EFFECT OF REGULAR EXERCISE ON HIGH SUCROSE DIET-INDUCED OBESITY IN RATS

Thesis submitted for fulfillment
Of master degree in physiology

BY

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2012

Acknowledgment

First thank **Allah** who let me able to accomplish this work.

I would like to express my unlimited appreciation to **Prof. DR MOHAMED S. EL-HAMMADY,** *professor of physiology, Benha Faculty of Medicine, Benha University for his valuable guidance and marvelous support not only in this work but throughout my life. It was a great honor for me to work under This kind supervision.*

My deepest sense of gratitude and heart felt thanks to **Prof. DR. ALAA EL-TALEES,** *professor and head of physiology department, Benha Faculty of Medicine, Benha University for his valuable supervision and endless help during the delivery of this work.*

My special praise is given to **DR. Abeer Showman**, *lecturer of physiology, Benha Faculty of Medicine, Benha University for her continuous advice, encouragement and support.*

I would also like to express my great appreciation to the staff members of physiology department, Benha Faculty of Medicine, Benha University for their help and support.

I would like to express my profound thanks to my family for their patience, help and support.

Finally, my thanks are extended to all who helped me to complete this work.

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(قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم)

صدق الله العظيم

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EE	: Energy expenditure
BMR	: Basal metabolic rate
TEF	: Thermal effect of food
TNF-α	: Tumor necrosis factor α
IL-6	: Interleukin 6
CCK	: cholecystokinin
ENS	: Enteric nervous system
GLP-1	: Glucagon-like peptide 1
APJ	: APelin J receptor
OXA	: Orexin A
OXB	: Orexin B
CHD	: Coronary heart disease
NIDDM	: Non-insulin dependent diabetes mellitus
BMI	: Body mass index
NPY	: Neuropeptide Y
LDL-C	: Low density lipoprotein-cholesterol
HDL-C	: High density lipoprotein-cholesterol
UDP	: Uridine diphosphate
NAFLD	: Non-alcoholic fatty liver disease
BDNF	: Brain-Derived Neurotrophic Factor
TGs	: Triglycerides
Tch	: Total cholesterol
SD	: Standard deviation
SE	: Standard error
HSU 30%	: High sucrose 30% untrained group
HSU 50%	: High sucrose 50% untrained group
HSE 30%	: High sucrose 30% exercise group
HSE 50%	: High sucrose 50% exercise group
VLDL	: Very low density lipoprotein
Ox- LDL	: Oxidized low density lipoprotein
WHO	: World health organization
LDL	: Low density lipoprotein
mcg	: Micrograms
mg	: Milligram
HS	: High sucrose

Introduction

Obesity can be defined as excessive storage of body fat to the point where it is seriously impairing a person's health. An obese person is anyone who is overweight and has a Body Mass Index (BMI) of greater than 30**.** also Obesity can be defined as imbalance between a high energy intake and low energy expenditure (EE) which appear to be driving the high prevalence of obesity in many countries **(Spruijt et al., 2011).**

Many factors can influence obesity such as genetic, individual, and environmental factors also metabolic and behavioral factors play a role in the development of obesity .The relative contributions of these factors to weight gain probably vary between individuals. Both food intake and physical activity have behavioral and metabolic components. With respect to physical activity, the behavior relates to the quantity and type of physical activity an individual chooses to engage in, whereas the metabolic factors relate to the amount of energy expended, the pattern of fuels utilized and the hormone and metabolite changes resulting from the exercise (**Lieb et al., 2009).**

A low level of physical activity can contribute to low total energy expenditure and is thought to promote positive energy balance and weight gain. A low activity level, however, could be a consequence rather than a cause of obesity which suggests that a low level of physical activity is associated with an increased risk of weight gain Thus; regular physical exercise appears to be important in helping maintain a normal body weight and body composition **(Pawloski et al., 2010).**

The beneficial effects of physical exercise include:

- Improved blood sugar control and increased insulin sensitivity (decreased insulin resistance).
- Reduced triglyceride levels and increased "good" HDL cholesterol levels.
- Lowered blood pressure.
- A reduction in abdominal fat.
- Reduced risk of heart disease **(Greenberg et al., 2006).**

Obesity is linked to a number of serious lifestyle related diseases such as Type 2 diabetes, increase risk of certain cancers, Asthma, Gallstones, heart disease, high blood pressure, fatty liver disorder, orthopedic problems, Stroke, respiratory problems and Gout (**Schaffler et al., 2006).**

Aim of the work

This study aims to examine the role of regular exercise training in correcting obesity-induced shift in metabolic spectra in high-sucrose diet fed rats. Exercise will be carried out in combination with different concentrations of sucrose to detect its effects.

Obesity

Obesity can be defined as a condition of abnormal or excessive fat accumulation (25% in male and 32% in female) in adipose tissue to the extent that health may be impaired leading to reduced life expectancy and/or increased health problems **(WHO. 2009).**

Obesity specifically refers to an excessive amount of adipose tissue while overweight refers to an increased body weight that may come from muscles, bone, adipose tissue or water **(Molarius et al., 2004).**

It is important to recognize that obesity is both a medical condition and a lifestyle disorder and both factors have to be seen within a context of individual, family and societal functioning **(Waters et al., 2011).**

Epidemiology of obesity

The global shift in diet towards increased intake of energy-dense-foods and the decreasing physical activity due to the increasingly sedentary nature of many types of work, changing ways of transportation and increasingly urbanization are some of the biggest causes of the increasing obese population in high-income countries **(Oreopoulos et al., 2010).** Furthermore, these days is also dramatically changing the situation in low and middle-income countries particularly in urban settings lead to the tendency to consume more food rich in sugar and to the insufficient access to sport and fitness facilities **(Olds et al., 2011).**

The global number of people who is suffering from overweight in adult population is approximately 1.6 billion people (age more than 15 years) and at least 400 million of that are obese. The projection of the WHO by the year 2015 is that approximately 2.3 billion adults will be overweight and more than 700 million will be obese **(Wang and Beydoun, 2010).**

Causes of obesity

Obesity appears to result from an imbalance between energy intake and energy expenditure (EE) which appear to be driving the high prevalence of obesity in many countries **(Spruijt et al., 2011).**

At an individual level a combination of excessive food energy intake and a lack of physical activity are thought to explain most cases of obesity. A limited number of cases are due primarily to genetics, medical reasons or psychiatric illness **(Wijga et al., 2010).**

At a societal level the increasing rates of obesity are felt to be due to an easily accessible and palatable diet, increased reliance on cars and mechanized manufacturing **(Montonen et al., 2011).**

(A) - Energy imbalance in the Development of Obesity.

Obesity can result from a minor energy imbalance which leads to a gradual but persistent weight gain over a considerable period **(Ludwig et al., 2011).** Positive energy balance occurs when energy intake is greater than energy expenditure and promotes weight gain .Conversely negative energy balance promotes decrease in body fat stores and weight loss **(Tamakoshi et al., 2010).**

Body weight is regulated by a series of physiological processes which have the capacity to maintain weight within a relatively narrow range. The body can exerts a stronger defense against under nutrition and weight loss than it does against weight gain **(Elbel et al., 2011).**

Also positive energy balance and weight gains are influenced by powerful societal and environmental forces which may overwhelm the physiological regulatory mechanisms that operate to keep weight stable such as increasing automation, lack of recreational facilities and opportunities, increase in food variety and availability. Moreover, the

susceptibility to these influences is affected by genetic and other biological factors such as sex, age and hormonal activities **(WHO. 2000).**

Dietary intake and physical activity are important contributing factors in the development of obesity. If calorie intake is in excess of requirement it will be stored mainly as body fat If the stored body fat is not utilized over time it will lead to overweight or obesity. Individual variations in energy intake, basal metabolic rate, spontaneous physical activity, the relative rates of carbohydrate-to-fat oxidation and the degree of insulin sensitivity seem to be closely involved in energy balance and in determining body weight in some individuals **(Ravussin. 1999).**

The various etiological factors that could lead to unhealthy weight gain are examined **(WHO. 2003)**. These factors were categorized based on strength of evidence, namely convincing, probable and possible as shown in **Table A** .

Table A: Summary of strength of factors that might promote or protect against weight gain and obesity.

Evidence	**Decreased risk**	**Increased risk**
Convincing	Regular physical activity. High dietary non-starch polysaccharide intake	High intake of energy-dense nutrient poor foods. Sedentary lifestyles.
Possible	Home and school environments that support healthy food choices for children Low glycaemic index foods	Heavy marketing of energy-dense foods and fast-food outlets. Sugar sweetened soft drinks and fruit juices. Large portion sizes. High proportion of food prepared outside the home (western countries) "Rigid restraint / periodic disinhibition" eating patterns.

Source: WHO 2003

(B)- Dietary Intake and obesity.

B1- Food consumption pattern.

Food consumption studies among different population showed that dietary energy intake varies widely. The contribution of sugar ranges from 5 to 10 percent of total calories and tends to be higher among urban population. However, it should be borne in mind that dietary intake is not the only factor contributing to obesity **(Li et al., 2010).**

B2- Macronutrient composition of the diet.

The association between energy intake and body weight relies on the ease with which excess macronutrients can be deposited as adipose tissue. The energy cost of nutrient storage is not identical for all macronutrients. The cost of fat storage from dietary fat is the lowest, followed by carbohydrate and protein **(Flatt. 2000)**. Macronutrients with a low storage capacity such as protein and carbohydrate will be oxidized when intakes exceeded requirements **(Horton et al., 2005).**

As shown in **table B** fats undermine the body's weight regulatory systems since it is very poorly regulated at the level of both consumption and oxidation. High sugar content of diet especially sucrose and fructose may have the same effects like fat **(WHO. 2000).**

Table B: Characteristics of macronutrients

	Protein	Carbohydrate		Fat
		Starch	sucrose	
Ability to bring eating to an end	High	Intermediate	Low	Low
Ability to suppress hunger	High	High	High	Low
Contribution to daily energy intake	Low	High	High	High
Energy density	Low	Low	High	High
Storage capacity in body	Low	Low	High	High
Metabolic pathway to transfer excess intake to another compartment	Yes	Yes	Yes	No
Auto regulation	Excellent	Excellent	Poor	Poor

Source: Who 2000

B3- High fat and sugar diets.

Dietary fat and sugar content is directly correlated with energy intake, produces only weak satiation in comparison with protein and carbohydrate and is thought to be processed efficiently by the body. A number of studies found that individuals on a high-fat-sucrose diets are more prone to become overweight and obese **(Popkin et al., 2005).**

B4- Energy dense foods and drinks.

Consuming too much or often high calorie foods and drinks may increase the total calories and thus result in obesity **(SIGN. 2009).** A high fat food will often be labeled as energy-dense; sugars for example table sugar, honey also contribute to energy density **(Jolly et al., 2011).**

B5- Fiber content in the diet.

A diet with adequate amounts of fiber-containing foods is usually less energy dense. Its greater bulk has a short-term satiety effect can help to prevent overeating and reduce risk of obesity **(WHO. 2003).**

B6- Food palatability.

Palatability is defined as the momentary subjective orosensory pleasantness of a food which indicates the sensory stimulation to eat. It is one of the most powerful influences in promoting calorie over consumption (positive energy balance) by increasing both the rate of eating and the sense of hunger during and between meals **(WHO. 2000).**

Foods that are energy dense are more palatable than those of lower energy density **(Drewnowski. 1998).** Sugar is associated with palatability and pleasurable mouth-feel that can induce behavior which favors overconsumption leading to obesity **(Blundell and King, 1999).**

B7- Unhealthy dietary practices.

A changing environment and increasing affluence have widened food options and changed eating habits. Supermarkets stock their shelves

with a greater selection of foods, also fast and soft drinks which are high in calories, from either fat or sugar are more accessible. All of these are unhealthy dietary practices that would result in excessive energy intake and thus overweight problems **(Ismail. 2002).**

(C)- Energy Expenditure.

Total energy expenditure has three main components namely, basal metabolic rate (BMR), thermo genesis or thermal effect of food (TEF) and physical activity **(Goodpaster et al., 2010).**

Basal metabolic rate is the energy expended by a person who is fasting and at rest in the morning under comfortable ambient conditions. BMR constitutes about 60% to 70% of the daily energy expenditure and it may vary intrinsically by ±25% between individuals of similar weight. Within each individual it is tightly controlled. Thus, the key variable of energy output is the degree of physical activity **(Cariou et al., 2011).**

In a dynamic phase, in which an individual gains weight as a result of energy intake exceeding energy expenditure over a prolonged period, BMR will increase due to the larger fat-free mass as well as to energy cost of activity imposed by the extra weight **(Diaz et al., 2009).**

Thermo genesis is the increase in basal metabolic rate in response to stimuli such as food intake, cold or heat exposure, psychological influences such as fear or stress, or the administration of drugs or hormones. The thermal effect of food accounts for approximately 10% of the total daily energy expenditure **(Goldfield et al., 2007).**

Physical activity is the most variable component of daily energy expenditure which may account for a significant number of calories in very active individuals. Sedentary adults however, exhibit a range of physical activity that still represents about 20% to 30% of the total caloric expenditure **(Gadde et al., 2007).**

(D)- Physical inactivity and sedentary lifestyle.

Modern life is becoming increasingly sedentary and has been associated with an increased risk of obesity. Most modern jobs can be carried out with less physical effort due to technical progress, urbanization, transport and availability of a large range of domestic electrical appliances resulting in substantial decline in the energy spent in these activities **(Oreopoulos et al., 2010).**

Physical inactivity or sedentary behavior as it is otherwise known can be defined as "a state when body movement is minimal and energy expenditure approximates resting metabolic rate" **(Diaz et al., 2009).**

Physical inactivity represents more than an absence of activity; it refers also to participation in physically passive behaviors such as television viewing, reading, working at computer, talking with friends on the telephone, driving a car or eating **(Ainsworth et al., 2000).**

Physical inactivity may contribute to weight gain through other means than reduction in energy expenditure. For example, there are significant relationships between inactivity and other adverse health practices, such as the consumption of less-healthy foods and an increased fat and sugars intake **(Lytle et al., 2005).**

(E)- Psychosocial Factors contributing to Obesity.

Behavior is governed by psychological aspects of human functioning and is learnt through various experiences including conditioning, reinforcements and modeling **(Franken. 2004).**

Calorie intake largely depends on behavior which is food-related and non-food related. The significance of behavioral factors in weight gain is that it can be modified more easily than genetics **(WHO. 2001).**

E1- Hunger and appetite.

Hunger is a physiological response to a need for food triggered by stimuli acting on the brain **(Liebowitz. 2005).** It can be affected by a number of factors such as the size and composition of preceding meal, habitual eating pattern, exercise and physical and mental states **(Franken. 2004).** In a normal eating pattern hunger begins after 4 to 6 hours after eating, when food has left the stomach and much of it has been absorbed. This pattern is highly influenced by psycho- physiological factors such as smell as well as environmental interactions **(French et al., 2001).**

Individuals who restrict food consumption at each meal may feel extra hungry for a few days, but then hunger diminishes for a time. However, at some point of food deprivation, hunger can be uncontrollable and lead to bouts of overeating that more than make up for the calories lost. The stomach capacity can also adapt to larger food quantities and until a normal meal size no longer feel satisfying **(Cox et al., 2003).**

Appetite also initiates eating but unlike hunger appetite is learned. Appetite intensifies hunger but an individual can experience appetite without hunger **(Franken. 2004).** A good example is the effect of seeing and smelling food after finishing a big meal, despite an already full stomach, appetite is still strong. Appetite can be affected by factors such as learned preferences, timings of meal, environmental cues and social interactions **(Blundell. 2005).**

E2- Food-related behavior.

Humans have the ability to override signals of hunger and satiety and eat whenever they wish, especially when presented with conditions that stimulate them to do so. Hence overeating is a learnt process with regards to modeling, conditioning and habituation **(WHO. 2002).**

The main behavioral factors that contribute to obesity include:

1- Excessive energy intake and diminished rate of physical activity.

2- Greater responsiveness to stimuli associated to food

3- Large bites of food and rapid eating (allow greater amount of food to be consumed before satiety signals are recognized) **(Hase et al., 2009).**

E3- Non-food-related behavior.

Non-food-related behavior can also lead to obesity. These behaviors are sedentary behaviors such as sitting or sleeping for long hours, using lifts as compared to stair walking, driving to places that are within walking distance **(French et al., 2001).**

E4- Culture.

One of the cultural influences with regards to socio-economic environment is eating out. Eating out has become popular as it is highly convenient for today's modern household. Eating out at restaurants has been found to be increasing in trend in the past 20 years **(French et al. 2001).** Moreover, food prepared away from home tends to be larger in portion, higher in fat and energy. Mass media plays a large role in eating out behavior by exposing messages that encourage food consumption which leads to the increase of food availability **(Matsumoto. 2004).**

E5- Personality factors and cognitive style.

Personality style as well as thinking patterns can help maintain behavior that leads to obesity. Feelings of hopelessness can demotivate individuals from reducing and maintaining weight **(Byrne. 2002).**

(F)- Genetic factors.

Like many other medical conditions, obesity is the result of interplay between genetic and environmental factors. Polymorphisms in various genes controlling appetite and metabolism predispose to obesity when sufficient food energy presents **(Hamdy et al., 2005).**

7% of people with early-onset severe obesity (onset before 10 years of age and body mass index over three standard deviations above normal), harbor a single point DNA mutation **(Abbasi et al., 2010).**

(F)- Environmental factors.

Since genetic factors account for only a third of the variance in body weight **(Abbasi et al., 2010);** Environmental influences must therefore account for the balance. Several environmental factors, involving both energy intake and energy output, contribute to obesity. Environmental influences on overweight and obesity are primarily related to food intake and physical activity behaviors. Most evidence suggests that the main reason for the rising prevalence is a combination of changes in eating patterns and less active lifestyles **(Ketterer et al., 2011).**

(G)- Infectious agents.

The study of the effect of infectious agents on metabolism is still in its early stages. Gut flora has been shown to differ between lean and obese humans **(Reinehr et al., 2009).** There is an indication that gut flora in obese and lean individuals can affect the metabolic potential (**Cummings et al., 2000).**

(H)- Other possible contributors to the recent increase of obesity:

1- Insufficient sleep
2- Endocrine disruptors (environmental pollutants that interfere with lipid metabolism)
3- Decreased variability in ambient temperature
4- Decreased rates of smoking because smoking suppresses appetite
5- Increased use of medications that can cause weight gain (e.g., atypical antipsychotics)
6- Proportional increases in ethnic and age groups that tend to be heavier
7- pregnancy at a later age (susceptibility to obesity in children)
8- Epigenetic risk factors passed on generations **(Lieb W et al., 2009).**

Pathophysiology of obesity

The role of adipokines and gastrointestinal peptides in obesity.

