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THE PROGRESSING GENESIS OF OBESITY: A REVIEW

Megha Rana, Shivani Dhiman, Monika, Amanpreet Kaur* and Naresh Singh Gill

Department of Pharmacology, Rayat Institute of Pharmacy, Rail, SBS Nagar-144533, Punjab, India.

*Assistant Professor, Department of Pharmacology, Rayat Institute of Pharmacy, Rail, SBS Nagar-144533, Punjab, India.

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***Corresponding Author**
Amanpreet Kaur
Assistant Professor, Department of Pharmacology, Rayat Institute of Pharmacy, Rail, SBS Nagar-144533, Punjab, India.

ABSTRACT

When abnormal amount of fats get deposited into an individual's body this condition leads to obesity. This takes place when energy intake exceeds than energy expenditure over time. The obesity epidemic is the outcome of a multifaceted intricate interaction between environmental factors, genetic susceptibility, and human behavior. Overweight and obese children are likely to stay obese into adulthood, and more likely to develop non-communicable co-morbid disease like diabetes and cardiovascular diseases at a younger age. Sedentary lifestyle, poor dietary habits and the aging of the population are the secondary causes of obesity generally go undetected and untreated. Hypothalamic obesity is a rare syndrome that can be caused by trauma, tumor, inflammatory disease, surgery in the posterior fosse increased intracranial pressure. The amount of energy intake relative to energy expenditure is the central reason for the development of obesity. Many other factors are accountable for the progression of obesity such as hyperthyroidism, certain drugs, PCOS, nicotine withdrawal and many more. Some patients may benefit from targeted behavioral therapies, while others may benefit from surgery. The most difficult task will be to alter our lifestyles so that weight gain does not become unavoidable for a huge percentage of the population.

KEYWORDS: Obesity, genesis of obesity, etiology of obesity, dietary fat, maternal obesity, factors, hypothalamic obesity, Cushing's disease, weight gain, hormonal imbalance.

1. INTRODUCTION

Obesity ensues when an individual's body accumulates abnormal amounts of fat. This takes place when energy intake exceeds energy expenditure over time.[1] The prevalence of obesity is depend upon some factors such as age, gender. Family characteristics parenting style, parent life style also play a significant in progressing of obesity role. Environmental factors such as school policies, demographics, and parents work related demands further influence eating and activity behaviors.[2] It has been linked to micro-organism epigenetic, increasing maternal age, greater fecundity, lack of sleep, endocrine disruptors.[3] At a physiological level, the principle gluco-corticoid cortisol, contributes significantly to the regulation of protein, carbohydrate, lipid and nucleic acid, enhancing the production of blood glucose by antagonism the secretion and action of insulin, increasing peripheral protein breakdown and enhancing the activation of lipo-protein lipase in adipocytes which in turn increase fat accumulation.[4] The main factors responsible for childhood obesity are high uses of unhealthy food parental responsibility, modern technology and the mass media. Research based evidences shows that psychological and behavioral causative agents of obesity among the children. Lack of physical activity and heredity both caused obesity among the children similarly the problem of obesity also found more in female as compared to male.[5] The emerging interest in systems based intervention responds to the growing awareness that the prevention of obesity must engage with the complexity of causes from an individual level through to policy settings. While system thinking has a long history in other scientific disciplines like engineering the application to obesity prevention is recent and not well described.[6]

2. Etiology of obesity

Obesity is caused by a combination of behavioral, genetic, environmental, physiological, social, and cultural variables, as listed below:

2.1 Dietary fat intake

Increased fast food consumption has been linked with obesity in the recent years. Many families, especially those with two parents working outside the home for these places as they are often favored by their children and are both convenient and inexpensive. Foods served at fast food restaurants tend to contain a high number of calories with low nutritional values. A study conducted examined the eating habits of lean and overweight adolescents at fast food restaurants. Researchers found that both groups consumed more calories eating fast food than they would typically in a home setting but the lean group compensated for the higher caloric

intake by adjusting their caloric intake before or after the fast food meal in anticipation or compensation for the excess calories consumed during the fast food meal. Though many studies have shown weight gain with regular consumption of fast food, it is difficult to establish a causal relationship between fast food and obesity.[7] Studies of total energy intake in the population suggest that this has been declining since the 1980s, which is paradoxical, given that this is the period that has seen the most rapid and sustained increase in the prevalence of obesity. The type of food that tends to be high in fat, and therefore tends to be energy dense, and thus the consumption of such food lead to less satiety than with less energy dense foods, and encourage overconsumption, suggesting that the peripheral and hypothalamic regulatory systems are less sensitive to a high fat diet.[8] Voluntary overeating (repeated ingestion of energy exceeding daily energy needs) can increase body weight in normal-weight men and women.[9] The relative weight in several populations is directly related to the percentage of dietary fat in the diet.[10] There are some recent studies to suggest that the consumptions of sugar-sweetened beverages in children may enhance the risk of more rapid weight gain.[11]

