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Review Paper

Obesity: An Introduction and Evaluation

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ABSTRACT

Obesity is a complex, multifactorial disease characterized by excessive body fat accumulation that has adverse effects on health and survival. It represents a fascinating interplay between genetic, metabolic, environmental, and behavioural elements, largely due to an imbalance among energy intake, energy expenditure, and energy storage. The prevalence of obesity has attained epidemic dimensions worldwide, with more than one billion adults and millions of children being affected, though it varies markedly from region to region. For instance, it has a strong association with a host of co-morbidities, which include type 2 diabetes mellitus, cardiovascular disease, hypertension, dyslipidaemia, cancers, osteoarthritis, and non-alcoholic fatty liver disease. Though the standard tool for classification remains the body mass index, waist-hip ratio, and other metabolic parameters are equally important in assessing the risk.This includes complex hormonal and neuroendocrine regulation of appetite and metabolism, including adipokines-leptin and adiponectin-along with central regulators such as neuropeptide Y and melanocortin pathways. Among the more important lifestyle factors that are major contributors are high-fat diets and physical inactivity, although there is also genetic susceptibility and emerging evidence of infectious contributors such as Adenovirus-36.Obesity, being a major health, social, and economic problem, calls for comprehensive management strategies that include dietary modification..

INTRODUCTION

Excess body fat accumulates in obesity, a medical condition that has a negative impact on life expectancy and health.[1] Genetic and environmental factors interact intricately in this chronic illness Genetic and environmental factors interact intricately in this chronic illness. High levels of fatty acids and cholesterol, an imbalance in metabolic energy, insulin desensitization, lethargy, gallstones, high blood pressure, dyspnea, emotional and social issues, and excessive adipose

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mass buildup with hyperplasia and hypertrophy are its hallmarks. [2] Evidence to support this view is provided by the fact that weight is gained by some obese people who eat little due to a slow metabolic rate[3][4]

Numerous secondary conditions, including heart disease, type 2 diabetes, breathing problems during sleep, cancer, and osteoarthritis, are linked to pathological obesity.[1] Exercise and food are the main treatments for obesity. Anti-obesity medications may be used to enhance this or, in the event of failure, to suppress appetite or prevent the absorption of fat. Within
In extreme cases, stool length and/or stomach volume are reduced through surgery or the placement of an intragastric balloon, which causes quicker satiation and reduces[5][6]

Worldwide prevalence:

Obesity is one of the leading preventable causes of death worldwide. [7] At least 300 million persons are clinically fat, and over 1 billion adults are overweight at the moment. Obesity rates today vary from less than 5% in China, Japan, and several African countries, to more than 75% in Samoan cities. In some places, childhood obesity is already a major problem, and in others, it is becoming worse. Globally, 17.6 million children under the age of five are thought to be overweight. The US Surgeon General claims that since 1980, the number of overweight children and adolescents in the country has doubled and tripled, respectively. Since the 1960s, the number of obese children between the ages of 6 and 11 has more than doubled. The prevalence of obesity among adolescents aged 12 to 17 has increased significantly between 1966–1970 and 1988–1991 in the USA, rising from 5% to 13% for boys and from 5% to 9% for girls. The issue is worldwide and is becoming more prevalent in emerging nations: The prevalence of childhood obesity in Thailand among children aged 5 to 12 increased in just two years from 12.2% to 15–6%. In many wealthy nations, 2-6% of all medical expenses are related to obesity. Since not all problems linked to obesity are taken into account in the estimates, the actual expenditures are definitely far higher. [8] IObesity is thought to be the cause of between 111,909 and 365,000 extra deaths annually in the United States and 1 million (7.7%) deaths in the European Union. [9, 10]

Classification:

A medical condition known as obesity occurs when extra body fat has accumulated to the point where it may be harmful to one's health. [11] Body mass index (BMI) is used to characterize it, and the waist-hip ratio and overall cardiovascular risk factors are used to further assess fat distribution. [12] Both the percentage and total body fat have a strong correlation with BMI. [13] A child's ideal weight varies depending on their age and gender. Children and adolescents are considered obese if their BMI is higher than the 95th percentile, which is determined by comparing them to a historical normal group rather than by an absolute figure. These percentiles are not impacted by the recent weight rises because the reference data they are based on spans the years 1963 to 1994. [7,14,15] BMI, which is usually given in either metric or US "customary" units, is computed by dividing the subject's mass by the square of their height. The US customary and imperial formula is BMI = lb * 703 / in2, where lb is the subject's weight in pounds and in is their height in inches (Table 1). The metric is BMI = kilos / meters2. The values in the table on the right are based on the most widely used definitions, which were created by the World Health Organization in 1997 and published in 2000. [2] The surgical literature divides "class III" obesity into other groups, the precise numbers of which are still up for debate. [16]