1. Dysregulation of adipokines in obesity.

In obesity the size and number of adipocytes are increased and this is accompanied by changes in the gene expression profile in large adipocytes **(Bluher et al., 2002).** Adipose tissue infiltration with macrophages, secretion of adipokines regulating food intake and insulin sensitivity all are altered in obesity **(Otto and Lane, 2005).**

Leptin is 167-amino acid adipokine secreted largely by adipose tissue **(Zhang et al., 2004).** Leptin production is augmented in large adipocytes **(Considine et al., 2006).** The circulating level of leptin parallels adipose tissue mass and is therefore increased in states of obesity and overfeeding. Conversely, leptin levels decrease in starvation in rodents and humans. Leptin is essential in the regulation of long-term food intake and energy expenditure **(Friedman and Halaas, 2008).**

Adiponectin is a 30-amino acid adipokine secreted exclusively from adipocytes **(Scherer et al., 2005).** Adiponectin circulates in several different isoforms with distinct biological functions. The insulin sensitizing functions are linked to the high-molecular weight isoform,

Adiponectin levels are decreased in obesity and insulin resistant states **(Weyer et al., 2001).** Low adiponectin levels have been linked to higher prevalence of diabetes, hypertension, atherosclerosis and endothelial dysfunction **(Chow et al., 2007).**

In addition to leptin and adiponectin variations in levels of adipose tissue-derived peptides including resistin, retinol-binding protein-4, visfatin, angiotensin II, acylation-stimulating protein, TNF-α and IL-6 have been observed in obesity **(Rasouli and Kern, 2008).**

2. Role of gastrointestinal peptides in obesity.

The gastrointestinal tract and pancreas secrete multiple peptides and hormones that regulate food intake, glucose metabolism, GI motility and secretion. These compounds signal to the brain and may be essential in the regulation of food intake **(Strader and Woods, 2005)**.

Presence of food in the bowel activates entero- endocrine cells leading to secretion of various GI peptides such as insulin, glucagon, cholecystokinin (CCK), glucagon-like peptide 1, peptide YY, pancreatic polypeptide, gastric inhibitory peptide and vasoactive intestinal peptide (**Kim et al., 2007).**

These peptides may regulate whole body energy homeostasis, gut motility and secretion by binding their receptors on enteric nervous system (ENS) neurons and secretary cells in the intestinal mucosa **(Bray. 2000).** Some of the GI peptides may also bind to their receptors on vagal afferent fibers that are widely dispersed throughout the gut and enter the brain via the blood stream **(Schwartz. 2000).**

A classical example of a GI peptide is CCK, which is secreted by the duodenal and jejunal mucosa in response to nutrients in the duodenum. CCK stimulates gallbladder contraction and bile and pancreatic secretion and inhibits gastric secretion. In addition, CCK binds to CCK1R receptors on the local vagus fibers decreasing gastric emptying and increasing satiety **(Schwartz and Moran, 2004).**

Glucagon-like peptide-1(GLP-1) is another GI peptide that belongs to the incretin hormone group and is produced by L cells in the distal small intestine and colon in response to food intake. Post-prandial GLP-1 levels have been decreased in obesity in some **(Verdich et al., 2001),** but not all studies **(Feinle et al., 2002).**

3. Role of Apelin in obesity.

Apelin is a peptide discovered from bovine stomach extracts as an endogenous ligand for the orphan receptor apelin receptor **(Tatemoto et al., 1998).** Apelin is a product of Apelin gene and translated as a 77 amino-acid prepropeptide. The prepropeptide is subsequently cleaved to form several bioactive peptides denoted by their length, including apelin-12, -13, -16, -17, -19 and -36 **(O'Shea et al., 2003).**

Apelin-13 and -36 may be the most abundant and biologically active fragments **(Kawamata et al., 2001).** Structural studies showed that APelin J receptor (APJ) has 31% structural similarity with angiotensin receptor 1 **(O'Dowd et al., 1998).** In addition, apelin-36 is degraded to apelin-13 by angiotensin-converting enzyme-related carboxypeptidase 2 **(Vickers et al., 2002).**

Both apelin and APJ expression have been localized in the hypothalamus in the anterior pituitary and around the supraoptic and paraventricular nuclei suggesting involvement in hormone release and regulation of food and water intake **(Reaux et al., 2001).**

Apelin may also modulate glucose homeostasis and improve insulin sensitivity in animals. Intra peritoneal administration of apelin-13 decreases insulin levels and improves glucose tolerance in lean and obese rats **(Higuchi et al. 2007).** Intravenous administration of apelin enhances glucose uptake in skeletal muscle and lowers glucose levels in mice **(Dray et al., 2008).** Instead, apelin-36 inhibits glucose stimulated insulin secretion in mice **(Sorhede Winzell et al., 2005).**

In the gut apelin-13 and -36 stimulate gastric cell proliferation. Apelin-12, -13 and -19 induce CCK-release from murine enter endocrine STC-1 cells **(Wang et al., 2004)**.

4. Role of Orexins in obesity.

Orexins are hypothalamic peptides with homology to GI peptide secretin. Orexin A increases food intake in rats **(Sakurai et al., 1998).** Orexin A (OXA; hypocretin 1) and orexin B (OXB; hypocretin 2) are 33- and 28-amino acid peptides**.** The actions of OXA and OXB are mediated via binding to closely related OX1 and OX2 receptors belonging to the family of G-protein coupled receptors **(Sakurai et al., 1998).** OXA selectively binds OX1, while OXB binds both OX1 and OX2 with slightly lower affinities. OXA has been more active in the stimulation of food intake in rats, while functions of OXB are generally less well characterized **(Haynes et al., 1999).**

Orexins highly expressed in rat hypothalamic areas known to regulate food intake, the sleep-wake cycle and neuroendocrine functions. **(Taheri et al., 1999).** In rats, both orexin receptors are abundantly expressed in hypothalamic areas, including the paraventricular nucleus, the locus coeruleus and the dorsal raphe nucleus **(Backberg et al., 2002).**

OXA has been detected in various peripheral tissues including stomach, duodenum, pancreas, adipose tissue, spleen, testis and ovaries **(Heinonen et al., 2008).** Both orexins and orexin receptors have been located in both myenteric and submucosal plexuses in the mouse, rat, guinea pig and human .OX1 is expressed in enteric neurons, while OX2 expression has been localized to endocrine cells **(Naslund et al., 2002).**

OXA neurons in ENS are activated upon fasting **(Kirchgessner and Liu, 1999).** Consistently, hypoglycemia stimulates release of OXA from pancreatic islets (**Ouedraogo et al. 2003)** where orexin was found to be co stored with insulin in secretory granules in pancreatic β-cells. Interestingly, orexins has been shown to modulate glucose homeostasis by affecting both insulin and glucagon release **(Nowak et al., 2005).**

5. Role of Ghrelin in obesity.

Ghrelin is an acylated 28-amino acid peptide that was isolated from rat stomach **(Kojima et al., 1999).** Ghrelin is secreted mainly by A/X-cells in oxyntic glands in the stomach submucosa. About 70% of the ghrelin is produced by stomach and the rest is mainly produced by the small intestine **(Jeon et al., 2004).** Minor amounts of ghrelin have been detected in pancreatic islets and brain **(Hosoda et al., 2000).**

Administration of pharmacological doses of ghrelin potently increases food intake and weight gain in rodents **(Murakami et al., 2002)** and humans **(Wren et al., 2001).** Ghrelin stimulates gastric motility, gastric acid secretion and pancreatic exocrine secretion suggesting that ghrelin prepares gut for effective transport and processing of food **(Miyasaka et al., 2002).**

Ghrelin concentrations in the circulation rises prior to and falls shortly after a meal suggesting involvement in the regulation of short-term food intake **(Shiiya et al., 2002).** Circulating ghrelin concentrations are decreased in obesity and in diabetic patients **(Tschop et al., 2001).**

Ghrelin has been shown to cross the blood brain barrier by nonsaturable transmembrane diffusion and to stimulate food intake by activating orexigenic neuropeptide Y (NPY) and orexin-expressing neurons in the lateral hypothalamic area **(Toshinai et al., 2003).**

Nutrients in the meal differently regulate postprandial secretion of ghrelin. A carbohydrate-rich meal induces a greater and more rapid suppression of postprandial ghrelin levels than protein and fat **(Erdmann et al., 2003),** while the suppression after high protein meal is prolonged compared with fat and carbohydrates **(Foster-Schubert et al., 2008).**

Effects on health

Excessive obesity is associated with various diseases particularly cardiovascular diseases, diabetes mellitus type 2, obstructive sleep apnea, certain types of cancer and osteoarthritis. As a result, obesity has been found to reduce life expectancy **(Tirosh et al., 2011).**

About 61% of non-insulin dependent diabetes mellitus (NIDDM) and 17% of both coronary heart disease (CHD) and hypertension can be attributed to obesity. Indeed, as a person's body mass index (BMI) creeps up through overweight into the obese category and beyond, the risk of developing a number of chronic non-communicable diseases increases rapidly, leading to increase risk of premature death **(WHO. 2011).**

There is a nine-year reduction in life expectancy amongst obese patients **(WHO. 2011).** Generalized obesity results in alterations in the blood circulation and heart function, while central/abdominal obesity restricts chest movements and alters breathing function. Fat around the abdomen is also a major contributor to the development of diabetes, hypertension, and alterations in blood lipid level **(Freeman et al., 2012).**

Table C Relative risk of health problems associated with obesity **(WHO. 2010).**

Greatly increased	Moderately increased	Slightly increased
NIDDM Gallbladder disease Dyslipidaemia Insulin resistance Breathlessness Sleep apnea	CHD Hypertension Osteoarthritis (knees) Hyperuricaemia and gout	Certain cancers Reproductive hormone abnormalities Polycystic ovary Syndrome Impaired fertility Low back pain due to obesity Increased anesthetic risk Fetal defects arising from maternal obesity

Obesity triggers a state of insulin resistance that drives a host of metabolic disturbances known as the metabolic syndrome and includes hypertension, hypercholesterolemia, hypertriglyceridaemia, hyper coagulation, hyper viscosity and hyperuricaemia. Each in itself is a risk factor for coronary artery disease, but together they are catastrophic, the so-called syndrome X or metabolic syndrome **(Ludwig et al., 2011).**

Around 14% of cancer deaths in men and 20% in women are attributed to obesity. Obesity is associated with breast, endometrial, esophageal and colonic cancers. Obesity is "far and away the most important avoidable cause of cancer in non-smokers **(Boggs et al., 2011).**

Osteoarthritis a joint disorder which typically affects the joints in knees, hips and lower back is exacerbated by obesity which appears to increase the risk of osteoarthritis by placing extra pressure on these joints and wearing away the protective cartilage **(Losina et al., 2011).**

Psychological damage caused by overweight and obesity is a huge health burden. In childhood, the first problems caused are likely to be emotional and psychological. Moreover, the psychological consequences of obesity can range from lowered self-esteem to clinical depression. Rates of anxiety and depression are three to four times higher among obese individuals. Obese women are around 37% more likely to commit suicide than women of normal weight **(Maffeis et al., 2011).**

Diagnosis of Obesity

As it is difficult to measure body fat directly so surrogate measures such as the body mass index (BMI) are commonly used to indicate overweight and obesity in adults. Overweight is generally defined as a BMI greater than 25 while individuals with a BMI greater than 30 are classified as obese **(Montonen et al., 2010).**

Additional tools are available for identification of individuals with increased health risks due to central fat distribution as evaluation of fat distribution via the waist–hip ratio **(Ogden et al., 2012).**

Measurement of General Obesity

The body mass index (BMI) also called Quetelet Index provides the most useful and practical indicator of overweight and obesity in adults. It is calculated by dividing bodyweight in kilograms by height in meters squared (BMI = weight kg/height m2) **(Flegal et al., 2012).**

In the new graded classification system developed by the World Health Organization (WHO) a BMI of 30 kg/m2 or above denotes obesity (**WHO. 2010).Table D** Classification of overweight and obesity in adults according to BMI.

Classification	**BMI (kg/m2)**	**Risk of co-morbidities**
Underweight	<18.5	Low
Normal range	18.5—24.9	Average
Overweight	≥25	Mildly increased
Pre-obese	25.0—29.9	
Obese class I	30.0—34.9	Moderate
Obese class II	35—39.9	Severe
Obese class III	≥40	Very severe

Source: WHO 2010

Caution is required when interpreting BMI measurements in certain individuals and ethnic groups. The relationship between BMI and body fat content varies according to body build and body proportion, and a given BMI may not correspond to the same degree of fatness across all populations (**Montonen et al., 2010).** Recently, a meta-analysis among different ethnic groups showed that for the same level of body fat, age and gender, American blacks have a 1.3 kg/m2 higher BMI and Polynesians have a 4.5 kg/m2 higher BMI compared to Caucasians. By

contrast, BMIs in Chinese, Ethiopians and Indonesians were shown to be 1.9, 4.6, and 2.9 kg/m2 lower than in Caucasians (**WHO. 2010).**

Measurement of Central Obesity

This can be done by simple and convenient measures such as the waist circumference (measured at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest) using a stretch-resistant tape that provides a constant 100 g tension. Hip circumference (measured around the widest portion of the buttocks with the tape parallel to the floor) and waist-to-hip ratio **(WHO. 2008).**

For both measurements, the subject should stand with feet close together, arms at the side and body weight evenly distributed, and should wear little clothing. The subject should be relaxed, and the measurements should be taken at the end of a normal expiration. Each measurement should be repeated twice; if the measurements are within 1 cm of one another, the average should be calculated. If the difference between the two measurements exceeds 1 cm, the two measurements should be repeated **(WHO. 2008).Table E** World Health Organization cut-off points and risk of metabolic complications.

Indicator	**Cut-off points**	**Risk of metabolic complications**
Waist circumference	>94 cm (M); >80 cm (W)	Increased
Waist circumference	>102 cm (M); >88 cm (W)	Substantially increased
Waist–hip ratio	≥0.90 cm (M); ≥0.85 cm (W)	Substantially increased

M: men W: women source: **WHO 2008**

Prevention and control of obesity

1- Diet-induced weight control.

Weight loss is important in reducing the risk of metabolic syndrome and type 2 diabetes and improving glucose homeostasis,

dyslipidemia and blood pressure in these patients. Dietary interventions often lead to the recommended weight loss (5 - 10%) during 6 months, but the real challenge is long-term weight control **(Avenell et al., 2004)**.

The energy content of the diet may be decreased by modifying the proportions of nutrients. Dietary fat and sugars has high energy content and therefore low-fat-sucrose diets have been widely applied to help patients to control weight **(Astrup et al., 2000).**

High-protein diets have also been shown to enhance weight maintenance, but the participants usually drop out partly due to difficulties in maintaining the high-protein diet **(Due et al., 2004).**

2- Exercise.

Increasing physical activity has been shown to be a key element for successful long-term weight maintenance. National Weight Control Registry contains the weight loss data of 4000 adults who have lost at least -13.6 kg and kept it off at least 1 year. The results suggested that regular high intensity exercise was the most important indicator for long-term success at weight loss and adjustment **(Wing and Phelan, 2005).**

Exercise alone decreased body weight by 4.0 kg and the change was accompanied with improvements in LDL and HDL profile. When exercise program was combined with dietary prescription, weight loss of 7.2 kg was achieved **(Wood et al., 2008).**

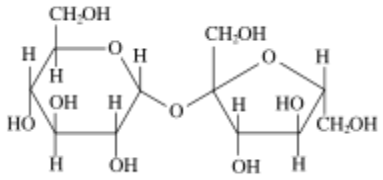
In addition, benefits of physical activity on glucose metabolism have been observed independently of decrease in body fat. It has been shown that exercise increases insulin-stimulated glycogen synthesis and glucose transport leading to improved plasma glucose concentrations **(Perseghin et al., 2006).** In addition, elevated capillary proliferation and increased muscle mass leads to improved insulin sensitivity **(Goodyear and Kahn, 2000).**

Sucrose and obesity

Sucrose or commonly known as table sugar is a disaccharide organic compound composed of glucose and fructose . Because of its disaccharide nature sucrose is the largest of the sugars and must actually be split in half or metabolized before it contributes to the energy systems of the body **(Teff et al., 2009).** Sucrose is the sweetest tasting of all the sugars so it is the main ingredient in most junk foods and beverages. It is best known for its role in human nutrition and is formed by plants but not by higher animals **(Vos et al., 2008).**

Structure of Sucrose

Sucrose is a disaccharide (glucose + fructose) with the molecular formula $C_{12}H_{22}O_{11}$. Its systematic name is α-D-glucopyranosyl-(1→2)-β-D-fructofuranoside **(Olsen et al., 2009).**



Sucrose is a molecule exists as a single isomer with five stereo centers and many sites that are reactive or can be reactive. In sucrose glucose and fructose are linked via an ether bond between C1 on the glucosyl subunit and C2 on the fructosyl unit. The bond is called a glycosidic linkage **(Stanhope et al., 2009).**

Glucose exists as two isomeric "pyranoses" (α & β) but only one of these forms the links to the fructose. Fructose itself exists as a mixture of "furanoses" each of which having α and β isomers but only one particular isomer links to the glucosyl unit. What is notable about sucrose is that unlike most disaccharides the glycosidic bond is formed between the reducing ends of both glucose and fructose and not between the

reducing end of one and the non reducing end of the other. This linkage inhibits further bonding to other saccharide units. Since it contains no anomeric hydroxyl groups, it is classified as a non reducing sugar **(Figlewicz et al., 2009).**

Physical and chemical properties

Pure sucrose is most often prepared as a fine, colorless, odorless crystalline powder with a pleasing sweet taste. Like other carbohydrates sucrose has hydrogen to oxygen ratio of 2:1. Sucrose melts and decomposes at 186 °C to form caramel and when combusted produces carbon, carbon dioxide and water **(Forshee et al., 2007).**

Water breaks down sucrose by hydrolysis converting sucrose into glucose and fructose; however the process is so gradual that it could sit in solution for years with negligible change. If the enzyme sucrase is added the reaction will proceed rapidly. Hydrolysis can also be accelerated with acids such as cream of tartar or lemon juice. Both weak acids and gastric acidity converts sucrose to glucose and fructose during digestion **(Fung et al., 2009).** Reacting sucrose with sulfuric acid dehydrates the sucrose and forms elemental carbon **(Grundy et al., 2004)** as demonstrated in the following equation:

$C_{12}H_{22}O_{11} + H_2SO_4$ catalyst --->12 C + 11 H_2O

Synthesis and biosynthesis of sucrose

The biosynthesis of sucrose proceeds via the precursors UDP-glucose and fructose 6-phosphate catalyzed by the enzyme sucrose-6-phosphate synthase. The energy for the reaction is gained by the cleavage of Uridine diphosphate (UDP). Sucrose is formed by plants and found naturally in many food plants and in many fruits, such as pineapple and apricot (sucrose is the main sugar) in others, such as grapes and pears, (fructose is the main sugar) **(Havel et al., 2005).**

Metabolism of sucrose

1- Digestion and Absorption.

