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Full Length Research Paper

Brewer's yeast (*Saccharamyces cervisiae*) has hypolipidemic effect in hyperlipidemic model

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The yeast has been used for food since ancient time, brewing beer, wine and other alcoholic drinks and in baking industry to expand or to raise dough. It is relatively inexpensive food stuff that contains many kinds of beneficial nutrients [vitamins, minerals, nucleic acid, glutathione, amino acids etc]. In addition brewer's yeast is commonly known as food supplement and traditional bowel movement normalizer in Japan. The aim of this study was to evaluate the role of dietary treatment by brewer's yeast in hyperlipidemic model. This model was done by feeding rats hyperlipidemic diet containing coconut oil [10 g/kg/day], cholesterol [4 g/kg/day] and colic acid [0.2 g/kg/day] for three weeks period. Oral yeast was given at a dose of 1 gm mixed with diet and was compared with Fibrate at a dose of 50 mg/kg/day. The result revealed that hyperlipidemia induced by hyperlipidemic diet can be reduced by dietary brewer's yeast supplementation.

Key words: Yeast, hyperlipidemia, peroxisomes, TG12 gene.

INTRODUCTION

Brewer's yeast (*Saccharamyces cerevisiae*) has an extensive history of use in the area of food processing (Broach and John, 1991). It has been used for centuries to ferment the sugars of rice, wheat, barley and corn to produce alcoholic beverages and in baking industry to expand or to raise dough (Bekatorov et al., 2006). The yeast function in baking is to ferment sugars present in the flour or added to the dough. This fermentation gives off carbon dioxide ($CO_{2)}$ and ethanol .The CO_2 is trapped within tiny bubbles and result in the dough expanding or raising. With prolonged history of industrial application, this yeast has been either the subject or the model for various studies in the principle of microbiology. Currently *S. cerevisiae* is the subject of major international effort to characterize eukaryotic genome (Anderson, 1992). Many human proteins were first discovered by studying their homology in the yeast as cell cycle protein, signaling protein and protein processing enzyme (Jacob and Henel, 1990). It is widely used for the production of macro-molecular cellular components such as lipids and proteins including enzymes and vitamins (Bigelis and Russell, 1985). It is useful in studying the cell cycle because because it is easy to culture and it shares the complex internal cell structure of plants and animals (Anderson, 1992). It is the first eukaryotic genome that was completely sequenced (Jacob and Henel, 1990). Hyperlipidemia is a serious problem that results in several complication including Atherosclerosis (AS) and coronary artery disease (CAD) The plaque in the arterial wall of Atherosclerotic patient. contains large amount of cholesterol (CH). The higher the LDL-CH, the greater the risk of AS and CHD; and conversely the higher the HDL-CH the lower the risk (Anderson et al., 1987). Several groups of hypolipidemic drugs are currently available for treating hyperlipidemia; however, their toxic side effect and the decrease in response on prolonged use are problematic. Hyperlipidemia occurs more rapidly and experiments take place over weeks, one week for lipid measurement. Atherosclerosis occurs over period of 3 to 6 months. Athermanous lesions are visible and can be measured. The area covered is known to be directly related to circulating lipid levels. Aorta can be removed and fat with connective tissue cleaned for examination for evidence of pathological changes. Atheromatous plaques can be measured planimetrically using camera lucida and the area covered expressed as a percentage (Stiskovic and White, 1991).

MATERIAL AND METHOD

The experiment involved 24 male Sprague-dawley rats. All animals weighed between (186 to 263 g). The animals were housed in cages and divided into four groups [n=6]. 6 rats in group 1 were fed

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Table 1. The effect of the yeast on average weight gained during 21 days and lipid profiles in hyperlipidemic diet fed rats.