2.2 Hypothalamic damage

Hypothalamic damage or injury can result in a severe and devastating form of obesity. The hypothalamus is an important brain centre that integrates neuronal and hormonal signals that regulate energy balance. Hypothalamic obesity is often severe, rapid, and inadequately managed with conventional treatment such as caloric restriction and lifestyle modification. The weight gain has a significant negative impact on quality of life and increases the risk for early cardiovascular co-morbidities and mortality in patients.[12] Hypothalamic obesity is a rare syndrome that can be caused by trauma, tumor, inflammatory disease, surgery in the posterior fosse increased intracranial pressure.[13]

The symptoms usually present in 1 of 3 patterns

(a) Headache, vomiting, and diminished vision;

(b) Impaired endocrine function affecting the reproductive system with amenorrhea or impotence, diabetes insipid us, and thyroid or adrenal insufficiency.

(c) Neurologic and physiologic derangements including convulsion, coma, somnolence, and hypothermia or hyperthermia.[13]

2.3 Cushing's disease

A common clinical feature of Cushing's disease is progressive central obesity, usually involving the face, neck (leading to a buffalo hump and obscuring of the clavicles), trunk, abdomen, and, internally, the mesentery and mediastinum.[10] Individuals with Cushing's syndrome can develop moon faces, facial plethora, supra-clavicular fat pads, truncal obesity.[14]

2.4 Hypothyroidism

Hypothyroidism is associated with decreased thermo genesis, decreased metabolic rate, and has also been shown to correlate with a higher body mass index (BMI) and a higher prevalence of obesity.[15] Patients with hypothyroidism frequently gain weight because of a generalized slowing of metabolic activity, and some of this gain is fat. The weight gain, however, is usually modest, a marked obesity is uncommon. Hypothyroidism is a common diagnosis, particularly in older women; in this group a thyroid-stimulating hormone test is valuable for diagnosis.[10]

2.5 Polycystic ovary syndrome

Nearly 50% of women with polycystic ovary syndrome are obese. The cardinal features of this syndrome are oligomenorrhea, hirsutism, and polycystic ovaries. Although obesity is not always present, it is seen more often than not. Insulin resistance is present in both normal-weight and overweight persons. Luteinizing hormone is usually increased and ovarian overproduction of testosterone, probably through stimulation of the ovary by insulin like growth factor-I, is a main source of testosterone.[10] Insulin resistance is a common finding in PCOS that is independent of obesity. Insulin-mediated glucose disposal, reflecting mainly insulin action on skeletal muscle is decreased by 35-40% in women with PCOS compared to weight comparable reproductively normal women.[16]

2.6 Growth-hormone deficiency

GH can contribute to insulin resistance that may develop when caloric supply exceeds demand, the reduction in GH secretion that occurs with obesity may be an adaptive phenomenon to prevent insulin resistance however, the reduction in GH secretion may further increase fat accumulation by reducing lipolysis, and therefore exacerbate obesity, establishing a dangerous vicious circle. According, truncal adiposity is one of the most important clinical findings of the adult GH.[17] Lean body mass is decreased and fat mass is increased in adults

who are deficient in growth hormone as compared with those who have normal growth-hormone secretion. Growth hormone-replacement therapy reduces body and visceral fat.[10]

2.7 Pregnancy

Gaining more than the recommended weight during pregnancy can put women at increased risk of becoming obese and developing related health problems, including high blood pressure, later in life. Pregnancy constitutes a biological cause of weight cycling. Since repeated weight variation has been association with sustained weight increase and subsequent metabolic complication, the role of pregnancy in affecting body weight was analyzed retrospectively in a group of 128 severely obese patients at the obesity unit. Their present mean age was 47.8 +/-10.7 years, the mean age at birth of their first child 24.1 +/-5.1 years and their mean present BMI was 37.8 +/-5.5 kg/m2. Seventy-three percent of the women reported weight retention of more than 10Kg one year after delivery.[18]

2.8 Drug-induced weight gain

Several drugs can cause weight gain, including a variety of psychoactive agents and hormone.[19] Most antipsychotics (phenothiazines and butyrophenones) often cause weight gain. The tricyclic antidepressant amitriptyline is particularly likely to cause weight gain and to increase carbohydrate preference. Lithium also has been implicated in weight gain. Valproate is an antiepileptic drug that acts on the N-methyl-Daspartate y-aminobutyric acid receptors and causes weight gain in more than half of the patient who receive it. Glucocorticoids cause fat accumulation, similar to that seen in Cushing's disease.[20]