You can't absorb sucrose so When ingested it is very rapidly hydrolyzed in the upper intestinal tract into fructose and glucose by a sucrase enzyme in the intestinal mucosa. The glucose is rapidly absorbed as such (active transport). In the intestinal wall of man, about 10% of the fructose may be converted to glucose but the remaining 90% is absorbed unmodified (facilitated diffusion). Absorption of fructose is a little slower than that of glucose but the difference generally is not physiologically significant. Overall absorption of glucose and fructose is rate-limiting, but the hydrolysis of sucrose is not **(Hoebel et al., 2006).**

About 25% of the glucose is removed by the liver and stored as glycogen or converted to triglyceride. The remainder is either oxidized as fuel or stored in peripheral tissue as glycogen. Nearly all of the fructose is taken up by the liver. In the peripheral circulation the fructose concentration rarely exceeds 15 mg/dl after oral fructose administration.
In the liver, fructose is converted to glucose and stored as glycogen or is converted to triglyceride. However, in large amounts it can cause an increase in lactic and uric acid production **(Le et al., 2006).**

2- Catabolism.

Catabolism is the part of metabolism that involves breaking molecules into smaller ones, a process that generally releases energy. You can break glucose and fructose down into smaller molecules called pyruvate; this releases a small amount of energy. You can then break pyruvate into carbon dioxide and water to release even more energy. These processes are critical to cellular function as they provide much of the energy cells need to maintain themselves **(Jurgens et al., 2005).**

3- Anabolism.

Your cells also use glucose and fructose for anabolic processes, which are parts of metabolism that involve building larger molecules from smaller ones. Also these processes generally require rather than release energy they're important because they produce useful products. One of the most important products you build from monosaccharide is glycogen which is an energy storage molecule that your liver and muscles make and use to provide for energy needs during times of fasting also excess sugar is stored as fats in adipose tissue **(Hubert et al., 2000).**

Commercial production and use

Sucrose is a natural and economical sweetener, it is the most important sugar in plants and it is generally extracted from sugar cane or sugar beet in which sugar can account for 12% to 20% of the plant's dry weight. Sucrose is obtained by extraction of these crops with hot water; concentration of the extract gives syrups from which solid sucrose can be crystallized **(Light et al., 2009).** Its uses include:

1- As a sweetener.

2- Acting as a preservative.

3- Enhancing flavor in foods such as tomato sauce.

4- Providing bulk and texture in ice-cream, custard and others.

5- Acting as a food for yeast in baking and in brewing beer.

6- Contributing to crust color and flavor and delaying staleness in cakes and biscuits.

7- Sucrose used in the plastics and cellulose industry, in rigid polyurethane foams, manuf of ink and of transparent soaps.

8- Sucrose used as starting material in the fermentative production of ethanol, butanol, glycerol, citric and levulinic acids **(Shapiro et al., 2008).**

The Adverse Effects of excess sucrose

Sucrose has some undesirable side effects when overly consumed. Eating foods that contain sucrose can lead to tooth decay, obesity, insulin resistance, elevated triglycerides, high blood pressure, hyperglycemia, yeast infections, inflammatory bowel disease and the hardening of the arteries. Consumption of sucrose in excessive amounts can cause the body to store the sucrose in the form of fat inside the organs, body and blood **(Seidell et al., 2009).**

Eating sucrose is far worse than eating fat due to its splitting to its two components (fructose + glucose) both of them has the following effects 1- Fructose elevates uric acid which decreases nitric oxide, raises angiotensin and causes your smooth muscle cells to contract thereby raising your blood pressure **(Hollister et al., 2007).**

2- Fructose tricks your body into gaining weight by fooling your metabolism; it turns off your body's appetite-control system. Fructose does not appropriately stimulate insulin which in turn does not suppress ghrelin (the "hunger hormone") and doesn't stimulate leptin (the "satiety hormone"), which together result in your eating more and developing insulin resistance **(Hollister et al., 2007).**

3- Fructose rapidly leads to weight gain and abdominal obesity ("beer belly"), decreased HDL, increased LDL, elevated triglycerides, elevated blood sugar and high blood pressure **(Hollister et al., 2007).**

4- Fructose metabolism is very similar to ethanol metabolism which has a multitude of toxic effects including non-alcoholic fatty liver disease (NAFLD) **(Hollister et al., 2007).**

5- One of the more recent findings that surprised researchers is that glucose actually accelerates fructose absorption, making the potential health risks from fructose even more profound **(Sato et al., 2010).**

Health problems associated with high sucrose diet are:

1- Tooth decay

This occurs when excess sucrose or other sugars are metabolized by oral bacteria to lactic acid. A high concentration of acid on the surface of a tooth leads to tooth demineralization **(Teff et al., 2004).**

2- Sugar depress your immune system

Your body uses white blood cells to destroy viruses and bacteria. Vitamin C was needed by the white blood cells to aid them in the process of destroying viruses and bacteria. Sugar in the form of glucose has a similar chemical structure to vitamin C. As your glucose levels increase it competes with vitamin C to enter the cell. If there is more glucose then there will be less vitamin C allowed into the cell **(White et al., 2008).**

3- The Glycemic Index and Sugar Side Effects

This index assigns a numbered rating to a food based on how that food affects blood glucose levels. The lower the glycemic index the slower the absorption of glucose into the bloodstream. A high rating means that the sugars in that food are rapidly absorbed into the bloodstream elevating blood glucose levels above normal. This stimulates the pancreas to secrete insulin. Insulin helps return your blood glucose levels back to normal **(Lindqvist et al., 2008).**This constant stimulation of the pancreas to secrete insulin is not healthy to your body because Insulin promotes fat storage and causes obesity **(Nguyen et al., 2009).**

4- Obesity

The National Health and Nutrition Examination Survey and their follow-on studies as part of a series indicate that the population in the United States has increased its proportion of energy consumption from carbohydrates and decreased its proportion from total fat while obesity has increased, this indicate that obesity may correlate better with sugar

consumption than with fat consumption and that reducing fat consumption while increasing sugar consumption actually increases the level of obesity **(Schwarz et al., 2009).**

High sugar diet increases triglycerides, cholesterol, free fatty acids, glucose, LDL and reduce good HDL in the serum through increasing feeding habits so contribute to obesity **(Faeh et al., 2005).**

5- Diabetes mellitus

Eating excess sucrose (60 - 70%) results in increase blood glucose and development of insulin resistance with destruction of insulin producing cells leading to the development of diabetes with building up of glucose in the blood stream which can cause two problems. Short term problem: cells become starved for energy because they do not have access to the glucose and long term problem: frequent glucose build-up increases the acidity of the blood, damaging many of the body's organs, including the eyes, kidneys, nerves, and/or heart **(Keim et al., 2009).**

6- Gout

A diet rich in sucrose may lead to gout as it raises the level of insulin which prevents excretion of uric acid from the body. Also fructose is converted to uric acid by the liver **(Le et al., 2009).**

7- Atherosclerotic heart disease

A role for sucrose in the pathogenesis of atherosclerotic heart disease was proposed by **Yudkin** hypothesis which was based on.

(1) Men afflicted with coronary heart disease are characterized by an unusually high consumption of sugar.

(2) Sucrose induces metabolic effects such as hyperinsulinism and elevation of the serum lipids which may contribute to development of atherosclerosis **(Fung et al., 2009).**

Physical exercise

There is convincing evidence that regular physical activity is protective against unhealthy weight gain whereas sedentary lifestyles, particularly sedentary occupations and inactive recreation such as watching television promote it **(Tataranni, et al., 2005).**

Physical exercise can be defined as any bodily movement produced by skeletal muscles that result in a substantial increase over the resting energy expenditure. It is performed for various reasons including strengthening muscles and the cardiovascular system, weight loss or maintenance and for the purpose of enjoyment **(Bouchard et al., 2004).**

Regular physical exercise boosts the immune system and helps prevent diseases such as heart disease, cardiovascular disease, Type 2 diabetes and obesity. It also improves mental health and helps prevent depression **(Ekelund et al., 2007).**

Types of physical exercise

Depending on the overall effect they have on the human body they are classified into three types:

1- Flexibility exercises such as stretching which improve the range of motion of muscles and joints **(Rainwater et al., 2000).**

2- Aerobic exercises as cycling, swimming, walking running which focus on increasing cardiovascular endurance **(Bell et al., 200١).**

3- Anaerobic exercises such as weight training and functional training which increase short-term muscle strength **(Schmitz et al., 2000).**

Component of physical exercise

It has three main components (**Ball et al., 2002);**

(I) Occupational work: - activities undertaken during the course of work.

(ii) Household work: - activities undertaken as part of day to day living.

(iii) Leisure-time physical activity: - activities undertaken in the individual's discretionary or free time. Activity is selected on the basis of personal needs and interest. It includes exercise and sports.

a. Exercise - a planned and structured subset of leisure-time physical activity that is usually undertaken for the purpose of improving or maintaining physical fitness **(Wagner et al., 2001).**

b. Sports - its definition varies around the world. It implies a form of physical activity that involves competition and also embraces general exercise and a specific occupation **(Wagner et al., 2001).**

Categories of physical exercise

1- Strength training: - is the use of resistance to muscular contraction to build the strength, anaerobic endurance and size of skeletal muscles. There are many different methods of strength training, the most common being the use of gravity or elastic/hydraulic forces to oppose muscle contraction **(Hu et al., 2003).**

2- Agility training: - is the ability to change the direction of the body in an efficient and effective manner and to achieve this you require a combination of balance, coordination, speed, reflexes, strength and endurance **(Koh-Banerjee et al., 2003).**

3- Continuous training: - is activity without rest intervals. This type of training may be of high intensity or moderate intensity with an extended duration it includes indoor and outdoor cycling, jogging, running, walking, rowing and stair climbing **(Macdonald et al., 2003).**

4- Resistance training:- any training that uses a resistance to the force of muscular contraction (termed strength training), and elastic or hydraulic resistance, which refers to a specific type of training that uses elastic tension to provide this resistance **(Droyvold et al., 2004).**

5- Interval training: - is a type of discontinuous physical training that involves a series of low to high-intensity exercise workouts interspersed with rest or relief periods, The high-intensity periods are typically at or close to anaerobic exercise while the recovery periods may involve either complete rest or activity of lower intensity **(Petersen et al., 2004).**

6- Eccentric training: - is the lowering phase of an exercise. For example in a biceps curl the action of lowering the dumbbell back down from the lift is the eccentric phase as long as the dumbbell is lowered slowly rather than letting it drop **(Molnar et al., 2000).**

Health effects of physical exercise

1- Cardiovascular system:

Exercise can reduce your risk of heart disease and stroke. Every year in the United Kingdom, around 53,000 people die from stroke and 94,000 from coronary heart disease. Inactive people have almost double the risk of dying from heart disease compared with people who are active. So doing a little more physical activity, for example walking each day can help reduce your risk of these conditions **(Berkey et al., 2003).**

Taking exercise can also help to reduce high blood pressure (hypertension). If you have high blood pressure, you're more likely to have a stroke or heart attack. Exercise can help to prevent high blood pressure and reduce it if it is already too high **(Horn et al., 2001).**

Exercise produces a beneficial "training effect" on the entire cardiovascular system. The heart's pumping muscle, the skeletal muscles (movement muscles), the blood vessels and the red blood cells all grow in size or number with exercise. These gradual changes are all geared towards transporting more blood (oxygen and nutrients) to the body and then efficiently removing waste (carbon dioxide and, importantly heat) away from the body **(Tammelin et al., 2004).**

Endurance or "aerobic" type exercises (running and swimming) increase muscle strength and stamina but the muscles do not enlarge as much as with resistance exercise. Endurance exercise causes the heart's left ventricle pumping function to increase gradually over time **(Moore et al., 2003).**

Exercise causes a growth in the number of small blood vessels (arterioles and capillaries) that supply the skeletal muscles. Regular vigorous exercise causes the body to produce more oxygen-containing red blood cells as well. This explains why regular "aerobic" endurance training greatly improves the "huffing and puffing" that can come from normal activities such as climbing stairs. With each breath, there are more blood vessels that are filled with more red blood cells pumped by a larger heart so that oxygen taken in with each breath is more quickly and efficiently delivered where it is needed **(Wells et al., 2001).**

2- Brain:

Exercise does everything from treating depression to improve memory, with the power to cure a host of problems while preventing even more. It also leads to the release of certain neurotransmitters in the brain that alleviate pain both physical and mental (**Pangrazi et al., 2003).**

Exercise exerts its effects on the brain through several mechanisms, including neurogenesis, mood enhancement and endorphin release. One of the most exciting changes that exercise causes is neurogenesis or the creation of new neurons (**Saris et al., 2003).**

The new neurons are created in the hippocampus the center of learning and memory in the brain; however the exact mechanism behind this neurogenesis is still being explored. At a cellular level, it is possible that the mild stress generated by exercise stimulates an influx of calcium, which activates transcription factors in existing hippocampus neurons.

The transcription factors initiate the expression of the Brain-Derived Neurotrophic Factor (BDNF) gene, creating BDNF proteins that act to promote neurogenesis. Thus the generation of BDNF is a protective response to stress and BDNF acts not only to generate new neurons, but also to protect existing neurons and to promote synaptic plasticity. However, BDNF's effects are more than protective; they are also reparative **(Neumark-Sztainer et al., 2003).**

Exercise alone is a potential prevention method and/or treatment for mild forms of depression. Endorphins which act as a natural pain reliever and antidepressant in the body have long been regarded as responsible for what is known as "runner's high", a euphoric feeling a person receives from intense physical exertion However, recent research indicates that serotonin may possibly play a greater role than endorphins in "runner's high". When a person exercises, levels of both circulating serotonin and endorphins are increased. These levels are known to stay elevated even several days after exercise is discontinued, possibly contributing to improvement in mood, increased self-esteem and weight management **(McMurray et al., 2002).**

3- Adipose tissue:

Taking exercise can help you to manage your weight through burning up calories and so helps to create a healthy energy balance.
You're more likely to be obese if you're inactive. Exercise alone can help you lose weight if you're overweight or obese **(Sahota et al., 2001).**

To understand how exercise can prevent obesity, it is important to understand the concept of energy expenditure versus energy intake and how the sum of these two variables can affect weight. Additionally, it should be appreciated that physical activity is an important factor for determining energy expenditure **(Simkin-Silverman et al., 2003).**

Energy expenditure (the amount of calories burned) is dictated by the metabolic demands of the body. Physical activity increases the body's metabolic demands and is therefore a primary contributor to energy expenditure. Energy intake (the amount of calories absorbed from food consumption). When the amount of calories an individual expends is equal to the amount they absorb from consuming food, they should maintain a steady weight .When an individual expends less calories than they absorb from food weight gain may occur **(Burke et al., 2003).**

Overweight and obese conditions can be prevented or treated with exercise along with a healthy diet. Activity helps to reduce body fat and increase muscle mass, thus improving your body's ability to burn calories. The beneficial effects of physical exercise in obesity include:

1- Improved blood sugar control and increased insulin sensitivity (decreased insulin resistance).

2- Reduced triglyceride levels and increased "good" HDL levels.

3- A reduction in abdominal fat **(Greenberg et al., 2006).**

4- Bone:

Weight-bearing exercise (such as walking, jogging, stair climbing, dancing or lifting weights) strengthens bone formation and helps prevent the osteoporosis or bone loss often seen in women after menopause. Combine a diet rich in calcium and vitamin D with regular weight-bearing exercise for maximum results **(Proper et al., 2003).**

Physical activity can increase bone mineral density in children and help to maintain strong bones in adolescents. It also slows down bone degeneration later in life so helps to prevent osteoporosis when your bones become brittle and more prone to break **(Sahota et al., 2001).**

5- Immune system

Moderate exercise has a beneficial effect on the human immune system. We don't know exactly how exercise increases your immunity to certain illnesses but there are several theories **(Kain et al., 2004);**

1- Physical activity may help by flushing bacteria out from the lungs (thus decreasing the chance of a cold, flu, or other airborne illness) and may flush out cancer-causing cells (carcinogens) by increasing output of wastes such as urine and sweat **(Saris et al., 2003).**

2- Exercise sends antibodies and white blood cells (the body's defense cells) through the body at a quicker rate. As these antibodies or white blood cells circulate more rapidly, they could detect illnesses earlier than they might normally. The increased rate of circulating blood may also trigger the release of hormones that "warn" immune cells of intruding bacteria or viruses **(Dennison et al., 2004).**

3- The temporary rise in body temperature may prevent bacterial growth, allowing the body to fight the infection more effectively **(Wareham et al., 2004).**

4- Exercise slows down the release of stress-related hormones. Stress increases the chance of illness **(Robinson et al., 2003).**

While exercise is beneficial, be careful not to "overdo" it. People who already exercise regularly are cautioned not to develop too vigorous a workout program in the hopes of increasing the immunity benefits. Heavy, long-term exercise (such as marathon running and intense gym training) could actually decrease the amount of white blood cells circulating through the body and increase the presence of stress-related hormones **(Pangrazi et al., 2003).**

Benefits of physical exercise

Participation in regular physical activity provides a large range of health benefits. Physical inactivity is one of most important risk factor that contributes to the burden of disease **(Neumark et al., 2003).**

1- Physical health benefits

• Reduction in risk of heart disease and better recovery from heart attacks.

• Reduced risk of stroke,high blood pressure and high cholesterol.

• Prevention and treatment of non-insulin dependent diabetes.

• Reduced risk of developing and dying from some cancers.

• Decreased body fat, prevention of obesity and weight management.