Parameter	Group 1 (ND)	Group 2 (HD)	Group 3 (HD+F)	Group 4 (HD+Y)
Weight gained in 21 days (g)	5.8 ±0.04	34.3± 3.01	17.8± 0.9	20.1± 1.5
CH	58± 5.08	184 ±12	115 ±7.5	86± 4.9
TG	59.6 ±4.3	266±24.7	70± 7	88 ±8.4
HDL	19 ±1.38	30± 2.86	33± 3.1	30± 1.5
LDL	27±2.6	100.6 ±9.9	68 ±3.9	38.4 ±3.4
VLDL	12 ±0.85	53 ±4.9	14± 1.05	17.6 ±1.01

Group 1 (ND) Normal diet; Group 2 (HD) - hyperlipidemic diet; Group 3 (HD + F) - hyperlipidemic diet + fibrate (50 kg/mg); Group 4 (HD + Y) - hyperlipidemic diet + yeast (1 g/diet).

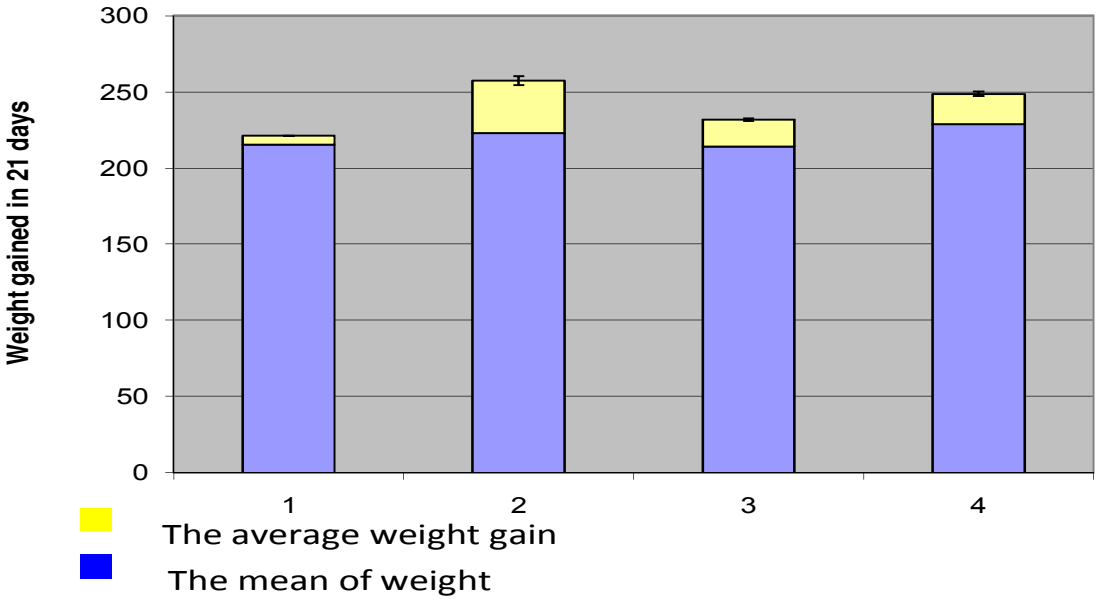

Figure 1. Changes in the body weight in normal, untreated and treated hyperlipidemic rats during 21 days.

a normal diet [laboratory pellet diet] obtained from the National Company of Animal Feeds-Benghazi-Libya for a period of 21 days and were used as control. 6 rats in group 2 were fed hyperlipidemic diet for 21 days. The diet contained coconut oil at dose of 10 g/kg/day, cholestrol at dose of 4 g/kg/day and cholic acid at dose 0.2 g/kg/day. Rats conceived high –lipid diet containing standard laboratory pellet diet supplemented with commercial coconut oil, cholesterol, and cholic acid in combination daily for 21 days. 6 rats in group 3 were fed hyperlipidemic diet and oral bezafibrat at dose 50 mg/kg/day for 21 days. 6 rats in group 4 were fed hyperlipidemic diet and oral yeast mixed with the diet at dose of 1g/diet as reported by Yang et al. (2005) for 21 days. At the end of the experiment over night fasted rats were sacrificed by decapitation using a guilatine, and blood was collected for estimation of lipid profiles cholesterol, HDL, LDL, TG and VLDL by enzymatic colorimetric test method. In this experiment we followed the experimental model of hyperlipidemia in rats reported by Wojcicki et al. (1983). Data were expressed by using descriptive analysis as mean ± standard error of mean (SEM) , test of significance was carried out using one way analysis of variance (ANOVA) as appropriated by Remington and Anthony (1985). The degree of significance was determined by using the Tukey. HSD, as well as Tamhane test for dependent samples. A probability values less than 0.05 was considered as significant.

