Introduction

Obesity is a clinical condition characterized by excessive deposition of adipose tissue, in which one's health can be potentially affected **(WHO, 2006).** Obesity is defined as a body mass index (BMI) greater than 30 kg/m² **(Wandell et al., 2009).**

Morbid obesity is defined as having a body mass index (BMI) ≥ 40 kg/m² **(Sturm, 2003).** Obesity is associated with a number of co-morbidities such as hypertension, dyslipidemia, type-2 diabetes or insulin resistance, sleep apnea, …etc **(Bray, 2004).**

Over the past few decades the number of bariatric surgery procedures has increased significantly, worldwide with the most commonly performed procedure being laparoscopic Roux-en-Y gastric bypass surgery (RYGB) **(Buchwald and Williams, 2004).**

Two major reasons can be defined behind this. First, the prevalence of morbid obesity increases rapidly, approaching 8% for some populations **(Hedley et al., 2004).** Secondly, there is a growing consensus that bariatric surgery is currently the most efficient treatment for morbid obesity, with a low mortality rate, and pronounced effect on comorbidities **(Farrell et al., 2009).**

Physical fitness is a set of attributes that people have or achieve. Being physically fit has been defined as "the ability to carry out daily tasks with vigor and alertness, without undue fatigue and with ample energy to enjoy leisure-time pursuits and to meet unforeseen emergencies **(President's Council on Physical Fitness and Sports, 1971).**

However, it remains to be determined whether poor physical fitness improves after bariatric surgery or not, and is a key factor in the vicious circle of obesity and physical inactivity in morbidly obese patients **(Pietilainen et al., 2008).**

A number of measurable components do contribute to physical fitness .The most frequently cited components fall into two groups: one related to health and the other related to skills that pertain more to athletic ability **(Pate, 1983).**

The health-related components of physical fitness are:

(1) Cardiorespiratory endurance.

(2) Muscular endurance.

(3) Muscular strength.

(4) Body composition.

(5) Flexibility.

These five health-related components of physical fitness are more important to public health than are the components related to athletic ability; therefore, we limit our discussion to them.

In this essay we will review the effect of bariatric surgery on health components of physical fitness and the effect of increasing physical activity during months following the surgery on its health related components and weight loss.

Aim of the study

The aim of this essay is to review the studies concerning the effect of bariatric surgery on physical fitness, and the effect of improving physical activity after the surgery.

Obesity

Definition:

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health, leading to reduced life expectancy and/or increased health problems **(Haslam and James, 2005).**

Classifications:

Obesity can be classified according to three measures, body mass index (BMI), waist circumference, waist-hip ratio and body fat percentage.

A) Body mass index (BMI):

Body mass index is a simple and widely used method for estimating body fat mass **(Mei et al., 2002),** and is calculated by dividing weight in kilograms by square height in meters (kg/m^2). Classification of overweight and obesity by BMI are shown in table one.

$$\mathrm{BMI} = \frac{\mathrm{kilograms}}{\mathrm{meters}^2}$$

B) Waist circumference and waist-hip ratio:

Central obesity, particularly visceral obesity (apple-shaped, android), confers increased risk for metabolic complications of obesity, whereas lower or peripheral obesity, preferential fat

accumulation in gluteofemoral region and leg (pear-shaped, gynoid), is associated with lower risk and may be protective Thus, measuring waist and hip circumferences and calculating waist-to-hip ratio (WHR) better identifies abdominally obese subjects at risk of developing metabolic diseases **(Lee et al., 2013)**

In the United States cutoffs of waist circumferences greater than 102 cm in men and 88 cm in women, and WHR greater than 1.0 in men and 0.8 in women are suggested to define those at increased risk, while in the European Union waist circumference of ≥ 94 cm in men and ≥ 80 cm in non pregnant women are used as cut offs for central obesity **(Tsigosa et al., 2008).** Classification of overweight and obesity by waist circumference are shown in table one.