• Increased lean muscle, muscle strength and bone density.

• Improvement in sleep **(Droyvold et al., 2004).**

2- Mental health benefits

• Improved self esteem and confidence.

• Reduction in stress, anxiety and depression.

• Improved mood and sense of wellbeing.

• Improved concentration and enhance memory .

• Reduced feelings of fatigue and depression.

• Improved psychological and mental awareness **(Droyvold et al., 2004).**

The Dangers of Over-Exercising

Exercise releases endorphins which provide a sense of euphoria and lessens anxiety. Some people feel more powerful when exercising and become addicted to over-exercising (**Hardeman et al., 2000).**

People who over-exercise may have problems such as stress fractures because of too much wear and tear on the body's muscle, joints and bones. Also people that restrict their calories in their efforts to increase muscle tone and mass may losing both fat and muscle which can negatively impact their health overall **(Muto et al., 2001).**

MATERIALS AND METHODS

Work plan:

The present work was carried out on adult male albino rats and designed to demonstrate the possible effect of physical exercise on obesity induced by high sucrose diet by measuring body mass index, level of glucose and lipid profile (cholesterol, LDL, HDL and triglycerides) in the blood.

I-Animals used:

The present study was conducted on 35 adult male albino rats, 60 days of age, weighing 202.8± 5.0 g, individually housed in polypropylene cages (7 /cage) in an environmentally controlled, clean-air room with a temperature of 22 ±3◦C, 12 h light: dark cycle, a relative humidity of 60 ± 5%, without any previous preparation and with free access to food and water **(Gadek and Bugajski, 2004)**.

II-Diets and chemicals used:

1- High density lipoprotein (HDL) Kits:

From Eli Tech Diagnostica Company**.**

2-Cholesterol kits:

Spinreact Company (Spain)

3-*Triglyceride kits:*

Spinreact Company (Spain)

4-*Glucose kits:*

Spinreact Company (Spain)

5- Thiopental Na:

500 mg /12.5ml distilled water. Was given 0.1 ml/100gm IP.

6- Composition of the diets used.

@ **Standard chow diet:** In this type of diet

- The fat represented 3.73% of the total caloric requirement.

- The carbohydrates represented 43.88% carbohydrate (40.75% starch and 3.13% sucrose) of the total caloric requirement.
- The protein represented 23.54% of the total caloric requirement.
- The fibers represent 13.85% of the total caloric requirement **(Timothy et al., 2005).**

-Ingredients%: Table F: composition of standard diet.

Components	Balanced diet
Soybean	30%
Yellow corn	60.8%
Hydrogenated vegetable oil	5.2%
Minerals mix	2%
Vitamins mix	2%

(NRC., 1994).

@ **High sucrose diet:** In this type of diet

- The fat represented 6.40% of the total caloric requirement.
- The carbohydrates 49.85% (24.5% starch and 27.35% sucrose) or (4.5% starch and 47.35% sucrose) of the total caloric requirement.
- The protein represented 23.60% of the total caloric requirement.
- The fibers represent 9.15% of the total caloric requirement.
- The high-sucrose diet was obtained mixing 300 or 600 g sucrose and 30or 60 g of soy oil to 1000 g of a previously triturated standard chow. Casein was added to achieve the same protein content as the standard chow **(Diniz et al., 2006).**

III-Groups of experiments:

The rats included in this study were classified into 3 main groups.

Group I (control group):

Consisted of 7 rats, served as control group, received standard diet in which sucrose represents 3% of the total caloric requirement

for 4 weeks and kept sedentary (untrained) until the end of the experiment.

Group II (High Sucrose untrained group): **HSU group.**

Consisted of 14 rats, served as high sucrose group, animals of this group received high sucrose diet in which sucrose represents >20% of the total caloric requirement for 4 weeks (a method for induction of obesity in experimental animals) and kept sedentary (untrained) until the end of the experiment. According to the concentration of sucrose in the diet animals were divided into 2 subgroups.

***Group II a: HSU 30% group.**

Consisted of 7 rats that received 27.35 % sucrose in diet and kept sedentary (untrained) until the end of the experiment

***Group II b: HSU 50% group.**

Consisted of 7 rats that received 47.35 % sucrose in diet and kept sedentary (untrained) until the end of the experiment

Group III (exercise group): **HSE group.**

Consisted of 14 rats, serves as exercise group, animals of this group received high sucrose diet in which sucrose represents >20% of the total caloric requirement for 4 weeks and were submitted for exercise for 45 minutes /day for the last 3 weeks before taking samples**.** The animals were classified into 2 subgroups.

***Group III a: HSE 30% group.**

Consisted of 7 rats that received 27.35 % sucrose in diet and was submitted for exercise for 45 minutes /day for the last 3 weeks before taking samples.

***Group III b: HSE 50% group.**

Consisted of 7 rats that received 47.35 % sucrose in diet and was submitted for exercise for 45 minutes /day for the last 3 weeks before taking samples.

IV-Exercise Protocol:

- Our swimming protocol is well established and is considered equivalent to moderate training ***(Craig and foley, 1993).***
- Circular tanks 80cm in diameter and 90cm in height were filled to 60cm mark with 32-35c water.
- All rats of the same group swam together in the same tank.
- The rats were initially acclimated to the water by 15 minutes swim intervals. The purpose of the adaptation was to reduce the stress of the animals without promoting the physiological changes that might arise from the physical training.
- Training times then were slowly increased to 45 minutes over 3 week period ***(Jamine et al., 1993).***

V- Procedure of the experiments:

1- At the end of 4 weeks the rats were left overnight fast then were anesthetized with diethyl ether.

2- They were fixed on operating table and the blood samples were taken.

Method of blood sample collection:

A craniocaudal incision of about 2 cm is made, parallel and with slightly to the left of the sternum through the skin and pectoral muscles to expose the ribs. A blunt curved forceps is then binged between the 5^{th} and 6^{th} ribs, through the intercostals muscles. The gap is widened so that the rapidly beating heart becomes visible**,** then the blood sample were taken from the right ventricle.

Serum preparation: The blood was left until clotting. Serum was separated by centrifugation at 3000 revolution per minute (rpm) for 15 min and stored at – 20°C for biochemical analysis of fasting blood glucose, total cholesterol, triglycerides and high density lipoproteins cholesterol and triglyceride.

VI- Assessment of Obesity:

After 28 days of dietary treatments, the animals were anaesthetized (0.1ml intra peritoneal of 1% Thiopental Na) for the measurement of body length (nose-to-anus or nose-anal length). The body weight and body length were used to confirm the obesity through the obesity parameters body mass index (body weight g/ length cm2).

VII- Serum determinants:

1) Measurement of Glucose:

Principle of the procedure:

Glucose oxidase (GOD) catalyses the oxidation of glucose to gluconic acid. The formed hydrogen peroxidase (H_2O_2), is detected by a chromogenic oxygen accepter, phenol-aminophenazone in the presence of peroxidase (POD):

$$\text{B-D-Glucose} + O_2 + H_2O \xrightarrow{GOD} \text{Gluconic acid} + H_2O_2.$$

$$H_2O_2 + \text{phenol} + \text{Aminophenazone} \xrightarrow{POD} \text{Quinone} + H_2O.$$

The intensity of the color formed is proportional to the glucose concentration in the samples **(Caraway et al., 2007).**

Procedure:

1. Assay conditions:

   Wavelength: ……. 505 nm (490-550)

   Cuvette: …….. …. 1 cm light path

   Temperature:……. 37 c/15-25 c

2. RA50 Spectrophotometer was adjusted at zero with distilled water.
3. Pipette into a cuvette:

	Blank	Standard	Sample
WR (ml)	1.0	1.0	1.0
Standard (ul)	-	10	-
Sample (ul)	-	-	10

4. Mix and incubate for 10 min. at 37 c or 10-15 min. at room temperature (15-20 c).

5. Read the absorbance (A) of the samples and standard against the Blank **(Young, 2001)**.

2) Measurement of triglycerides:

*** Principle of the procedure:** Enzymatic colorimetric assay.

The series of the reaction involved in the assay system are as follow:

1-Triglycerides are hydrolyzed by lipoprotein lipase (LPL) to glycerol and fatty acids

2-Glycerol is then phosphorylated to glycerol-3-phosphate by ATP In a reaction catalyzed by glycerol kinase (GK).

3-The oxidation of glycerol-3-phosphate is catalyzed by glycerol phosphate oxidase to form di hydroxyacetone phosphate and hydrogen peroxide

3-In the presence of peroxidase, hydrogen peroxide affects the oxidative coupling of 4-chlorophenol and 4-aminoantipyrine to form a red color quinoneimine dye.

*** Procedure:**

1. Adjust the instrument to zero with distilled water.
2. Pipette into a cuvette:

	Blank	**Standard**	**Sample**
WR (ml)	1.0	1.0	1.0
Standard (UL)	--	10	--
Sample (UL)	--	--	10

3. Mix and incubate for 5 mins**.** At 37oC or 10 min at room temperature.
4. Read the absorbance (A) of the samples and standard, against the blank. The color is stable for at least 30 minutes **(Young, 2001)**.

3)Total Cholesterol determination:

*** Principle of the procedure:** Enzymatic colorimetric assay.

1. Cholesterol esters are enzymatically hydrolyzed by cholesterol esterase to cholesterol and free fatty acids.
2. Free cholesterol, including that originally present, is then oxidized by cholesterol oxidase to cholest-4.en-3-one and hydrogen peroxide.
3. The hydrogen peroxide combines with phenol and 4-aminoantipyrine in the presence of peroxidase to form a chromophore (quinoneimine dye) which may be quantitated at 500 — 550 nm. For bichromatic analyzers the blank wavelength should be set to 600 or 650 nm.

*** Procedure:** ***We have done the following steps:***

1-Adjust the instrument to zero with distilled water.

2-Pipette into a cuvette:

	Blank	Standard	Sample
WR (ml)	1.0	1.0	1.0
Standard (UL)	--	10	--
Sample (UL)	--	--	10

3-Mix and incubate for 5 mins**. :** At 37oC or 10 min at room temperature.

4. Read the absorbance (A) of the samples and standard, against the blank. The colour is stable for at least 60 minutes **(Young, 2001)**.

4) Measurement of HDL cholesterol:

*** Principle of the procedure:** Enzymatic colorimetric assay.

The chylomicrons, VLDL and LDL are precipitated by addition of phosphotugstic acid and magnesium chloride. After centrifugation the

supernatant fluid contains the HDL-c fraction, which is assayed for HDL Cholesterol with the cholesterol test kit.

1) Sample preparation:

- We added to 500µl of sample, 50 µl of precipitating reagent.
- We mix, wait for 10 minutes and centrifuged at 5000r.p.m. for 15 minutes.
- The supernatant was collected for HDL determination.

2) HDL determination:

We read against reagent blank

	Blank	Standard	Sample
Cholesterol reagent	300µl	300µl	300µl
Distilled water	15µl	-	-
Standard 50mg/dl	-	15µl	-
Supernatant	-	-	15 µl

Then we read the optical density after 5 minute incubation **(Naito, 2003).**

5) LDL determination:

The LDL Cholesterol is calculated from total cholesterol, HDL concentration and triglycerides concentration by using Friedewald formula as follow:

LDL= Total cholesterol - HDL- estimated VLDL

Estimated VLDL = triglycerides/ 5 **(Friedewald et al., 1972).**

STATISTICAL ANALYSIS OF RESULTS

The collected data of the present study were tabulated and analyzed using the student “t” test and paired "t" test according to ***Armistag, (1983)*** using computer with SPSS version 16 programme. This is summarized as follow:

1-Calculation of arithmetic mean (μ):

$$\mu = \frac{\Sigma(x)}{(n)}$$

Where

[∑ (x)] = sum of the values.
(n) = number of values in the group.

2-Calculation of the standard deviation (SD):

$$SD = \sqrt{\frac{\Sigma(X - \overline{X})^2}{n-1}}$$

Where $(X - \overline{X})^2$ = sum of squared difference between arithmetic mean and each individual result.

3-Calculation of the standard error (SE):

$$S.E. = \frac{S.D}{\sqrt{n}}$$

4-Calculation of the "t" value:

$$t = \frac{X_1 - X_2}{\sqrt{\frac{SD_1^2}{n_1} + \frac{(SD_2)^2}{n_2}}}$$

Where

X_1: mean value of the first group of observations.

X_2: mean value of the second group of observations.

SD_1^2 : The square of SD of the first group of observations.

SD_2^2 : The square of SD of the second group of observations.

N1: no. of observations in the first group.

N2: no. of observations in the second group.

5- Paired t- test:

$$\text{Paired t} = \frac{\overline{X}\ of\ the\ difference}{\sqrt{SE}}$$

Where $\overline{X}$ = mean of the difference between the two means of values

SE= standard error of mean

After we calculate the "t" value we consulted the "t" distribution table to get the "p" (probability value). P is a fraction which expresses the probability that the difference between Z means was due to change variation. Statistical significance was accepted at P value <0.05 or lower.

Result

Group 1: control group

Table (1) Body mass index (weight g/ height cm2), blood glucose level mg/dl and lipid profile (TGs, Tch., HDL.C and LDL.C mg/dl) in control group.

N	BMI (g/cm2)	*S. glucose mg/dl*	*TGs mg/dl*	*TCh mg/dl*	*HDL.C mg/dl*	*LDL.C mg/dl*
1	0.538	98	80	99	54	16.2
2	0.529	101	72	91	57	12
3	0.533	97	89	91	60	16.2
4	0.540	107	90	90	49	16.2
5	0.531	103	87	88	54	21.6
6	0.538	107	89	86	58	13
7	0.534	99	89	88	56	24.5
mean	0.534	101	85	90	55	17
SD	0.004	4.112	6.719	4.198	3.552	4.477
SE	0.001	1.554	2.539	1.587	1.343	1.692

TGs: triglycerides TCh: total cholesterol
HDL.C: HDL cholesterol LDL.C: LDL cholesterol
N: number BMI: body mass index

Body mass index in rats = weight in grams/ (length:nose to anus cm)2

Average weight of this group: 283.14 gm Average length: 22 cm

Tables (1) : **Represent the control group that was formed of 7 adult male rats. Given standard diet orally per day and remained untrained for 28 days show that:**

The mean of body mass index is 0.534 ± 0.004.The mean of blood glucose level is 101 ± 4.112. The mean of blood triglycerides level is 85 ± 6.719**.** The mean of blood cholesterol level is 90 ± 4.198**.** The mean of blood HDL level is 55 ± 3.552. The mean of blood LDL level is 17 ± 4.477.

Group 2: High sucrose untrained group (HSU group).

Subgroup a : High Sucrose 30% untrained (2-a) (HSU 30%group).

Table (2-a) Body mass index(g/cm2), blood glucose level mg/dl and lipid profile (TGs , Tch., HDL.C and LDL.C mg/dl) in high sucrose 30% untrained group.

N	BMI (g/cm2)	*S. glucose mg/dl*	*TGs mg/dl*	*TCh mg/dl*	*HDL.C mg/dl*	*LDL.C mg/dl*
1	0.699	147	120	102	40	28
2	0.708	152	124	110	44	30
3	0.716	150	122	104	38	42
4	0.712	142	128	109	42	36
5	0.708	154	120	104	44	40
6	0.703	152	128	106	36	34
7	0.705	146	126	107	36	42
mean	0.707	149	124	106	40	36
SD	0.005	4.203	3.464	2.886	3.464	5.656
SE	0.002	1.588	1.309	1.091	1.309	2.138

TGs: triglycerides TCh: total cholesterol
HDL.C: HDL cholesterol LDL.C: LDL cholesterol
N: number BMI: body mass index

Body mass index in rats = weight in grams/ (length:nose to anus cm)2

Average weight of this group: 374.42 gm Average length: 23 cm

Tables (2-a) : **Represent the high sucrose 30% untrained group that was formed of 7 adult male rats. Given high sucrose 30% diet orally per day and remained untrained for 28 days show that:**

The mean of body mass index is 0.707 ± 0.005.The mean of blood glucose level is 149 ± 4.203. The mean of blood triglycerides level is 124 ± 3.464**.** The mean of blood cholesterol level is 106 ± 2.886**.** The mean of blood HDL level is 40 ± 3.464. The mean of blood LDL level is 36 ± 5.656.

Subgroup b : High Sucrose 50% untrained (2-b) (HSU 50%group).

Table (2-b) Body mass index(g/cm2), blood glucose level mg/dl and lipid profile (TGs ,Tch., HDL.C and LDL.C mg/dl) in high sucrose 50% untrained group.

N	BMI (g/cm2)	*S. glucose mg/dl*	*TGs mg/dl*	*TCh mg/dl*	*HDL.C mg/dl*	*LDL.C mg/dl*
1	0.718	150	130	112	38	40
2	0.720	149	132	108	40	34
3	0.735	149	124	116	40	38
4	0.733	156	130	104	40	42
5	0.724	151	122	110	32	46
6	0.718	153	120	102	30	36
7	0.722	142	124	104	32	44
mean	0.724	150	126	108	36	40
SD	0.006	4.320	4.618	5.033	4.472	4.320
SE	0.002	1.632	1.745	1.902	1.690	1.632

TGs: triglycerides TCh: total cholesterol
HDL.C: HDL cholesterol LDL.C: LDL cholesterol
N: number BMI: body mass index

Body mass index in rats = weight in grams/ (length:nose to anus cm)2

Average weight of this group: 383.28 gm Average length: 23 cm

Tables (*2-b*) : **Represent the high sucrose 50% untrained group that was formed of 7 adult male rats. Given high sucrose 50% diet orally per day and remained untrained for 28 days show that:**

The mean of body mass index is 0.724 ± 0.006.The mean of blood glucose level is 150 ± 4.320. The mean of blood triglycerides level is 126 ± 4.618**.** The mean of blood cholesterol level is 108 ± 5.033**.** The mean of blood HDL level is 36 ± 4.472. The mean of blood LDL level is 40 ± 4.320.

Group 3: High sucrose exercise group (HSE group).

Subgroup a : High Sucrose 30% + Exercise (3-a) (HSE 30%group).

Table (3-a) Body mass index(g/cm2), blood glucose level mg/dl and lipid profile (TGs , Tch., HDL.C and LDL.C mg/dl) in high sucrose 30% exercise group.