RESULT

A-The effect on the body weight: Changes in the body weight during 21 days in normal, untreated and treated hyperlipidemic rats are shown in Table 1 and Figure 1. It was noted from this experiment that hyperlipidemic diet alone significantly increases ($p < 0.05$) body weight gained up to 34.3 ± 3.0 g as compared to control group (5.8 ± 0.04 g), while there was no statistical significant weight gained neither in the fibrate nor in the yeast treated hyperlipidemic group (17.8 ± 0.9 g; 20.1 ± 1.5 g, respectively).

B-_The effect on lipid profile: The effects on the lipid profile are presented in Table 1 and Figure 2 (Cholesterol), Figure 3 (TG), Figure 4 (HDL), Figure 5 (LDL), Figure 6 (VLDL).

●**B-1 on serum cholesterol:** Our investigation shows that hyperlipidemic diet significantly increases (p value <

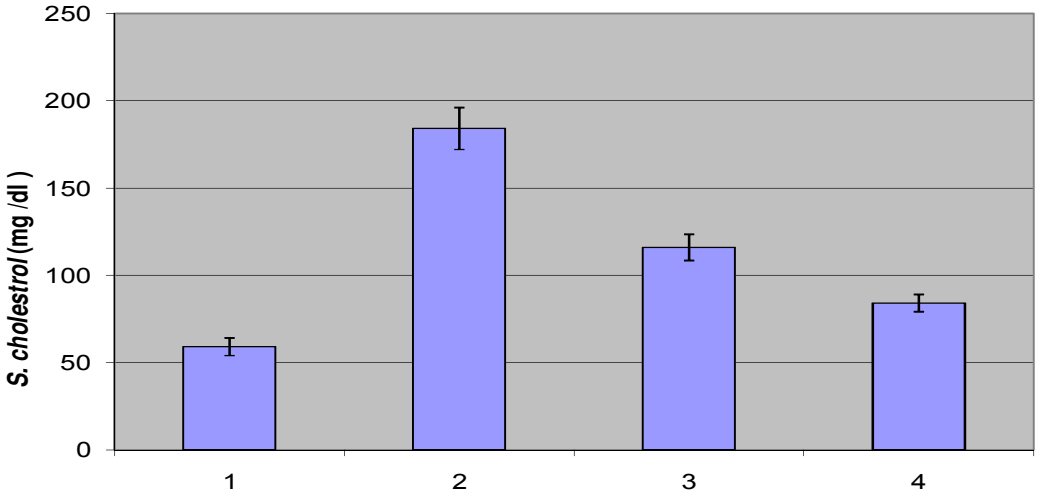

Figure 2. The effect of B- on lipid profile (Cholesterol).

