increased susceptibility of the animals to atherosclerosis as a result of starvation. The data of this work also suggest that food deprivation causes an increased lipolysis simultaneously with lowered lipogenesis during the 48 hours period.

Triacylglycerol in this work increased significantly ($p<0.05$) depending on duration . The increase in TGs concentrations may be due to the release of TGs from storage sites for the formation of glucocorticoids in response to starvation (Singhal *et al.*, 1997).

The results of HDL in this study indicate that HDL levels did not show significant difference ($p>0.05$) after starvation. Previous studies of Vaisman *et al.* (1990) and Savendhal and Underwood, 1999) have reported either no change or decrease in HDL concentrations. This may be due to different experimental conditions and organism differences in susceptibility to stress. Again, the low level groups 2-4 of HDL may exert anti-atherogenic and antioxidative effects when present in sufficient amounts and the reduced HDL concentrations in group 1 is often accompanied by elevations in plasma TG levels (Lamarche *et al.*, 1996).

In conclusion, the findings from this study support the hypothesis that: Starvation is characterized by increased oxidative damage, oxidative injury and oxidative damage to lipids; thus decreasing the availability of antioxidants.

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