N	BMI (g/cm2)	*S. glucose mg/dl*	*TGs mg/dl*	*TCh mg/dl*	*HDL.C mg/dl*	*LDL.C mg/dl*
1	0.557	110	100	90	62	23
2	0.548	112	88	96	54	16
3	0.550	106	90	92	60	20
4	0.553	104	96	99	55	24
5	0.550	106	86	94	57	11
6	0.548	95	100	99	61	14
7	0.551	102	84	95	50	18
mean	0.551	105	92	95	57	18
SD	0.003	5.567	6.633	3.366	4.320	4.527
SE	0.001	2.104	2.507	1.272	1.632	1.786

TGs: triglycerides TCh: total cholesterol
HDL.C: HDL cholesterol LDL.C: LDL cholesterol
N: number BMI: body mass index

Body mass index in rats = weight in grams/ (length:nose to anus cm)2

Average weight of this group: 291.71 gm Average length: 22 cm

Tables (3-a) : **Represent the high sucrose 30% exercise group that was formed of 7 adult male rats. Given high sucrose 30% diet orally and perform regular exercise per day for 28 days show that:**

The mean of body mass index is 0.551 $\pm$ 0.003.The mean of blood glucose level is 105 $\pm$ 5.567. The mean of blood triglycerides level is 92 $\pm$ 6.633**.** The mean of blood cholesterol level is 95 $\pm$ 3.366**.** The mean of blood HDL level is 57 $\pm$ 4.320. The mean of blood LDL level is 18 $\pm$ 1.786.

Subgroup b : High Sucrose 50% + Exercise (3-b) (HSE 50%group).

Table (3-b) Body mass index(g/cm2), blood glucose level mg/dl and lipid profile (TGs , Tch., HDL.C and LDL.C mg/dl) in high sucrose 50% exercise group.

N	BMI (g/cm2)	*S. glucose mg/dl*	*TGs mg/dl*	*TCh mg/dl*	*HDL.C mg/dl*	*LDL.C mg/dl*
1	0.565	116	90	93	60	24
2	0.576	114	99	99	56	20
3	0.578	105	93	94	56	22
4	0.557	104	88	96	55	18
5	0.582	106	86	95	50	20
6	0.565	116	95	95	60	16
7	0.580	102	100	100	62	13
mean	0.571	109	93	96	57	19
SD	0.009	6.082	5.354	2.581	4.041	3.696
SE	0.003	2.229	2.023	0.975	1.527	1.397

TGs: triglycerides TCh: total cholesterol
HDL.C: HDL cholesterol LDL.C: LDL cholesterol
N: number BMI: body mass index

Body mass index in rats = weight in grams/ (length:nose to anus cm)2

Average weight of this group: 302.71 gm Average length: 22 cm

Tables (3-b) : **Represent the high sucrose 50% exercise group that was formed of 7 adult male rats. Given high sucrose 50% diet orally and perform regular exercise per day for 28 days show that:**

The mean of body mass index is 0.571 $\pm$ 0.009.The mean of blood glucose level is 109 $\pm$ 6.082. The mean of blood triglycerides level is 93 $\pm$ 5.354**.** The mean of blood cholesterol level is 96 $\pm$ 2.581**.** The mean of blood HDL level is 57 $\pm$ 4.041. The mean of blood LDL level is 19 $\pm$ 3.696.

Table (4) & (Figure1) : Comparison between control group and high sucrose 30% untrained group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

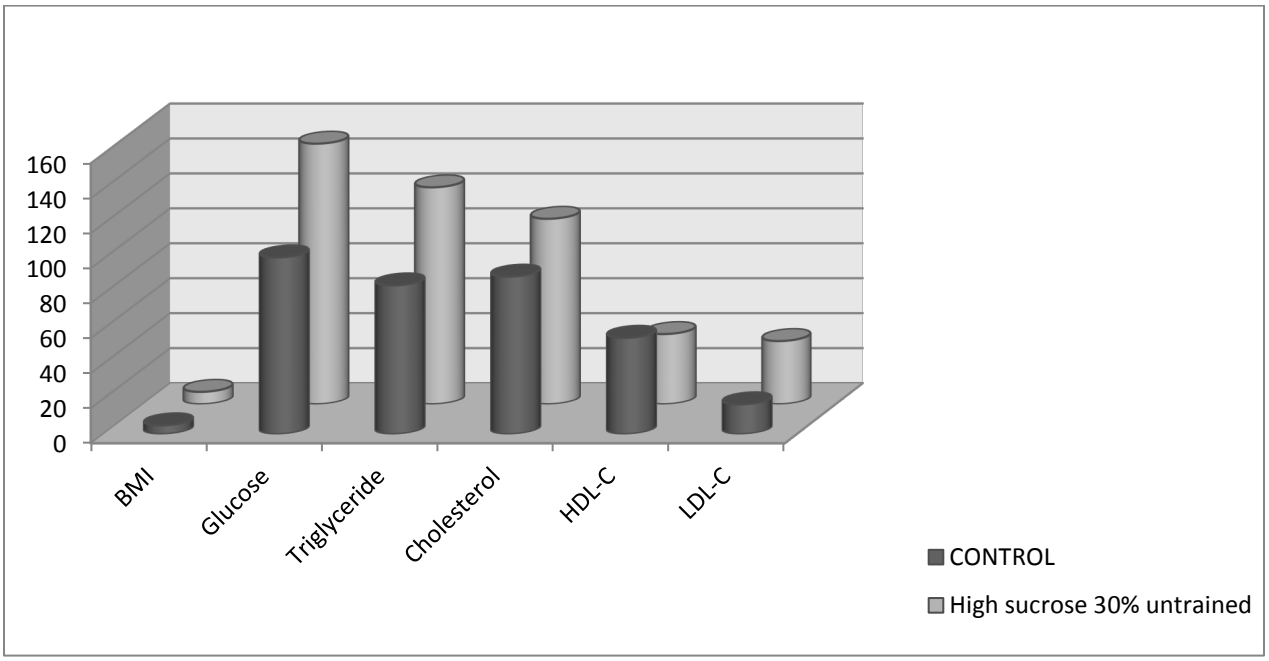
	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	Control Group	*HSU 30% group*	*Control Group*	*HSU 30% group*	*Control Group*	*HSU 30% group*	*Control Group*	*HSU 30% group*	*Control Group*	*HSU 30% group*	*Control Group*	*HSU 30% group*
Mean	**0.534**	**0.707**	**101**	**149**	**85**	**124**	**90**	**106**	**55**	**40**	**17**	**36**
SD	**0.004**	**0.005**	**4.111**	**4.203**	**6.718**	**3.464**	**4.197**	**2.886**	**3.552**	**3.464**	**4.476**	**5.656**
SE	**0.001**	**0.002**	**1.554**	**1.588**	**2.539**	**1.309**	**1.586**	**1.091**	**1.342**	**1.309**	**1.692**	**2.138**
t	**65.57**		**21.27**		**13.60**		**8.08**		**8.22**		**6.93**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

SD: Standard deviation. **t**: Student test

SE: Standard error **p**: values as compared with the corresponding value

HSU 30%: High sucrose 30% untrained group



(Figure1)

From Table (4) & (Figure1) it is clear that there was a significant increase in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant decrease in HDL-C in high sucrose 30% untrained group when compared with control group as:

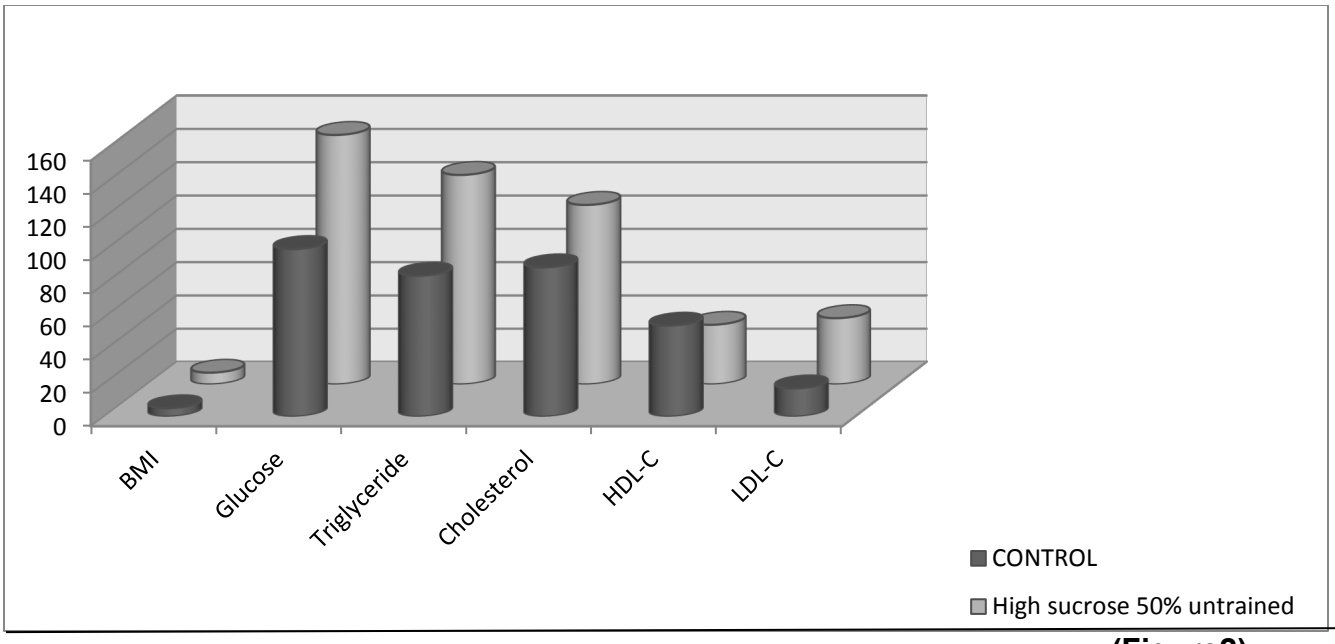
- Mean Body mass index was changed from 0.534±0.004 in control group to 0.707±0.005 in high sucrose 30% untrained group.
- Mean Serum glucose level was changed from 101±4.111 mg/dl in control group to 149±4.203 mg/dl in high sucrose 30% untrained group.
- Mean Serum triglyceride was changed from 85±6.718 mg/dl in control group to 124±3.464 mg/dl in high sucrose 30% untrained group.
- Mean Serum total cholesterol was changed from 90±4.197 mg/dl in control group to 106+2.886 mg/dl in high sucrose 30% untrained group.
- Mean Serum HDL-C was changed from 55±3.552 mg/dl in control group to 40±3.464 mg/dl in high sucrose 30% untrained group.
- Mean Serum LDL-C was changed from 17±4.476 mg/dl in control group to 36±5.656 mg/dl in high sucrose 30% untrained group.

Table (5) & (Figure2) : Comparison between control group and high sucrose 50% untrained group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	Control Group	*HSU 50% group*	*Control Group*	*HSU 50% group*	*Control Group*	*HSU 50% group*	*Control Group*	*HSU 50% group*	*Control Group*	*HSU 50% group*	*Control Group*	*HSU 50% group*
Mean	**0.534**	**0.724**	**101**	**150**	**85**	**126**	**90**	**108**	**55**	**36**	**17**	**40**
SD	**0.004**	**0.006**	**4.111**	**4.320**	**6.718**	**4.618**	**4.197**	**5.033**	**3.552**	**4.472**	**4.476**	**4.320**
SE	**0.001**	**0.002**	**1.554**	**1.632**	**2.539**	**1.745**	**1.586**	**1.902**	**1.342**	**1.690**	**1.692**	**1.632**
t	**61.98**		**21.42**		**13.25**		**7.09**		**9**		**9.73**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

HSU 50%: High sucrose 50% untrained group.



(Figure2)

From Table (5) & (Figure 2) it is clear that there was a significant increase in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant decrease in HDL-C in high sucrose 50% untrained group when compared with control group as:

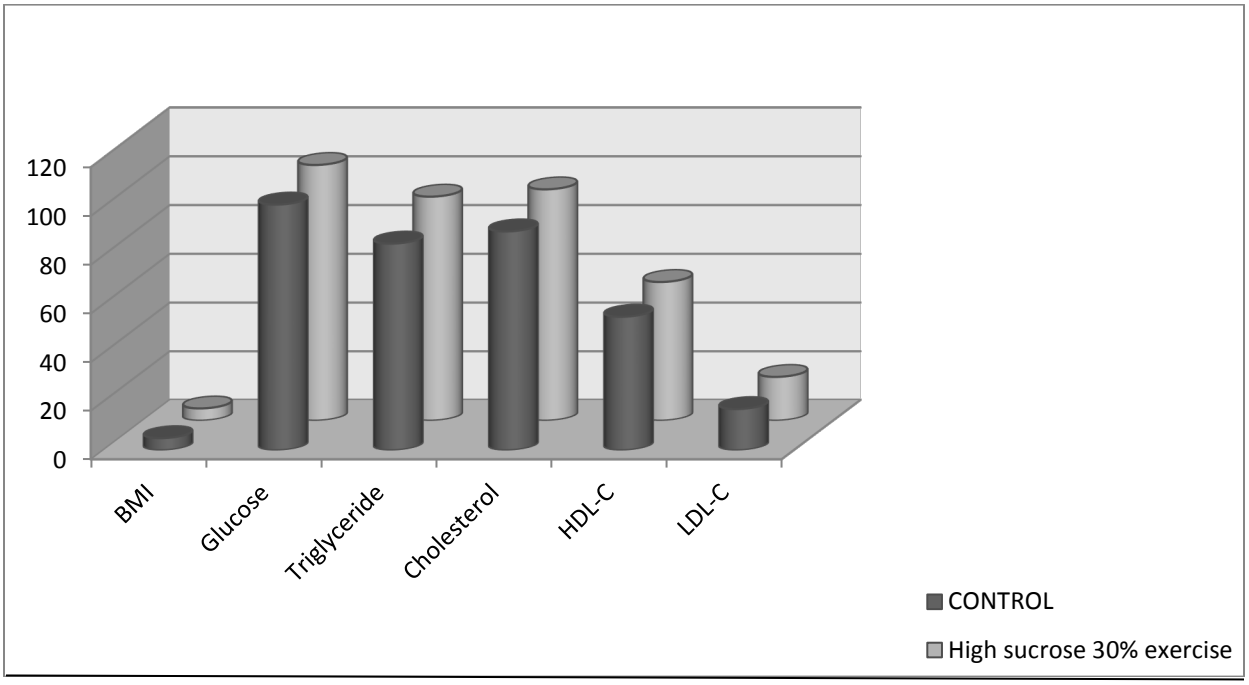
- Mean Body mass index was changed from 0.534±0.004 in control group to 0.724±0.005 in high sucrose 50% untrained group.
- Mean Serum glucose level was changed from 101±4.11 mg/dl 1 in control group to 150±4.320 mg/dl in high sucrose 50% untrained group.
- Mean Serum triglyceride was changed from 85±6.718 mg/dl in control group to 126±4.618 mg/dl in high sucrose 50% untrained group.
- Mean Serum total cholesterol was changed from 90±4.197 mg/dl in control group to 108+5.033 mg/dl in high sucrose 50% untrained group.
- Mean Serum HDL-C was changed from 55±3.552 mg/dl in control group to 36±4.472 mg/dl in high sucrose 50% untrained group.
- Mean Serum LDL-C was changed from 17±4.476 mg/dl in control group to 40±4.320 mg/dl in high sucrose 50% untrained group.

Table (6) & (Figure3) : Comparison between control group and high sucrose 30% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	Control Group	*HSE 30% group*	*Control Group*	*HSE 30% group*	*Control Group*	*HSE 30% group*	*Control Group*	*HSE 30% group*	*Control Group*	*HSE 30% group*	*Control Group*	*HSE 30% group*
Mean	**0.534**	**0.551**	**101**	**105**	**85**	**92**	**90**	**95**	**55**	**57**	**17**	**18**
SD	**0.004**	**0.003**	**4.111**	**5.567**	**6.718**	**6.633**	**4.197**	**3.366**	**3.552**	**4.320**	**4.476**	**4.725**
SE	**0.001**	**0.001**	**1.554**	**2.104**	**2.539**	**2.507**	**1.586**	**1.272**	**1.342**	**1.632**	**1.692**	**1.786**
t	**8.35**		**1.25**		**1.92**		**2.24**		**0.47**		**0.36**	
p	**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**	

** Non Significant change compared with the corresponding value (p>0.05).

HSE 30%: High sucrose 30% exercise group.



(Figure3)

From Table (6) & (Figure 3) it is clear that there was non- significant change in body mass index, serum glucose, triglyceride, total cholesterol, HDL-C and LDL-C in high sucrose 30% exercise group when compared with control group as:

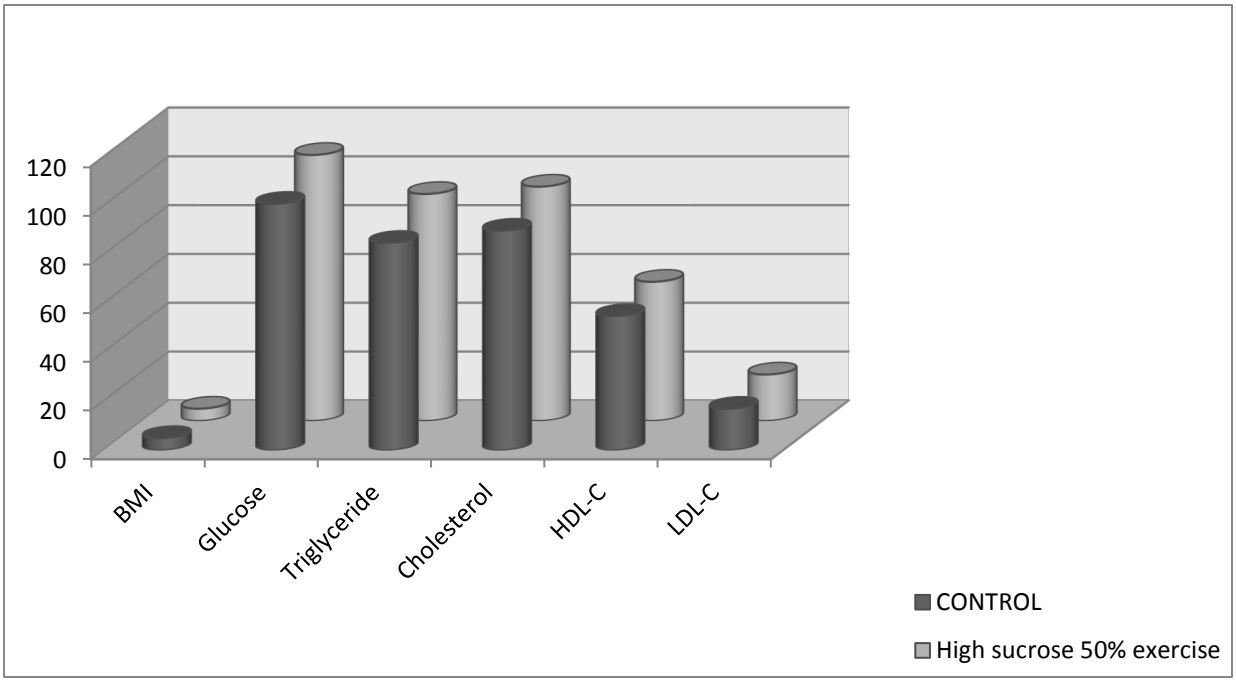
- Mean Body mass index was changed from 0.534±0.004 in control group to 0.551±0.003 in high sucrose 30% exercise group.
- Mean Serum glucose level was changed from 101±4.11 mg/dl 1 in control group to 105±5.567 mg/dl in high sucrose 30% exercise group.
- Mean Serum triglyceride was changed from 85±6.718 mg/dl in control group to 92±6.633 mg/dl in high sucrose 30% exercise group.
- Mean Serum total cholesterol was changed from 90±4.197 mg/dl in control group to 95+3.366 mg/dl in high sucrose 30% exercise group.
- Mean Serum HDL-C was changed from 55±3.552 mg/dl in control group to 57±4.320 mg/dl in high sucrose 30% exercise group.
- Mean Serum LDL-C was changed from 17±4.476 mg/dl in control group to 18±4.725 mg/dl in high sucrose 30% exercise group.