2.9 Cessation of smoking

Weight gain is very common when people stop smoking and is at least partly mediated by nicotine withdrawal a gain of 1 to 2 kg in the first few weeks after a patient stops smoking is often followed by an additional gain of 2 to 3 kg over the next 4 to 6 month. Average weight gain is 4 to 5 kg but can be much greater. It has been estimated that smoking cessation increase the odds ratio of obesity compared with nonsmokers by 2.4 times in men and 2.0 times in women.[21] The differences in weight gain between those who quit and those who continue to smoke range from 2.6 to 5.3 Kg (3, 10, 18-24). Using data from 35 population-based prospective cohort studies worldwide and comparing 63,403 smokers who had quit smoking with 388,432 who continued to smoke, found that people who quit smoking gained an average of 4.1 Kg over 5 years, compared with 1.5 Kg for continuing smokers, a difference of 2.6 Kg.[22] In addition to increased weight, cessation can also result in an

increase in waist circumference or central fat, which could attenuate some of the beneficial effects of smoking cessation, especially among quitters who reduced their physical activity and those who had been heavy smokers.[23]

2.10 Sedentary lifestyle

A sedentary lifestyle lowers energy expenditure and promotes weight gain. In an affluent society, energy-sparing devices in the workplace and at home reduce energy expenditure even further and may enhance the tendency to gain weight.[24] Data from the centers for disease control (CDC) show that in areas of the united states where rates of obesity are higher than 30%. The prevalence of adults who repot no leisure-time physical activity is also higher than 30%. Likewise, the prevalence of obesity and physical inactivity predicts the presence of CVD death.[25] The sedentary nature of today's society makes many people spend a substantial amount of time per day on sedentary behaviors. In recent years there has been a rapid growth of evidence generation and gathering concerning the health impact of sedentary behavior and in particular on the relationship between obesity/overweight and sedentary behavior.[26]

2.11 Psychological and Social factors

Psychological factors in the development of obesity are widely recognized, although attempts to define a specific personality type that is associated with obesity have been unsuccessful. One condition linked to weight gain is SAD, which refers to the depression that occurs during the winter season in some people living in the North, where days are short. These patients, who tend to gain weight in the winter, can be effectively treated with exposure to high-intensity artificial lighting during winter months.[27] A follow-up survey was conducted of 325 ever-married women aged 20-54 years, systematically selected from 1998-99 national family health survey samples, who were re-interviewed after 4 years in 2003. Information on day-to-day problems, body image dissatisfaction, sexual dissatisfaction, and stigma and discrimination were collected and anthropometric measurements were obtained from women to compute their current body mass index. Three out of four overweight women (BMI between 25 and 29.9 Kg/m^2) were not happy with their body image, compared to four out of five obese women (BMI of 30 Kg/m^2 or greater), and almost all (95 percent) morbidly obese women (BMI of 35Kg/m^2 or greater) ($p< .0001$).[28]

2.12 Genetic and Congenital disorders

Discovery of the basis for the five single-gene defects that produce obesity in animals was followed by the recognition that these same defects, though rare, also produce human's obesity.[29] Modern genetic technology with precise definition of single nucleotide changes has advanced our understanding of the molecular mechanisms of weight regulation. Specifically, high throughput sequencing with whole exome, genome and targeted sequencing in individual subjects and cohorts of children with severe obesity has identified little known genetic aberrations.[30]

2.13 Hormonal causes

A relatively sudden increase in weight may suggest a neuro-endocrine cause. We screen for hypothyroidism, as this is associated with a modest weight gain. This is especially recommended if patients present with other symptoms such as dry skin, feeling cold. However, the weight gain in hypothyroidism seems mostly due to additional edema. Also, obesity is often associated with a slightly increased TSH that is most often the result of excess adipose tissue rather than the cause of obesity. This can be explained by the presence of peripheral thyroid resistance and also by increased levels of leptin, stimulating TRH and subsequently TSH.[31] Insulinoma is an extremely rare disease characterized by tumoral excessive insulin secretion, manifesting clinically by frequent hypoglycemias, inconstantly associated with neuroglycopenic and autonomic symptoms. Insulinomas are associated with obesity in 18–39% of individuals. Weight gain in these patients is believed to be related to excessive caloric intake in order to avoid hypoglycemia and is usually reversible after surgical treatment.[32]

3. CONCLUSIONS

When energy intake exceeds energy expenditure over time, then it leads to the conditions like obesity. Social change is undoubtedly the underlying reason for the rapid increase in obesity. Biological response to environmental change will determine who is likely to become obese. Solving the problem will require a multi-factorial approach understanding the biology of energy balance, and the underlying genetics. It may eventually help many patients with the development of more effective drug. Targeted behavioral interventions may help some patients and surgery a few patients. The greatest challenge will be to change our way of living so that weight gain does not become inevitable in a large proportion of the population.

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