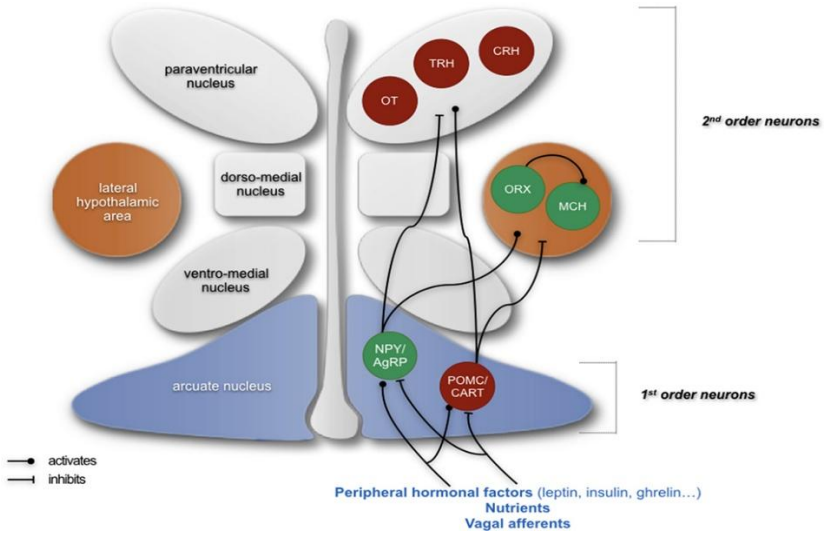
Classification of obesity based on BMI

Weight Status Category	BMI (kg/m²)
Underweight	< 18.5
Normal weight	18.5 – 24.9
Overweight	25.0 – 29.9
Class I Obesity	30.0 – 34.9
Class II Obesity	35.0 – 39.9
Class III Obesity	≥ 40

Some countries have redefined obesity; the Japanese have classified it as any BMI above 25 because Asian populations have adverse health effects at lower BMIs than Caucasians [17]
However a BMI of more than 28 is used in China. [18]

Mechanism:

The main cause of lipid and glucose-related metabolic dysfunction is obesity. Additionally, it promotes secondary problems such as reproductive, endocrine, digestive, pulmonary, hepatic, and heart dysfunctions. Anti-inflammatory and anti-atherogenic adipocyte hormones like adiponectin, visfatin, and acylation-stimulating protein counteract the atherogenic inflammatory, insulin-resistant, hypertensive, and thrombotic-promoting adipokines that are triggered, while some leptin and resistin actions are pro-atherogenic. [19] I It happens when energy expenditure and food intake are out of balance. Potential participation of Agouti gene-related peptide (AGRP), neuropeptide Y (NPY), melanocortin hormone (MCH), and orexin-A The following substances have been found to occur during the regulation of food intake: -B, galanin, α-MSH (α-Melanin stimulating hormone), CRF (corticotrophin releasing hormone), CART (caffeine and amphetamine releasing hormone), glucagonlike peptide-1 (GLP-1), CCK (coli cystokine), 5-HT (5-Hydroxy triptamine), insulin, and leptin. [20, 21] Additionally, it exacerbates metabolic syndrome and causes immunological dysfunction through the release of inflammatory adipokines. Genetic and molecular analyses of Numerous genes that may cause or contribute to the development of obesity have been found in animal models. The peripheral and central regulatory cascades that govern energy intake and expenditure have also been greatly elucidated by them. These include (i) the peripheral ones, which include insulin, leptin, gheralin, CCK, 5-HT [20], and (ii) the central ones, which include NPY, AGRP, α-MSH, Orexin, CART, and MCH [21]. [Fig 1] Genes and mutations connected to or associated with obesity, body fat distribution, and other pertinent characteristics have been discovered through genetic studies of families and populations. [22]



Co-morbidities associated with obesity:

Obesity predisposes individuals to various co-morbid health conditions, both physically and mentally. The metabolic syndrome, which includes type 2 diabetes, high blood pressure, high blood cholesterol, as well as high levels of triglycerides, is where the co-morbid conditions manifest more likely. [23] The complications associated with obesity, whether direct or indirect, involve various factors that have a common etiology, such as a poor lifestyle or diet.