Table (7) & (Figure4) : Comparison between control group and high sucrose 50% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	Control Group	*HSE 50% group*	*Control Group*	*HSE 50% group*	*Control Group*	*HSE 50% group*	*Control Group*	*HSE 50% group*	*Control Group*	*HSE 50% group*	*Control Group*	*HSE 50% group*
Mean	**0.534**	**0.571**	**101**	**109**	**85**	**93**	**90**	**96**	**55**	**57**	**17**	**19**
SD	**0.004**	**0.009**	**4.111**	**6.082**	**6.718**	**5.354**	**4.197**	**2.581**	**3.552**	**4.041**	**4.476**	**3.696**
SE	**0.001**	**0.003**	**1.554**	**2.299**	**2.539**	**2.023**	**1.586**	**0.975**	**1.342**	**1.527**	**1.692**	**1.397**
t	**9.52**		**2.62**		**2.42**		**2.99**		**0.77**		**0.86**	
p	**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**	

** Non Significant change compared with the corresponding value (p>0.05).

HSE 50%: High sucrose 50% exercise group.



(Figure4)

From Table (7) & (Figure 4) it is clear that there was non- significant change in body mass index, serum glucose, triglyceride, total cholesterol, HDL-C and LDL-C in high sucrose 50% exercise group when compared with control group as:

- Mean Body mass index was changed from 0.534±0.004 in control group to 0.571±0.009 in high sucrose 50% exercise group.
- Mean Serum glucose level was changed from 101±4.11 mg/dl 1 in control group to 109±6.082 mg/dl in high sucrose 50% exercise group.
- Mean Serum triglyceride was changed from 85±6.718 mg/dl in control group to 93±5.354 mg/dl in high sucrose 50% exercise group.
- Mean Serum total cholesterol was changed from 90±4.197 mg/dl in control group to 96+2.581 mg/dl in high sucrose 50% exercise group.
- Mean Serum HDL-C was changed from 55±3.552 mg/dl in control group to 57±4.041 mg/dl in high sucrose 50% exercise group.
- Mean Serum LDL-C was changed from 17±4.476 mg/dl in control group to 19±3.696 mg/dl in high sucrose 50% exercise group.

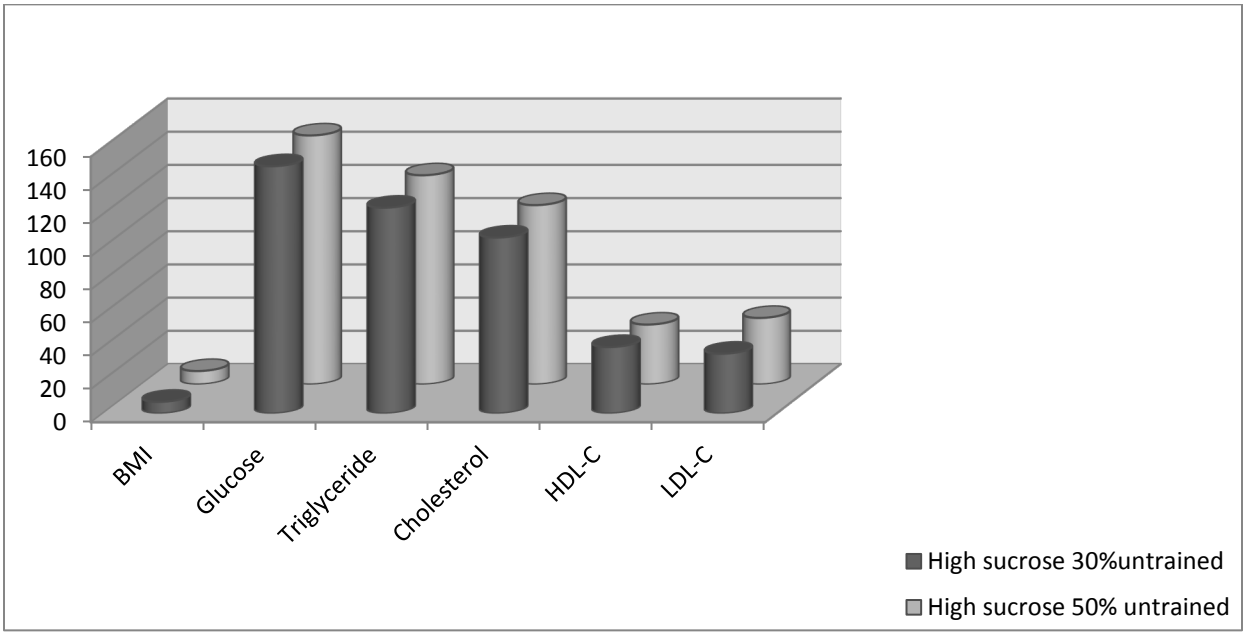
Table (8) & (Figure5) : Comparison between high sucrose 30% untrained group and high sucrose 50% untrained group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSU 30% group	*HSU 50% group*	*HSU 30% group*	*HSU 50% group*	*HSU 30% group*	*HSU 50% group*	*HSU 30% group*	*HSU 50% group*	*HSU 30% group*	*HSU 50% group*	*HSU 30% group*	*HSU 50% group*
Mean	**0.707**	**0.724**	**149**	**150**	**124**	**126**	**106**	**108**	**40**	**36**	**36**	**40**
SD	**0.005**	**0.006**	**4.203**	**4.320**	**3.464**	**4.618**	**2.886**	**5.033**	**3.464**	**4.472**	**5.656**	**4.320**
SE	**0.002**	**0.002**	**1.588**	**1.632**	**1.309**	**1.745**	**1.091**	**1.902**	**1.309**	**1.690**	**2.138**	**1.632**
t	**5**		**0.43**		**0.91**		**0.91**		**1.87**		**1.48**	
p	**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**	

** Non Significant change compared with the corresponding value (p>0.05).

HSU 30%: High sucrose 30% untrained group

HSU 50%: High sucrose 50% untrained group.



(Figure5)

From Table (8) & (Figure 5) it is clear that there was non- significant change in body mass index, serum glucose, triglyceride, total cholesterol, HDL-C and LDL-C in high sucrose 50% untrained group when compared with high sucrose 30% untrained group as:

- Mean Body mass index was changed from 0.707±0.005 in high sucrose 30% untrained group to 0.724±0.006 in high sucrose 50% untrained group.
- Mean Serum glucose level was changed from 149±4.203 mg/dl in high sucrose 30% untrained group to 150±4.320 mg/dl in high sucrose 50% untrained group.
- Mean Serum triglyceride was changed from 124±3.464 mg/dl in high sucrose 30% untrained group to 126±4.618 mg/dl in high sucrose 50% untrained group.
- Mean Serum total cholesterol was changed from 106±2.886 mg/dl in high sucrose 30% untrained group to 108+5.033 mg/dl in high sucrose 50% untrained group.
- Mean Serum HDL-C was changed from 40±3.464 mg/dl in high sucrose 30% untrained group to 36±4.472 mg/dl in high sucrose 50% untrained group.
- Mean Serum LDL-C was changed from 36±5.656 mg/dl in high sucrose 30% untrained group to 40±4.320 mg/dl in high sucrose 50% untrained group.

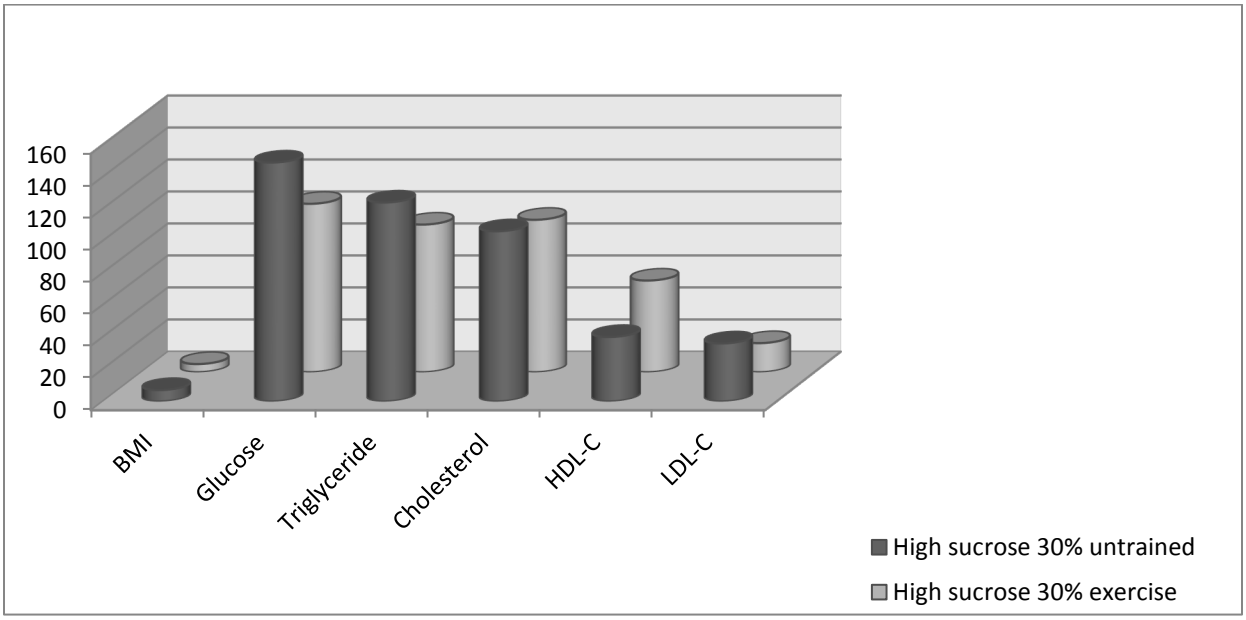
Table (9) & (Figure6) : Comparison between high sucrose 30% untrained group and high sucrose 30% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSU 30% group	*HSE 30% group*	*HSU 30% group*	*HSE 30% group*	*HSU 30% group*	*HSE 30% group*	*HSU 30% group*	*HSE 30% group*	*HSU 30% group*	*HSE 30% group*	*HSU 30% group*	*HSE 30% group*
Mean	**0.707**	**0.551**	**149**	**105**	**124**	**92**	**106**	**95**	**40**	**57**	**36**	**18**
SD	**0.005**	**0.003**	**4.203**	**5.567**	**3.464**	**6.633**	**2.886**	**3.366**	**3.464**	**4.320**	**5.656**	**4.725**
SE	**0.002**	**0.001**	**1.588**	**2.104**	**1.309**	**2.507**	**1.091**	**1.272**	**1.309**	**1.632**	**2.138**	**1.786**
t	**63.87**		**16.68**		**11.31**		**6.56**		**8.12**		**6.46**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

HSU 30%: High sucrose 30% untrained group.

HSE 30%: High sucrose 30% exercise group.



(Figure6)

From Table (9) & (Figure 6) it is clear that there was a significant decrease in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant increase in HDL-C in high sucrose 30% exercise group when compared with high sucrose 30% untrained group as:

- Mean Body mass index was changed from 0.707±0.005 in high sucrose 30% untrained group to 0.551±0.003 in high sucrose 30% exercise group.
- Mean Serum glucose level was changed from 149±4.203 mg/dl in high sucrose 30% untrained group to 105±5.567 mg/dl in high sucrose 30% exercise group.
- Mean Serum triglyceride was changed from 124±3.464 mg/dl in high sucrose 30% untrained group to 92±6.633 mg/dl in high sucrose 30% exercise group.
- Mean Serum total cholesterol was changed from 106±2.886 mg/dl in high sucrose 30% untrained group to 95+3.366 mg/dl in high sucrose 30% exercise group.
- Mean Serum HDL-C was changed from 40±3.464 mg/dl in high sucrose 30% untrained group to 57±4.320 mg/dl in high sucrose 30% exercise group.
- Mean Serum LDL-C was changed from 36±5.656 mg/dl in high sucrose 30% untrained group to 18±4.725 mg/dl in high sucrose 30% exercise group.

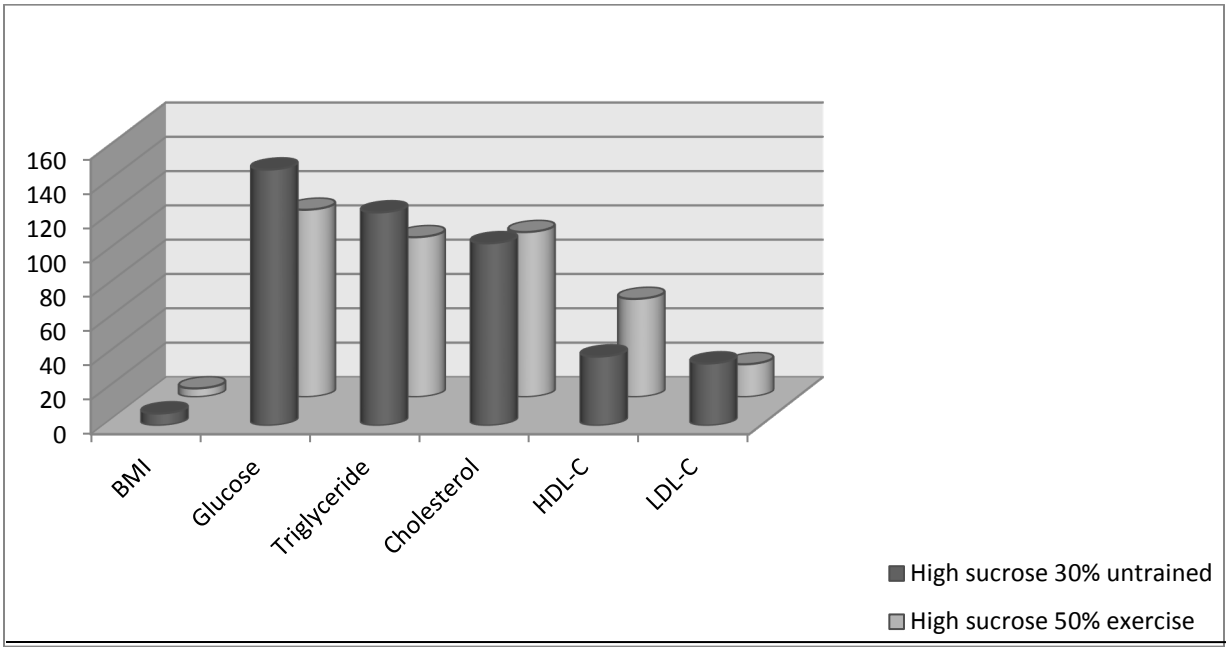
Table (10) & (Figure7) : Comparison between high sucrose 30% untrained group and high sucrose 50% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSU 30% group	*HSE 50% group*	*HSU 30% group*	*HSE 50% group*	*HSU 30% group*	*HSE 50% group*	*HSU 30% group*	*HSE 50% group*	*HSU 30% group*	*HSE 50% group*	*HSU 30% group*	*HSE 50% group*
Mean	**0.707**	**0.571**	**149**	**109**	**124**	**93**	**106**	**96**	**40**	**57**	**36**	**19**
SD	**0.005**	**0.009**	**4.203**	**6.082**	**3.464**	**5.354**	**2.886**	**2.581**	**3.464**	**4.041**	**5.656**	**3.696**
SE	**0.002**	**0.003**	**1.588**	**2.299**	**1.309**	**2.023**	**1.091**	**0.975**	**1.309**	**1.527**	**2.138**	**1.397**
t	**32.47**		**14.31**		**12.86**		**6.83**		**8.45**		**6.65**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

HSU 30%: High sucrose 30% untrained group

HSE 50%: High sucrose 50% exercise group.



(Figure7)

From Table (10) & (Figure 7) it is clear that there was a significant decrease in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant increase in HDL-C in high sucrose 50% exercise group when compared with high sucrose 30% untrained group as:

- Mean Body mass index was changed from 0.707±0.005 in high sucrose 30% untrained group to 0.571±0.009 in high sucrose 50% exercise group.
- Mean Serum glucose level was changed from 149±4.203 mg/dl in high sucrose 30% untrained group to 109±6.082 mg/dl in high sucrose 50% exercise group.
- Mean Serum triglyceride was changed from 124±3.464 mg/dl in high sucrose 30% untrained group to 93±5.354 mg/dl in high sucrose 50% exercise group.
- Mean Serum total cholesterol was changed from 106±2.886 mg/dl in high sucrose 30% untrained group to 96+2.581 mg/dl in high sucrose 50% exercise group.
- Mean Serum HDL-C was changed from 40±3.464 mg/dl in high sucrose 30% untrained group to 57±4.041 mg/dl in high sucrose 50% exercise group.
- Mean Serum LDL-C was changed from 36±5.656 mg/dl in high sucrose 30% untrained group to 19±3.696 mg/dl in high sucrose 50% exercise group.