C) Body fat percentage:

Body fat percentage is total body fat expressed as a percentage of total body weight. Most researchers have used >25% in men, and >30% in women, as cutoff points to define obesity **(Okorodudu et al., 2010).**

Body fat percentage can be estimated from a person's BMI by the following formula:

Body fat percentage = 1.2 x BMI + 0.23 x age – 5.4 – 10.8 x gender

Where gender is 0 if female and 1 if male

There are many other methods used to determine body fat percentage such as hydrostatic weighing, skin fold test, bioelectrical impedance analysis, computed tomography (CT scan), magnetic resonance imaging (MRI) and dual energy x-ray absorptiometry (DEXA).

Table (1): Classification of overweight and obesity by BMI, waist circumference, and associated disease risk **(WHO, 1997).**

			Disease risk (relative to normal weight and waist circumference)	
Classification	**BMI (kg/m^2)**	**Obesity class**	Men ≤40 in (102 cm) Women ≤35 in (88 cm)	>40 in >35 in
Underweight	< 18.5			
Normal	18.5-24.9			
Overweight	25 to 29.9		Increased	High
Obesity	30 to 34.9	I	High	Very high
obesity	35 to 39.9	II	Very high	Very high
Extreme obesity	≥ 40	III	Extremely high	Extremely high

In=inch, cm=centimeter

Pathophysiology of Obesity:

There are many possible pathophysiological mechanisms involved in the development and maintenance of obesity Substantial evidence suggests that body weight is regulated by both endocrine and neural components that ultimately influence the effector arms of energy intake and expenditure. This complex regulatory system is necessary because even small imbalances between energy intake and expenditure will ultimately have large effects on body weight **(Flier, 2004).**

Since the discovery of leptin in 1994, many other hormonal mechanisms have been elucidated that participate in the regulation of appetite and food intake, storage patterns of adipose tissue, and development of insulin resistance. Also many other mediators have been studied such as ghrelin, insulin, orexin, PYY 3-36, cholecystokinin, and adiponectin **(Flier, 2004).**

Leptin and ghrelin are considered to be complementary in their influence on appetite, with ghrelin produced by the stomach modulating short-term appetitive control (i.e. to eat when the stomach is empty and to stop when the stomach is stretched). Leptin is produced by adipose tissue to signal fat storage reserves in the body, and mediates long-term appetitive controls (i.e. to eat more when fat storages are low and less when fat storages are high) **(Flier, 2004).**

While leptin and ghrelin are produced peripherally, they control appetite through their actions on the central nervous system. In particular, they and other appetite-related hormones act on the hypothalamus, a region of the brain central to the regulation of food intake and energy expenditure. There are several circuits within the hypothalamus that contribute to its role in integrating appetite, the melanocortin pathway being the most well understood **(Flier, 2004).**

The circuit begins with an area of the hypothalamus, the arcuate nucleus, that has outputs to the lateral hypothalamus (LH) and ventromedial hypothalamus (VMH), the brain's feeding and satiety centers, respectively **(Flier, 2004).**

The arcuate nucleus contains two distinct groups of neurons. The first group coexpresses neuropeptide Y (NPY) and agouti-related peptide (AgRP) and has stimulatory inputs to the LH and inhibitory inputs to the VMH. The second group coexpresses pro-opiomelanocortin (POMC) and cocaine- and amphetamine-regulated transcript (CART) and has stimulatory inputs to the VMH and inhibitory inputs to the LH. Consequently, NPY/AgRP neurons stimulate feeding and inhibit satiety, while POMC/CART neurons stimulate satiety and inhibit feeding. Both groups of arcuate nucleus neurons are regulated in part by leptin. Leptin inhibits the NPY/AgRP group while stimulating the POMC/CART group. Thus a deficiency in

leptin signaling, either via leptin deficiency or leptin resistance, leads to overfeeding and may account for some genetic and acquired forms of obesity **(Flier, 2004)** (Fig 1).