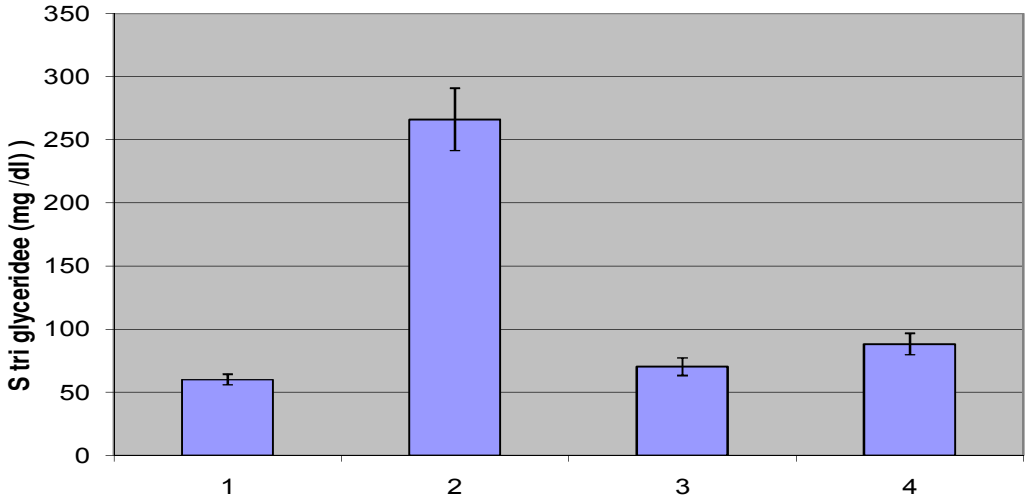

Figure 3. The effect of B- on lipid profile (TG).

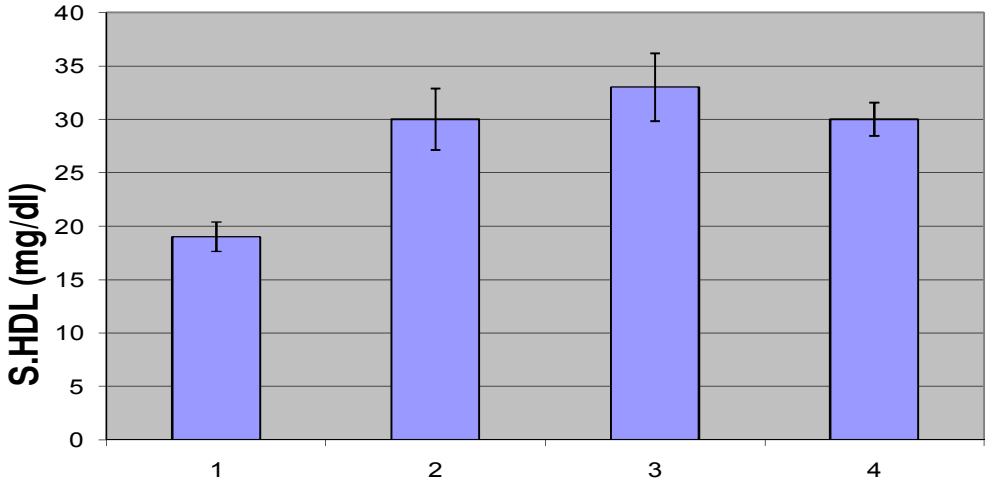

Figure 4. The effect of B- on lipid profile (HDL).