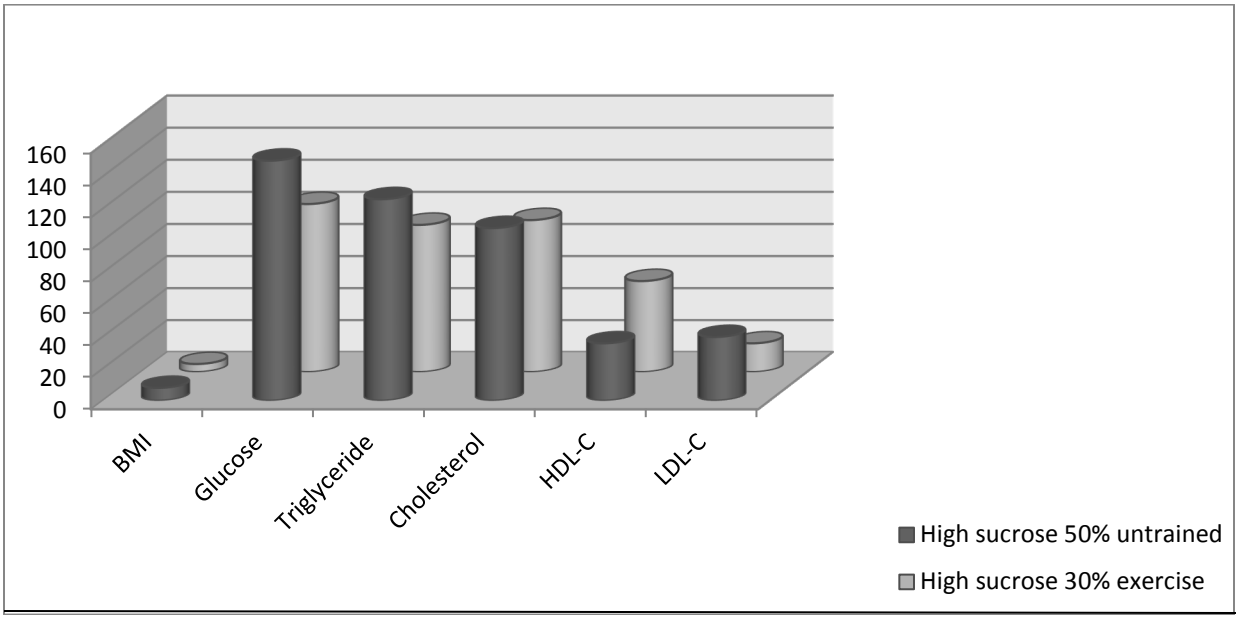
Table (11) & (Figure8) : Comparison between high sucrose 50% untrained group and high sucrose 30% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSU 50% group	*HSE 30% group*	*HSU 50% group*	*HSE 30% group*	*HSU 50% group*	*HSE 30% group*	*HSU 50% group*	*HSE 30% group*	*HSU 50% group*	*HSE 30% group*	*HSU 50% group*	*HSE 30% group*
Mean	**0.724**	**0.551**	**150**	**105**	**126**	**92**	**108**	**95**	**36**	**57**	**40**	**18**
SD	**0.006**	**0.003**	**4.320**	**5.567**	**4.618**	**6.633**	**5.033**	**3.366**	**4.472**	**4.320**	**4.320**	**4.725**
SE	**0.002**	**0.001**	**1.632**	**2.104**	**1.745**	**2.507**	**1.902**	**1.272**	**1.690**	**1.632**	**1.632**	**1.786**
t	**59.73**		**0.52**		**0.14**		**0.26**		**0.55**		**0.82**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

HSU 50%: High sucrose 50% untrained group.

HSE 30%: High sucrose 30% exercise group.



(Figure8)

From Table (11) & (Figure 8) it is clear that there was a significant decrease in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant increase in HDL-C in high sucrose 30% exercise group when compared with high sucrose 50% untrained group as:

- Mean Body mass index was changed from 0.724±0.006 in high sucrose 50% untrained group to 0.551±0.003 in high sucrose 30% exercise group.
- Mean Serum glucose level was changed from 150±4.320 mg/dl in high sucrose 50% untrained group to 105±5.567 mg/dl in high sucrose 30% exercise group.
- Mean Serum triglyceride was changed from 126±4.618 mg/dl in high sucrose 50% untrained group to 92±6.633 mg/dl in high sucrose 30% exercise group.
- Mean Serum total cholesterol was changed from 108±5.033 mg/dl in high sucrose 50% untrained group to 95+3.366mg/dl in high sucrose 30% exercise group.
- Mean Serum HDL-C was changed from 36±4.472 mg/dl in high sucrose 50% untrained group to 57±4.320 mg/dl in high sucrose 30% exercise group.
- Mean Serum LDL-C was changed from 40±4.320 mg/dl in high sucrose 50% untrained group to 18±4.725 mg/dl in high sucrose 30% exercise group.

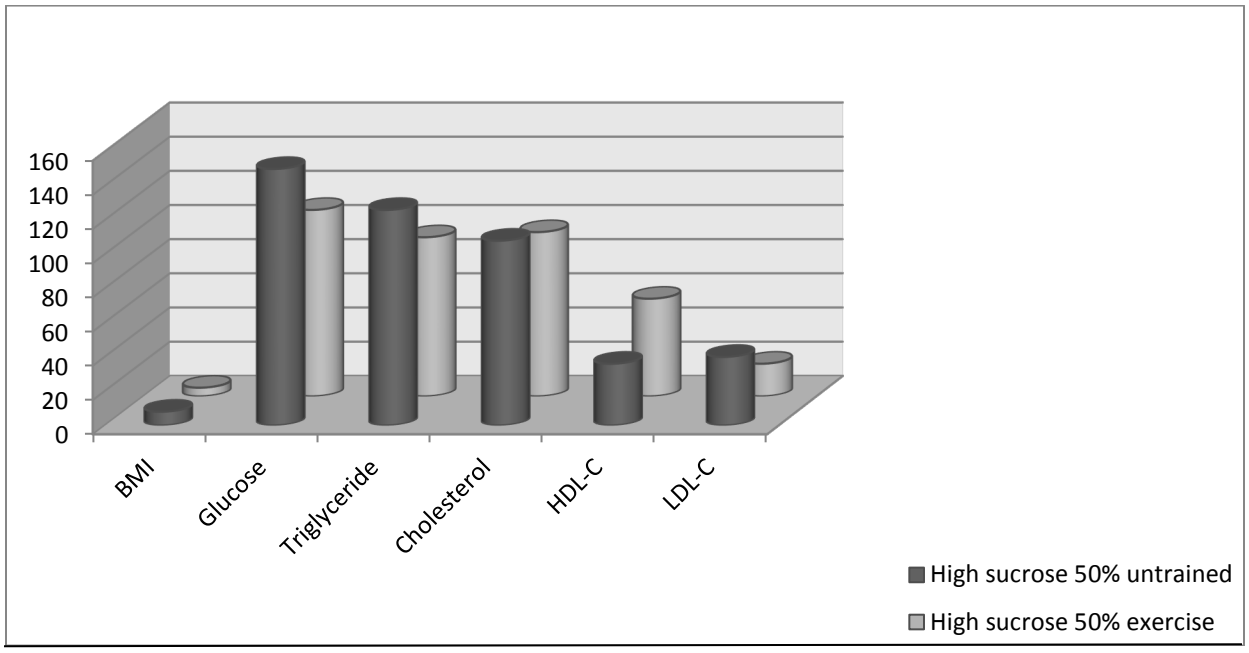
Table (12) & (Figure9) : Comparison between high sucrose 50% untrained group and high sucrose 50% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSU 50% group	*HSE 50% group*	*HSU 50% group*	*HSE 50% group*	*HSU 50% group*	*HSE 50% group*	*HSU 50% group*	*HSE 50% group*	*HSU 50% group*	*HSE 50% group*	*HSU 50% group*	*HSE 50% group*
Mean	**0.724**	**0.571**	**150**	**109**	**126**	**93**	**108**	**96**	**36**	**57**	**40**	**19**
SD	**0.006**	**0.009**	**4.320**	**6.082**	**4.618**	**5.354**	**5.033**	**2.581**	**4.472**	**4.041**	**4.320**	**3.696**
SE	**0.002**	**0.003**	**1.632**	**2.299**	**1.745**	**2.023**	**1.902**	**0.975**	**1.690**	**1.527**	**1.632**	**1.397**
t	**34.24**		**14.53**		**12.34**		**5.61**		**9.21**		**9.77**	
p	**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***		**<0.001***	

* Significant change compared with the corresponding value (p<0.001).

HSU 50%: High sucrose 50% untrained group.

HSE 50%: High sucrose 50% exercise group.



(Figure9)

From Table (12) & (Figure 9) it is clear that there was a significant decrease in body mass index, serum glucose, triglyceride, total cholesterol, LDL-C and significant increase in HDL-C in high sucrose 50% exercise group when compared with high sucrose 50% untrained group as:

- Mean Body mass index was changed from 0.724±0.006 in high sucrose 50% untrained group to 0.571±0.009 in high sucrose 50% exercise group.
- Mean Serum glucose level was changed from 150±4.320 mg/dl in high sucrose 50% untrained group to 109±6.082 mg/dl in high sucrose 50% exercise group.
- Mean Serum triglyceride was changed from 126±4.618 mg/dl in high sucrose 50% untrained group to 93±5.354 mg/dl in high sucrose 50% exercise group.
- Mean Serum total cholesterol was changed from 108±5.033 mg/dl in high sucrose 50% untrained group to 96+2.581mg/dl in high sucrose 50% exercise group.
- Mean Serum HDL-C was changed from 36±4.472 mg/dl in high sucrose 50% untrained group to 57±4.041 mg/dl in high sucrose 50% exercise group.
- Mean Serum LDL-C was changed from 40±4.320 mg/dl in high sucrose 50% untrained group to 19±3.696 mg/dl in high sucrose 50% exercise group.

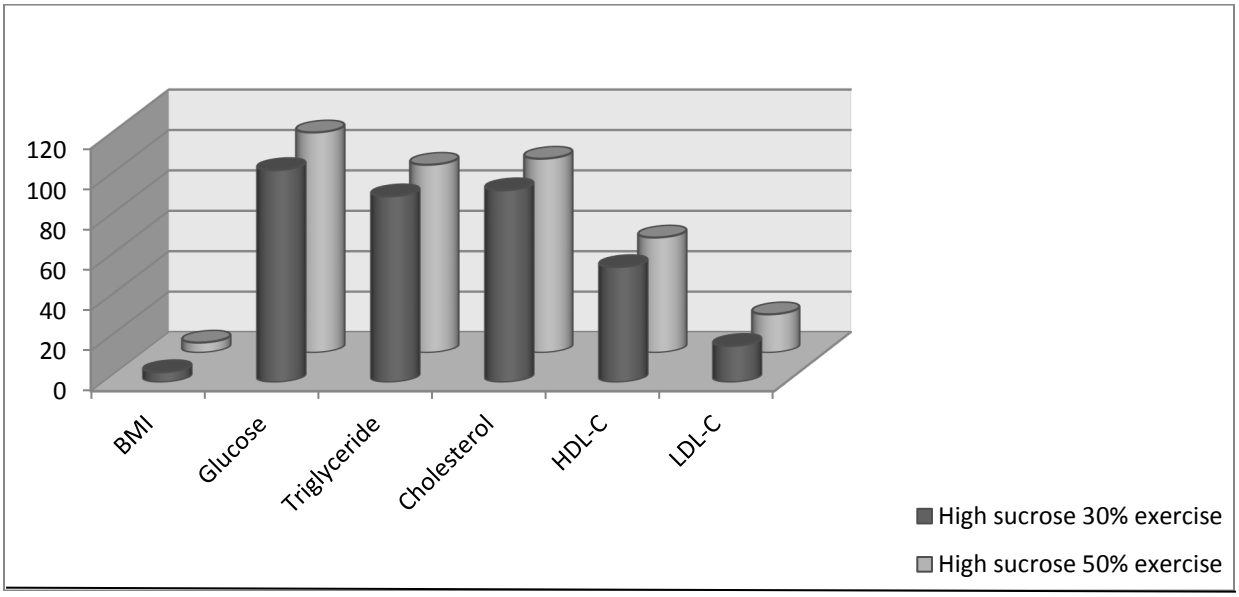
Table (13) & (Figure10) : Comparison between high sucrose 30% exercise group and high sucrose 50% exercise group as regard body mass index, serum glucose and lipid profile (Triglycerides , Total cholesterol, HDL.C and LDL.C mg/dl).

	Body mass index		*Glucose Mg/dl*		*Triglycerides Mg/dl*		*Cholesterol Mg/dl*		*HDL-C Mg/dl*		*LDL-C Mg/dl*	
	HSE 30% group	*HSE 50% group*	*HSE 30% group*	*HSE 50% group*	*HSE 30% group*	*HSE 50% group*	*HSE 30% group*	*HSE 50% group*	*HSE 30% group*	*HSE 50% group*	*HSE 30% group*	*HSE 50% group*
Mean	**0.551**	**0.571**	**105**	**109**	**92**	**93**	**95**	**96**	**57**	**57**	**18**	**19**
SD	**0.003**	**0.009**	**5.567**	**6.082**	**6.633**	**5.354**	**3.366**	**2.581**	**4.320**	**4.041**	**4.725**	**3.696**
SE	**0.001**	**0.003**	**2.104**	**2.299**	**2.507**	**2.023**	**1.272**	**0.975**	**1.632**	**1.527**	**1.786**	**1.397**
t	**5.52**		**1.28**		**0.31**		**0.62**		**0**		**0.44**	
p	**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**		**Non-significant**	

** Non Significant change compared with the corresponding value (p>0.05).

HSE 30%: High sucrose 30% exercise group.

HSE 50%: High sucrose 50% exercise group.



(Figure10)

From Table (13) & (Figure 10) it is clear that there was non- significant change in body mass index, serum glucose, triglyceride, total cholesterol, HDL-C and LDL-C in high sucrose 50% exercise group when compared with high sucrose 30% exercise group as:

- Mean Body mass index was changed from 0.551±0.003 in high sucrose 30% exercise group to 0.571±0.009 in high sucrose 50% exercise group.
- Mean Serum glucose level was changed from 105±5.567 mg/dl in high sucrose 30% exercise group to 109±6.082 mg/dl in high sucrose 50% exercise group.
- Mean Serum triglyceride was changed from 92±6.633 mg/dl in high sucrose 30% exercise group to 93±5.354 mg/dl in high sucrose 50% exercise group.
- Mean Serum total cholesterol was changed from 95±3.366 mg/dl in high sucrose 30% exercise group to 96+2.581mg/dl in high sucrose 50% exercise group.
- Mean Serum HDL-C was changed from 57±4.320 mg/dl in high sucrose 30% exercise group to 57±4.041 mg/dl in high sucrose 50% exercise group.
- Mean Serum LDL-C was changed from 18±4.725 mg/dl in high sucrose 30% exercise group to 19±3.696 mg/dl in high sucrose 50% exercise group.

DISCUSSION

The prevalence of being overweight or obese is progressively high in most affluent countries and this trend seems to be increasing despite all efforts to decrease it. Being obese can seriously affect health and longevity**.** The basic mechanism of becoming obese is an imbalance between calorie intake and energy expenditure, so two major factors thought to contribute to obesity , a sedentary lifestyle and a diet high in fat or recently a diet high in sugar **(*Treuth et al., 2003)***).

As it has been suggested that increased consumption of refined sugar may be causally related to obesity, it was necessary to develop an animal model for studying this obesity. This experiment may be a good starting point for this model. One advantage of this model of obesity would be that it is based on overeating rather than traumatic intervention or genetic abnormalities. Additionally, animals are not forced to consume a single high carbohydrate diet to obtain all of their necessary nutrients, but rather are provided a free choice of a nutritionally complete diet and a palatable, relatively non nutritious, food source (sugar).

The present work was carried out to study high sucrose diet as a risk factor for the development of obesity and the possible role of regular physical activity in preventing the development of this obesity.

In this study obesity was assessed by measurement of body mass index (BMI) and the blood levels of glucose, triglyceride, cholesterol, high density lipoproteins (HDL) and low density lipoproteins (LDL).

The current results revealed that consumption of high sucrose diet in which sucrose represents 30% or 50% of the total caloric requirements by the mice for 4 weeks resulted in increase body weight manifested by

a significant increase ($p < 0.001$) in body mass index as shown in **table (4) and figure (1)** for sucrose 30% and **table (5) and figure (2)** for sucrose 50%. These finding are in agreement with the results of **(Castonguay *et al., 2004)*** as they reported that studying the effect of dietary sucrose on weight gain, food efficiency and body composition has been shown that adult rats given access to sugar in addition to a single standard laboratory diet consume slightly more calories per day and gain more weight than animals given access to only the standard diet. despite rats provided with sugar (sucrose) consume only 40-50 % as much of the standard diet as animals not given sucrose, their feeding of sugar results in a daily energy intake that is 10-20% greater than that of animals not given the sugar option. In most instances this increase in energy intake results in increases in both body weight and adiposity.

Other study by **(Reiser *et al., 2001;* Hirsch *et al., 2001)**observed that rats fed a high sucrose diet usually gained more weight and always deposited more adipose tissue than rats fed a comparable high starch diet. Also, in a other study providing rats with a choice of either sucrose or dextrinized starch found that although the daily caloric intakes of animals in these two groups were almost identical, rats given sucrose gained substantially more weight than animals given starch and this is due to increase energy input from feeding sucrose.

In other words **(Scoccia *et al., 2001)*** reported that addition of sucrose to standard diet enhanced the energy intake in spite of the lower food consumption. Therefore, a lower energy intake from standard diet was compensated by additional calories from sucrose added. But results concerning weight gain in sucrose-fed rats are not entirely conclusive and high sucrose-induced obesity was found even in absence of increased energy intake. This fact indicated that one mechanism by which the body

weights were increased in spite of reduced energy intake in high-sucrose group compared with control was the higher feed efficiency in high-sucrose fed rats which enhances capacity for energy conversion and storage.