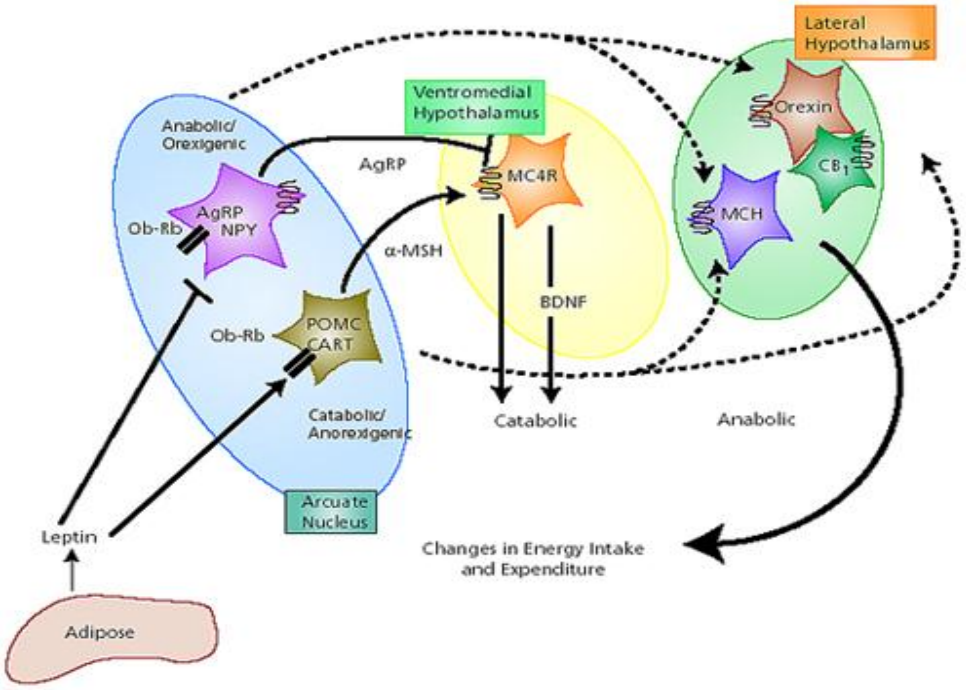

Figure (1): Pathophysiology of obesity **(Flier, 2004).**

There are several factors other than leptin and ghrelin that share in the pathogenesis of obesity such as:

1. Lipoprotein lipase enzyme:

The enzyme lipoprotein lipase, produced by the adiposity and residing on its capillary endothelium, permits fat cells to take up fatty acids from circulating chylomicrons (dietary fat) and very low density lipoprotein **(Pi-sunyer, 2000).**

Well fed fat cells can grow to maximum of 1 μg. Storage of more fat requires an increase in adipocyte number by differentiation of preadipocytes. The signals for this hyperplasia are unknown; however, excess caloric consumption can drive an increase in adipocytes number. The converse that increased adiposity number derives increased food intake is also possible **(Flier, 2001).**

2. Adiponectin:

It is a protein derived exclusively from adipose tissue. It seems to have protective metabolic and anti inflammatory properties and reduces the inflammatory changes in the cardiovascular system that lead to heart disease. It decreases with obesity and increase with weight loss **(Campbell and Haslam, 2005).**

3. Resistin:

It's a peptide hormone secreted by white fat cells discovered and named after resistance to insulin. Genetic studies show a relationship between obesity, insulin resistance and resist in hormone **(Ukkola, 2002).**

4. Cytokines:

Obesity is associated with increased plasma levels of cytokines (inflammatory factors) such as interleukins,

C-reactive protein, tumor necrosis factor and insulin like growth factor 1. These cytokines are produced by adipose tissue and are responsible for inflammatory changes in the cardiovascular and other systems **(Fain , 2006).**

Many other proteins are in various stages of research including pro 12 Ala polymorphism, omentin, adipocyte complement related protein, pancreatic peptide YY, uncoupling 2 and 3, neuropeptide Y and GLP-1 and 2 all of which can be considered as energy regulators **(Fain , 2006).**

Etiology of Obesity:

Weight regulation appears to be much more complex than a simple problem of energy in versus energy out. It was estimated that as much as 70% of a predisposition to obesity is genetic .Those implicated genes might not necessarily convey an inevitability of future weight gain. Our genes are switched on or switched off by environmental, neurological and psychogenic influences all of which could have a veering on an individual's predisposition and ultimate development of obesity. So, to understand the best ways to reverse the trend towards obesity, it is needed to understand its causes **(Campbell and Haslam, 2005)** (Fig 2) .