Excess body fat causes 64% of diabetes cases in men and 77% of cases in women.[24] Under health effects, it is either brought on by high levels of fat mass, such as osteoarthritis, obstructive sleep apnea, stigmatization, or brought on by an increase in fat cells. These are diabetes, cancer, cardiovascular disease, or non-alcoholic fatty liver disease. Increases in body fat contribute to insulin response. It could therefore lead to insulin Ghrelin levels are decreased by eating, and this effect is more noticeable when carbs are consumed than when protein or fat are.[25 resistance. It brings about a pro-inflammatory state and a prothrombotic state, as explained by Shoelson SE. [24, 26 Data collected from extensive American and European studies indicated that the mortality risk is low among nonsmokers[25] with a BMI of 22.5-25 kg/m2, while it is found in individuals who are smoking, given a BMI of 24-27 kg/m2. [27, 28]

Co-Morbidities associated with Obesity:

Leptin:

• Leptin, which is secreted from adipose tissue, mainly works within the hypothalamus to curb neuropeptide Y and consequently hunger.

• Increased lipid levels in the blood encourage the secretion of leptin. Leptins' levels are directly proportional to the amount of adipose tissue in the body.

• Although mealtime causes secretion of Leptin, the sensitivity of the stimulant effect is comparative to the total body adipose tissue. As a result, hunger drive reduction is present in people with normal or low bodily weight. However, it is less obvious in people who are overweight or have conditions of obesity as a result of high levels of Leptin. This is because subjects who have normal weights, together with exogenous Leptin administration, lose weight, although this is not observed in patients with obesity conditions.[29][30]

Ghrelin: It is primarily synthesized in the stomach and duodenum.[31]

• Ghrelin has higher levels in the fasting state and spikes before food intake, called the cephalic response.[32]

Cardiovascular System

Category	Condition / Factor	Key Points
Metabolic Disorders	Type 2 Diabetes	64% (men) & 77% (women) cases linked to obesity; insulin resistance
	Hypertension	Increased BP due to fat accumulation
	Dyslipidemia	High cholesterol & triglycerides
	Metabolic Syndrome	Combination of diabetes, BP, cholesterol issues
	Non-alcoholic Fatty Liver Disease	Fat deposition in liver
Hormonal Factors	Leptin	High in obesity but reduced sensitivity (leptin resistance)
	Ghrelin	Increased before meals; regulates appetite
Cardiovascular Diseases	Coronary Artery Disease	Risk ↑ 30% per 5 BMI increase
	Heart Failure with Preserved Ejection Fraction	Due to inflammation, lipotoxicity
	Atrial Fibrillation	5% risk ↑ per BMI unit
	Stroke	Higher BMI → higher stroke risk
Respiratory Disorders	Obstructive Sleep Apnea	Breathing interruptions during sleep
Musculoskeletal Disorders	Osteoarthritis	Due to excess body weight
Cancer Risk	Cancer	Increased risk due to fat cell activity
Psychological Effects	Stigmatization	Mental stress, reduced quality of life
Inflammatory State	Pro-inflammatory State	Chronic inflammation
	Prothrombotic State	Increased clot risk
Infectious Causes	Adenovirus 36	Promotes fat cell formation
	Borna Disease Virus	Affects hypothalamus → obesity (animals)

Coronary artery disease:

The occurrence of coronary artery disease (CAD) is commonly linked to diabetes, hypertension, dyslipidaemia, and OSA. These conditions are well-established and common comorbidities of obesity. However, the exact role of obesity in the progression of CAD is uncertain. Studying the relationship of obesity with CAD is difficult because patients with obesity have high proportions of metabolic and respiratory disorders. However, with an increase of 5 units in BMI, the risk of patients developing CAD rises by 30%. [33][34][35]

Heart failure:

Obesity has been directly correlated with congestive heart failure with preserved ejection fraction. Although the risk factors for heart failure are commonly found in the obese population with congestive heart failure, the incidence rates are found to be higher when risk stratified for obesity.