The current results revealed that consumption of high sucrose diet in which sucrose represents 30% or 50% of the total caloric requirements by the mice for 4 weeks resulted in a significant increase ($p < 0.001$) in blood glucose level as shown in **table (4) and figure (1)** for sucrose 30% and **table (5) and figure (2)** for sucrose 50%. These finding are in agreement with the results of **(Kanarek *et al.*, 2008; Diniz *et al., 2008)*** as they report that consumption of high-sucrose diet induce obesity and impair the glucose response as high-sucrose fed rats had high-glycemic response and high blood glucose after the oral glucose tolerance test, indicating decreased rate of glucose utilization and that insulin-induced inhibition of hepatic glucose production were blunted in high sucrose rats. The serum glucose level remaining for more time in high-levels at the postprandial period has an important role on obesity related hyperglycemia.

The current results revealed that consumption of high sucrose diet in which sucrose represents 30% or 50% of the total caloric requirements by the mice for 4 weeks resulted in a significant increase ($p < 0.001$) in triglyceride level, total cholesterol level, LDL-C level and a significant decrease ($p < 0.001$) in HDL-L level as shown in **table (4) and figure (1)** for sucrose 30% and **table (5) and figure (2)** for sucrose 50%. These finding are in agreement with the results of **(Brizzi *et al., 2003)*** as they report that there is a growing awareness that variation in the ratio of starch to sucrose can affect the serum levels of lipids. During periods of high-sucrose intake the hepatic tissue can convert glucose into fatty acids,

from which triglyceride (TG) are made, transported to the blood stream as VLDL and stored as fat in adipose tissue. Also high-sucrose diet elevated plasma triglycerides by increasing the triglyceride secretion rate and decreasing the fractional catabolic rate

Furthermore **(Sclafani *et al., 2005;* Glendinning *et al., 2005)** reported that mice gained excess weight and body fat when their chow diet was supplemented with 34% sucrose. This may be due to the fact that sugar activates its own pathways in your body; those metabolic pathways become up regulated this means the more the sugar you eat, the more effective your body is to absorbing it; the more you absorb, the more is converted to fat and stored. Specifically, rats fed high sucrose diets have an increase in serum triglycerides, liver glucose-6-phosphate dehydrogenase (G-6-PD), liver malic and liver fatty acid synthetase enzymes this leads to conversion of glucose to fat followed by its storage in adipose tissue.

In contrast to our findings ***(*Danforth *et al., 1995)*** have reported that a high-sucrose 10% diet does not induce obesity and doesn't elevate blood glucose, cholesterol, LDL-C and HDL-C but elevate serum triglyceride in lean rats feeding isocaloric diet for 2 weeks. This difference may be due to feeding lower concentration of sucrose < 30%, time of study is only 2 weeks.

Other study by_ ***(*Hallfrisch *et al., 1997)*** have reported that Sucrose feeding by rats in a concentration of 11% in high fat diet did not affect glucose levels, but resulted in significantly higher insulin levels than starch feeding because higher insulin levels were required to maintain the same glucose levels in rats fed high fat- sucrose diet. This difference may be due to use of sucrose 11%, use of combined high fat- sucrose diet.

Studying the effect of exercise training for 45 minutes /day for 4 weeks resulted in an improvement in all parameters of obesity on comparing them with the same aged group as there was a significant ($p<0.001$) decrease in body mass index, plasma glucose, triglyceride, total cholesterol, LDL-C and significant increase in HDL-C as shown in **table (9-10-11-12)** and **figure (6-7-8-9)**. These results highly suggest that the exercise is one of the main strategies in the prevention and control of obesity. these finding are in agreement with the results of **(Baranowski *et al., 2003;* Dennison *et al., 2004;* Burke *et al., 2003)** as they reported that regular physical exercise reduce the feed efficiency and the body weight gain in high sucrose fed rats, also it has a protective effect normalizing the glucose response seen in high sucrose (HS) group compared with HS-exercise group. It has been shown that exercise prevented sucrose-induced insulin resistance.

Several Mechanisms have been proposed to account for these exercise-related improvements in insulin resistance state **(Ivy et al., 1999)**have proposed many pre receptor events e.g. increased muscle glucose delivery because of increased muscle capillary density, and changes in muscle composition favoring increased glucose disposal as well as post receptor adaptations e.g. enhanced glucose transport via increased concentration of GLUT-4 in skeletal muscle ***(Dela et al., 1994), (Saengsirisuwan et al., 2002)*** and greater activity of the enzymes hexokinase II ***(Koval et al., 1998)*** and glycogen synthase ***(Ebeling et al., 1993).***

Lastly, the anti-inflammatory effects of exercise are well known and studies have shown that exercise reduces TNF-α concentrations, which may in part explain the increases in GLUT4 expression ***(Petersen and Pedersen, 2005)***.

This improvement in insulin resistance in mice under the effect of exercise was reported by ***(Hu et al., 2006)*** and in humans by ***(Dela et al., 1994; John et al.,2000; Houmard et al.,2003 ;Devlin and Horton, 2005).*** Also ***(SoJung et al., 2005)*** reported that moderate-intensity exercise was associated with significant reductions in abdominal fat and skeletal muscle lipid content in type II diabetes which was associated with improvement in insulin resistance state. And more over ***(Ross et al., 2000)*** showed that exercise training does not necessarily need to reduce body weight to have beneficial physiological effects on insulin resistance.

Other study by ***(*Petersen *et al., 2009)*** report that exercise has beneficial effects on high-sucrose diet-induced dyslipidemia enhancing HDL/TG ratio and reducing oxidized LDL (ox- LDL) Cholesterol levels enhanced in HS group. There is a growing awareness that total serum cholesterol level is a reflection of the individual lipoprotein level and metabolism. HS-exercise group had lower LDL than HS rats, additionally; exercise could have properties depressing hepatic cholesterol synthesis. We can affirm that exercise improved the lipid profile in high-sucrose intake condition. Judging from their experimental results, it is evident that the exercise effects on serum lipids, glucose tolerance and body weight were at least in part via antioxidant/oxidant mediated process, thus diminishing the cellular uptake of lipids from the blood and changing lipid constituents of LDL, inducing LDL-oxidation. And so improves the lipid profile. Secondary hyperglycemia even within normal nor diabetic range is directly involved in pathogenic processes because it creates oxidative stress. Exercise decreasing the oxidative stress and thus improving the glucose tolerance.

Furthermore our results are in agreement with the results of **(Bullough *et al., 1995;* Calles-Escandon *et al., 1996)** as they reported that during aerobic exercise, the contribution of fat to energy production decreases **(Brooks & Mercier, 1994)**. Once exercise stops, fat oxidation is increased during the post-exercise period **(Horton *et al.* 1995).** In addition, increased fat oxidation at rest has been observed on the day following aerobic exercise **(Calles-Escandon *et al.* 1996).** Little information is available on daily rates of fat oxidation with aerobic exercise; nevertheless, it appears that aerobic exercise can increase total fat oxidation as well as 24 h energy expenditure. Also Adaptations in skeletal muscle following aerobic exercise training contribute to the increased ability to utilize lipid as a fuel during exercise **(Kiens *et al.* 1993)**. These changes include an increase in the proportion of oxidative muscle fibers, an increase in the muscle mitochondrial content and an increase in the activity of various enzymes involved in oxidative metabolism **(Holloszy & Coyle, 1984).**

Aerobic exercise training also leads to an increased sensitivity of adipose tissue to the lipolytic action of catecholamines **(Depres *et al.* 1984).** This increases the ability to mobilize lipid. Resistance exercise and high-intensity exercise training tend to increase the number of glycolytic muscle fibers **(*Wajchenberg*, 2000)** which may be less favorable to lipid oxidation. It could be known that aerobic training, rather than resistance and/or high-intensity exercise training, may be best for creating a metabolic environment geared towards fat utilization and decreasing fat mass.

In contrast to these results **(Muto et al., 2001)** showed that acute exercise training failed to change obesity parameters of increased body weight and failed to improve levels of glucose and lipid profile. This may

be due to the fact that acute exercise arose from differences in exercise duration and intensity and sustained exercise of sufficient intensity might provide a way to increase oxidant/antioxidant ratio in skeletal muscle and thereby ameliorate reduced insulin sensitivity and enhance oxidative stress.

By comparing the effects of regular physical exercise on obesity induced by feeding 30% sucrose of the total caloric requirement with its effects on obesity induced by feeding 50% sucrose of the total caloric requirement there was none significant changes regarding body mass index, blood level of glucose, triglyceride, cholesterol, HDL-C and LDL-C these finding are in agreement with **(Cohen et al., 2002)** who reported that certain studies have not demonstrated additional benefits of training programs on obesity induced by different concentrations of sucrose > 27%.

CONCLUSION

We conclude that high dietary sucrose is a risk factor of obesity through its high feed efficiency effect (increase energy intake) and this obesity effect doesnot depend on concentration of sucrose as it occurs with both concentration of sucrose (30% - 50%) and that regular physical exercise can protects from obesity results from sucrose (30% - 50%) through increase fat oxidation and hence energy expenditure so it reduce the feeding efficiency of sucrose.

SUMMARY

Obesity has became one of the most important causes of heart diseases, cancer and other diseases that increase the incidence of premature death in recent years and during the last ten years, the increase consumption of sugars in diet and lack of daily physical activities due to reliance on modern technology have became the most dangerous risk factors that leads to development of obesity and its hazards.

By studying the effect of overconsumption of sugar in diets on different experimental animals, we found a strong relationship between overconsumption of sugar and obesity.

Sucrose or cane sugar play an important role in supplying energy necessary for body functions. It breaks down in small intestine to glucose and fructose by sucrase enzyme and both of them are absorbed where fructose is converted to glucose in liver and glucose used by every cell in the body in production of energy. With overconsumption of sucrose much of glucose produced not burned but converted stored in the body in the form of fat leading to obesity.

Physical activity play an important role in equalizing energy intake and energy expenditure in human body by burning extra energy gained to the body and thus prevent its storage in the body as fat so low physical activity is associated with obesity.

This study is done to detect the possible effect of regular physical activity on high-sucrose diet-induced obesity and its relation to the levels of lipids in the blood.

Adult white male albino rats are used which were divided into 3 main groups:

Group I: control group

Receive standard diet and remain untrained for 4 weeks.

Group II: high sucrose group subdivided to:

Group IIa: receive sucrose 30% and remain untrained for 4 weeks.

Group IIb: receive sucrose 50% and remain untrained for 4 weeks.

Group III: exercise group subdivided to:

Group IIIa: receive sucrose 30% and perform exercise for the last 3weeks of the experment (45min/day) in tanks.

Group IIIb: receive sucrose 50% and perform exercise for the last 3weeks of the experment (45min/day) in tanks.

Obesity was assesed by measuring body mass index (BMI) which equall weight of rat in grams/ length of rat in Cm2, also blood glucose and lipid profile levels are measured. All the previous parameters are used also to asses the effects of regular exercise.

The obtained results of this study could be summarized as follow:

- Consumption of high surose diet in which sucrose represent 30% and 50% of caloric requirement causes a significant increase in body weight manifested by increase BMI serum glucose and lipid profile when compared with control group.
- Regular exercise results in significant decrease in BMI, serum glucose and lipid profile in exercise group when copmared with high sucrose group

RECOMMENDATIONS

- High sucrose consumption is a risk factor for obesity and other diseases so further studys needed to detect the effects of feeding sucrose in concentrations > 50% on liver enzymes, insulin level, serum oxidative stress enzymes and parenchyma of some organs.
- Regular physical exercise is protective againest high sugar-induced obesity but further studys needed to detect the effects of regular exercise on liver enzymes and fatty liver.

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الملخص العربى

لقد أصبح مرض السمنة سبباً هاماً لأمراض القلب والسرطان وغيرها من الامراض المؤدية إلى الوفاة فى السنوات الأخيرة وخلال السنوات العشرة الأخيرة ازدادت أهمية تناول السكريات بكميات كبيرة فى الوجبات المختلفة وعدم ممارسة التمارين الرياضية بل وقلة الانشطة اليومية العادية نتيجة للتطور والاعتماد الكامل على التكنولوجيا الحديثة كعوامل خطورة ادت الى زيادة السمنة وزيادة المخاطر التى قد تؤدى اليها.

وقد تم دراسة تأثير تناول السكريات بكميات كبيرة على زيادة الوزن فى مختلف أنواع حيوانات التجارب وثبت ان هناك ارتباط شديد بين زيادة تناول السكريات والسمنة

ويلعب السكروز دوراً هام فى امداد الجسم بالطاقة حيث يتم تكسيره فى الامعاء الى جلوكوز وفركتوز بواسطة انزيم السكاريز وامتصاص كليهما الى الجسم حيث يتم تحويل الفركتوز الى جلوكوز فى الكبد ويتم استخدام الجلوكوز كمصدر للطاقة لجميع خلايا الجسم ولكن مع زيادة تناول السكروز يقوم الجسم بتحويل الجلوكوز الزائد الى دهون تخزن بالجسم وتؤدى الى السمنة.

ويعمل النشاط البدنى على مساواة الطاقة المكتسبة للجسم بالطاقة المفقودة من الجسم وبالتالى عدم تخزين الطاقة الزائدة حيث ان زيادة الطاقة المكتسبة مع عدم زيادة الطاقة المفقودة يؤدى الى زيادة تخزين هذة الطاقة على شكل دهون بالجسم ويؤدى الى السمنة.

وقد أجريت هذه الدراسة لتوضيح دور التمارين الرياضية فى حماية الجسم من السمنة التجريبية الناتجة عن استخدام وجبات غذائية عالية السكروز، وعلاقة ذلك بزيادة الدهون فى الدم.

وقد تمت هذه الدراسة على ثلاث مجموعات رئيسية من ذكور فئران التجارب البيضاء البالغة أولى هذه المجموعات مجموعة ضابطة تتناول الوجبة العادية ولا تقوم بممارسة اى تمارين رياضية، المجموعة الثانية تتناول وجبة عالية السكروز وتنقسم الى مجموعتين من حيث تركيز السكروز فى الوجبة ولا تقوم بممارسة اى تمارين رياضية، المجموعة الثالثة تتناول وجبة عالية السكروز وتنقسم الى مجموعتين من حيث تركيز السكروز فى الوجبة وتقوم بممارسة التمارين الرياضية بانتظام ٤٥ دقيقة من العوم فى خزان المياه يوميا لمدة ثلاث اسابيع.

وقد تم تقييم السمنة التجريبية بقياس مؤشر كتلة الجسم الذى يساوى الوزن بالكيلوجرام على الطول بالمتر مربع فى الانسان ولكن فى الفئران يساوى الوزن بالجرام على الطول بالسنتيمتر مربع وايضا قياس السكر والدهون الثلاثية والكوليستيرول عالى الكثافة ومنخفض الكثافة فى الدم وتقييم اثر التمارين الرياضية بقياس هذة المؤشرات ايضا.

هي لقم تكخي ش متئ ث منطخخز ب قئية و:

أحدث تناول السكروز بتركيز ٣٠٪ او ٥٠٪ زيادة فى الوزن عن طريق زيادة ملحوظة فى مؤشر كتلة الجسم وذلك نتيجة لوجود زيادة ملحوظة فى نسبة الجلوكوز والدهون بالدم عندما تم مقارنة هذه النتائج بنتائج المجموعة الضابطة

ممارسة التمارين الرياضية بانتظام أحدث نقصاً ملحوظاً فى مؤشر كتلة الجسم كما احدث نقص ملحوظا فى نسبة الجلوكوز والدهون بالدم عندما تم مقارنة النتائج بنتائج المجموعة التى تتناول السكروز ولا تقوم بممارسة اى تمارين رياضية.

ومن هذه النتائج نستخلص أن زيادة تناول السكروز يمثل عامل خطورة لمرض السمنة وأن ممارسة التمارين الرياضية بانتظام تحمى من السمنة عن طريق حرق الطاقة الزائدة المكتسبة من تناول السكروز بكميات كبيرة وعدم تخزينها فى الجسم على شكل دهون.

SUMMARY

Obesity has become one of the most important causes of heart diseases, cancer and other diseases that increase the incidence of premature death in recent years and during the last ten years, the increase consumption of sugars in diet and lack of daily physical activities due to reliance on modern technology have become the most dangerous risk factors that leads to development of obesity and its hazards.

By studying the effect of overconsumption of sugar in diets on different experimental animals, we found a strong relationship between overconsumption of sugar and obesity.

Sucrose or cane sugar play an important role in supplying energy necessary for body functions. It breaks down in small intestine to glucose and fructose by sucrase enzyme and both of them are absorbed where fructose is converted to glucose in liver and glucose used by every cell in the body in production of energy. With overconsumption of sucrose much of glucose produced not burned but converted stored in the body in the form of fat leading to obesity.

Physical activity plays an important role in equalizing energy intake and energy expenditure in human body by burning extra energy gained to the body and thus prevent its storage in the body as fat so low physical activity is associated with obesity.

This study is done to detect the possible effect of regular physical activity on high-sucrose diet-induced obesity and its relation to the levels of lipids in the blood.

Adult white male albino rats are used which were divided into 3 main groups:

Group I: control group

Receive standard diet and remain untrained for 4 weeks.

Group II: high sucrose group subdivided to:

Group IIa: receive sucrose 30% and remain untrained for 4 weeks.

Group IIb: receive sucrose 50% and remain untrained for 4 weeks.

Group III: exercise group subdivided to:

Group IIIa: receive sucrose 30% and perform exercise for the last 3weeks of the experiment (45min/day) in tanks.

Group IIIb: receive sucrose 50% and perform exercise for the last 3weeks of the experiment (45min/day) in tanks.

Obesity was assessed by measuring body mass index (BMI) which equal weight of rat in grams/ length of rat in Cm2, also blood glucose and lipid profile levels are measured. All the previous parameters are used also to assess the effects of regular exercise.

The obtained results of this study could be summarized as follow:

- Consumption of high sucrose diet in which sucrose represent 30% and 50% of caloric requirement causes a significant increase in body weight manifested by increase BMI serum glucose and lipid profile when compared with control group.
- Regular exercise results in significant decrease in BMI, serum glucose and lipid profile in exercise group when compared with high sucrose group

RECOMMENDATIONS

- High sucrose consumption is a risk factor for obesity and other diseases so further studys needed to detect the effects of feeding sucrose in concentrations > 50% on liver enzymes, insulin level, serum oxidative stress enzymes and parenchyma of some organs.
- Regular physical exercise is protective againest high sugar-induced obesity but further studys needed to detect the effects of regular exercise on liver enzymes and fatty liver.

دراســة أثر ممارسة التمارين الرياضية بانتظام على السمنة الناجمة عن اتباع نظام غذائى عالى السكروز

دراسة للحصول على درجة الماجيستير

فى الفسيولوجى

مقدمة من الطبيب

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