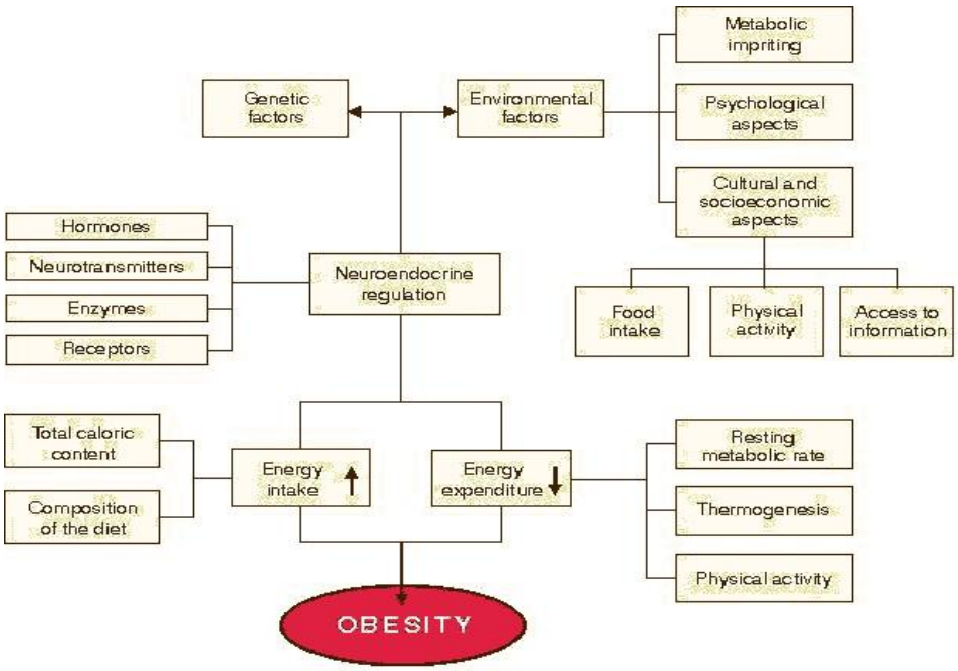

Figure (2): Etiology of obesity **(Balaban and Silva, 2004).**

ROLE OF GENES VERSUS ENVIRONMENT

Obesity is commonly seen in families, and the heritability of body weight is similar to that for height. It is difficult to distinguish the role of genes and environmental factors. Adoptees more closely resemble their biologic than adoptive parents with respect to obesity, providing strong support for genetic influences. Likewise, identical twins have very similar BMIs whether reared together or apart, and their BMIs are much more strongly correlated than those of dizygotic twins. These genetic effects appear to relate to both energy intake and expenditure **(Chaput et al., 2014) .**

Whatever the role of genes, it is clear that the environment plays a key role in obesity, as evidenced by the fact that famine prevents obesity in even the most obesity-prone individual. In addition, the recent increase in the prevalence of obesity in the United States is far too rapid to be due to changes in the gene pool. Undoubtedly, genes influence the susceptibility to obesity in response to specific diets and availability of nutrition. Cultural factors are also important—these relate to both availability and composition of the diet and to changes in the level of physical activity. In industrial societies, obesity is more common among poor women, whereas in underdeveloped countries, wealthier women are more often obese. In children, obesity correlates to some degree with time spent watching television. Although the role of diet composition in obesity

continues to generate controversy, it appears that high-fat diets may promote obesity when combined with diets rich in simple, rapidly absorbed carbohydrates **(Chaput et al., 2014) .**

Additional environmental factors may contribute to the increasing obesity prevalence. Both epidemiologic correlations and experimental data suggest that sleep deprivation leads to increased obesity. Changes in gut microbiome with capacity to alter energy balance are receiving experimental support from animal studies, and a possible role for obesigenic viral infections continues to receive sporadic attention **(Chaput et al., 2014) .**

SPECIFIC GENETIC SYNDROMES

For many years, obesity in rodents has been known to be caused by a number of distinct mutations distributed through the genome. Most of these single-gene mutations cause both hyperphagia and diminished energy expenditure, suggesting a physiologic link between these two parameters of energy homeostasis. Identification of the *ob* gene mutation in genetically obese (ob/ob) mice represented a major breakthrough in the field. The ob/ob mouse develops severe obesity, insulin resistance, and hyperphagia, as well as efficient metabolism The product of the *ob* gene is the peptide leptin, which is secreted by adipose cells and acts primarily through