The theorized mechanisms for the development of HFpEF include various elements, which when combined precipitate the development of the condition. These mechanisms are described as including left ventricular hypertrophy, which is caused by enhanced cardiac output, leading to decreased myocardial function through diastolic dysfunction as a result of lipotoxicity, systemic inflammation, and compromised protein function brought on by elevated glycosylation and collagen cross-links. These effects may also be exacerbated by elevated epicardial fat and inflammation.[36] [37]

Atrial fibrillation:

The most prevalent cardiac arrhythmia linked to the onset of heart failure and thromboembolism, atrial fibrillation also increases the risk of heart failure, albeit less than that of metabolically unhealthy obese and healthy people. Theperhaps resulting in potentially fatal infarctions and strokes in other parts of the body. The Framingham Heart Study showed that atrial fibrillation is common in obese people, with a 5% increase in the incidence of atrial fibrillation for every unit rise in BMI, especially above 30 kg/m2. fat and atrial fibrillation are thought to be related because fat increases cardiac output, which raises pressures on the left side of the heart, especially in the left atrium. Diastolic dysfunction may make this effect even worse.[38] [39] [40]

Stroke: As BMI rises, there is a greater chance of stroke, including early-onset ischemic stroke. A nationwide survey research that found patients with metastatic conditions further supports this association. survey suggests that obesity independently serves as a risk factor for stroke development.[41][42]

Diet, physical activity and excess body weight:

Considering the compelling data linking high body weight to negative blood pressure, blood cholesterol, and blood glucose levels[43] The question, therefore, becomes whether cardiovascular disease and obesity are directly caused or whether it is the resulting augmenting effect on cardiovascular risk factors that is the actual culprit behind cardiovascular disease in europe Maybe, though, the actual factors in the diet which are creating the obesity are at the same time creating the increase in high blood pressure, high blood cholesterol levels, and increased blood glucose and lowered levels of HDL cholesterol. The precise demarcation and delineation of the actual effect and contributory roles of weight gain versus the other contributory factors such as low levels of physical activity and low-quality diets from the aforementioned factors are, however, difficult because all these factors are interlinked and integral with one another such that it cannot be precisely defined whether one or the other factor contributes more or less than the othersDietary fat does not have a selective metabolic effect on total energy balance; this is made evident by the fact that obese adults would not reduce body weight if they selectively reduce fat calories in the food that they consume, provided that total energy intake is precisely controlled. From research carried out by Stubbs, it was discovered that if, instead of a modest or a low fat diet, an individual was presented with a high fat diet of similar form, taste, and appearance, an individual would unknowingly ingest their normal amount (mass) of food.

However, they would not know that the content of fat within their diet is directly correlated to the amount of energy they will retain in subsequent days. If the individuals were more physically active, they did know that if they took in diets of different fat content, they ingested a similar amount of food. However, if they were even more physically active through exercises like walking and cycling, they just ingested enough energy to simply maintain energy balance if they had a 40% fat diet. However, they readily went into a positive energy balance as fat intakes were raised. .[44,45]

Dietary fat, weight gain and weight loss:

short-term intervention physiological studies chime with the observations of the impact of the extraordinary nutrition transition, which has affected most poor societies in the world over the last 25 years, where the fat content has escalated in association with economic improvements from about 5-15% up to 30- 35%.13[46]

induced Viral obesity:

Canine distemper virus- Canine distemper virus is a morbillivirus and is antigenically related to measles. However, CDV primarily infects dogs and other wild mammals, not humans.[47]

especially Adenovirus-36 (Ad-36), which have been shown to directly promote the differentiation of stem cells to fat cells (adipocytes) and increase levels of triglycerides in these cells. It has been associated with human obesity as research indicates that it causes obese people to have a higher BMI despite lower levels of cholesterol and triglyceride.[48]

Borna disease virus:

It is a neurotropic virus that also causes significant obesity in animal models (rats) by inducing inflammatory lesions in brain centers that modulate food intake and body weight regulation, such as the hypothalamus. Research studies show that hyperphagia, fat accumulation, and high triglycerides are developed by infected rats.[48]

Rats with BDV. Infected rats showed hypertrophy of the pancreatic islets, hypothalamic inflammation, and increased levels of triglycerides and glucose. The emergence of obesity depends on a number of variables in BDV-infected rats, including the virus strain, rat strain, and infection age. Subsequent studies comparing the impact of various BDV strains on obesity hypothesized that BDV is the cause of obesity.[49]

CONCLUSION

Obesity is known to be a complex multifactorial chronic disease with complex determinants. It is a critical issue in the area of global health due to its increased prevalence and the strong association it has with various metabolic, cardiovascular, as well as psychiatric conditions. To be able to address this health threat, it is vital to consider and undertake an integrated approach that considers lifestyle changes, medical management, and even surgical interventions. There is a need for much more investigation of the molecular, environmental, and infectious etiologies.

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