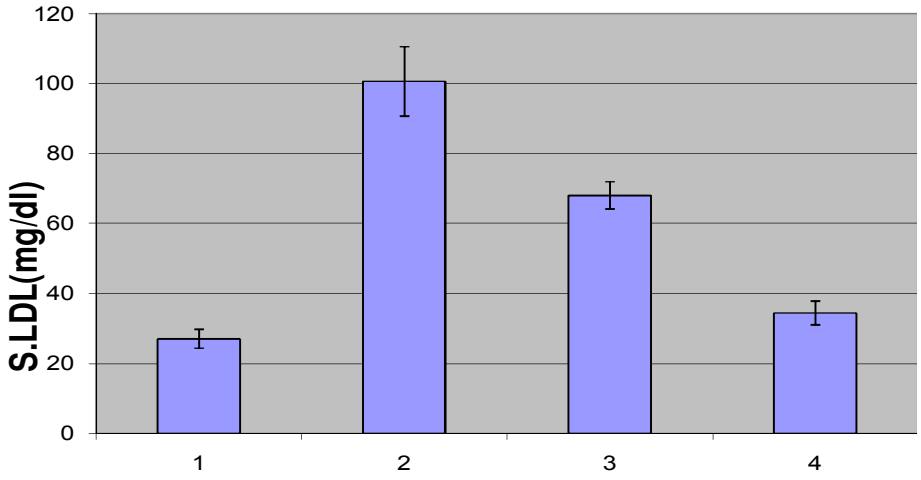

Figure 5. The effect of B- on lipid profile (LDL).

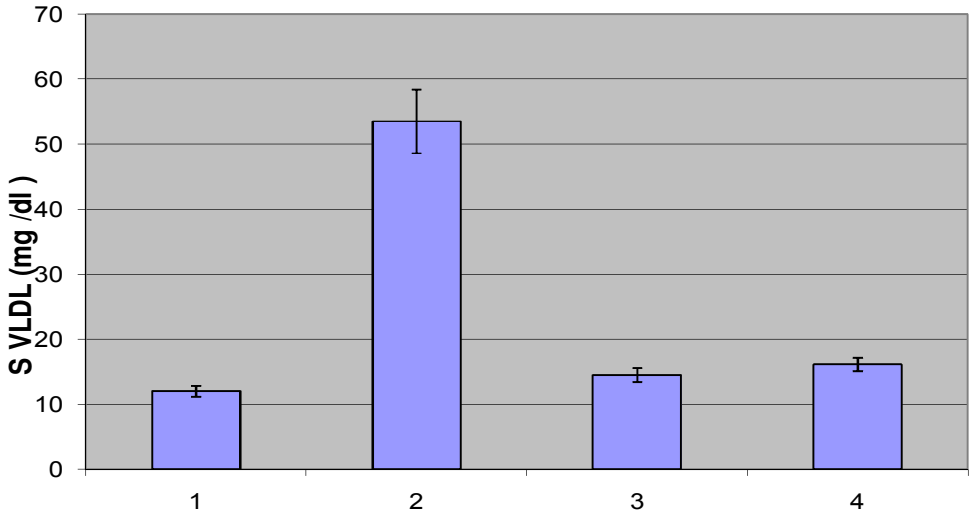

Figure 6. The effect of B- on lipid profile (VLDL).

0.001) serum cholesterol up 184 ± 12 mg/dl as compared to control group (58 ± 5.08 mg/dl), while fibrate significantly decreases serum cholesterol ($p < 0.05$) to 115 ± 7.5 mg/dl as compared to hyperlipidemic group. The yeast group significantly decreases cholesterol ($p < 0.001$) up to 86 ± 4.9 mg/dl as compared to hyperlipidemic group.

●**B-2 on serum TG:** -It also shows that hyperlipidemic diet markedly increases serum TG p value < 0.001 to 266 ± 24.7 mg/dl as compared to control group (59.6 ± 4.3 mg/dl), while in fibrate group there was highly statistically significant decrease in serum TG $p < 0.001$ up to 70 ± 7 mg/dl as compared to hyperlipidemic group. The yeast group significantly decreases TG $p < 0.05$ to 88±8.4 mg/dl as compared to hyperlipidemic group.

●**B-3 on serum HDL:** - It also shows that hyperlipidemic diet led to increase in serum HDL $p < 0.05$ value to 30 ± 2.86 mg/dl as compared to control group 19±1.38 mg/dl. An interesting finding is that fibrate group has increased serum HDL to 33 ± 3.1 mg/dl as compared to hyperlipidemic group. In the yeast group no statistically significant change in HDL 30 ± 1.5 mg/dl as compared to hyperlipidemic group.

●**B-4 on serum LDL :-** Our investigation shows that hyperlipidemic diet led to increase in serum LDL p value < 0.001, up to 100.6 ± 9.9 mg/dl as compared to control group (27 ± 2.6 mg/dl), while fibrate group significantly decreases serum LDL $p < 0.05$ up to 68 ± 3.9 mg/dl as compared to hyperlipidemic group. The yeast group significantly decreases LDL $p < 0.001$ to 38.4 ± 3.4 mg/dl as compared to hyperlipidemic group.

●**B-5 on serum VLDL:** -Our investigation also shows that hyperlipidemic diet led to increase in serum VLDL p value

< 0.001 53 ± 4.9 mg/dl as compared to control group (12 ± 0.85 mg/dl), while fibrate significantly decreases serum VLDL p < 0.001 to 14 ± 1.05 mg/dl as compared to hyperlipidemic group. In the yeast group there was significant decrease in VLDL p < 0.05 to 17.6 ± 1.01 mg/dl as compared to hyperlipidemic group.

DISCUSSION

The present study was proposed to evaluate role of dietary treatment of brewer's yeast in hyperlipidemia. To study the effect, it was essential to produce model which resembles that disease. Hyperlipidemic model was done by the model of diet induced hyperlipidemia which is useful only for detection of agents interfering with absorption, degradation and excretion of cholesterol rather than cholesterol biosynthesis (Yamaguchi et al., 1993). The small intestine brush border is the major site for lipid digestion and absorption, because of the presence of powerful lipase enzyme. Dietary fat was metabolised by lipase to free fatty acids (50 -70%), monoglycerides (25%) and to di and triglycerides (5%), whereas cholesterol ester was hydrolised by esterase to cholesterol and fatty acids. In mucosal cells fatty acids were resynthesized into triglycerides which then coated with proteins and go through lymphatic to adipose tissues and muscle cells, whereas cholesterol is reesterfied again and then go through lymphatic to the liver to form bile acids and lipoprotein cholesterol (Rommelk and Bohmer, 1986).

An important finding in present study was that administration of yeast with the hyperlipidemic diet for 3 weeks caused a significant decrease in the serum lipid profiles CH, LDL, TG and VLDL. We used fibrate as lipid lowering drug compared to yeast. Fibrate acts by stimulating lipase (Strandberg and vanhanen, 2004) which led to fall in TG, VLDL, CH and LDL. An interesting finding was that fibrate induced an increase in HDL. The role of yeast in reducing hyperlipidemia can be explained in terms of these possible mechanisms - It may have a role in digestion of lipids at the level of intestinal lumen. This fits with the *in vitro* study done by Gurvitz et al. (2001). In that study, they propagated a yeast cells on turbid media containing free fatty acids and this led to formation of clear zone around cell growth. They argued this result to up-regulation of SPS19 gene which has oleate response element through induction of peroxisomes. Peroxisomes lead to degradation of trans-unsaturated fatty acids.

On the other hand, yeast may have a role in metabolism of lipids. This fits with the in vitro study done by Heusden et al. (1998). In that study they extracted TG12 gene (a protein resembling lipase) from *S. cerevisiae* which has lipolytic activity with same measured potency of lipase toward triacylglycerol and diacylglycerol with short chain fatty acids. So *S. cerevisiae* may reduce the hyperlipidemia through its lipase like action protein. Also, the role of the yeast in reducing hyperlipidemia may be explained by their role in the adipocyte cell pathway. This fits with the study done by Edens et al. (2002). In that study they extracted different fractions {fxs} from the yeast by chromatography and they concluded that novel yeast extract and its fractions affect the pathway of adipocyte metabolism. They inhibit lipolysis by 63% and when mixed with insulin they augment insulin antilipolytic activity to 81% on the other hand, yeast contains chromium and betaglycan, both chromium (Wang et al., 1989) and betaglycan (Nomura et al., 2000) potentiate fat loss and promote lean muscle tissue gain, decrease CH, and total lipids, increase HDL and increase the effectiveness of lipid lowering drugs.

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