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REVIEW OF LITERATURE OF OBESITY

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Abstract

Overweight was defined as a BMI ≥23 kg/m2 but <25 kg/m2 for both genders (based on the World Health Organization Asia Pacific Guidelines) with or without abdominal obesity (Abdominal obesity). WHO reports indicate that 48% of women and 41% of men in high-income countries do not get enough physical activity, and that physical inactivity causes almost 3.2 million deaths each year. Although obesity is clearly associated with an increased risk for diabetes, coronary heart disease (CHD), degenerative joint disease, and a number of cancers, there has been controversy over the relationship between BMI and mortality. Globally, the prevalence of obesity has almost doubled between 1980 and 2008, and increases have occurred in every region evaluated by the World Health Organization.

Key words- overweight, BMI, WHO, CHD.

General description of obesity

The term 'obesity' generally refers to an excess of body fat, but most information regarding the effects of obesity on health depends on measuring body weight. Body mass index (BMI), calculated as weight (in kilograms) divided by height (in meters), is a common measure of adiposity because it is readily measurable and correlates with adverse outcomes such as vascular disease and diabetes.(1) Weight gain is a global health problem, and those who are affected require treatment from multidisciplinary teams, including specialists in mental health, medicine, and sometimes even surgery. Obesity has multiple causes, but overconsumption plays a fundamental role. This kind of eating becomes compulsive and out of control when it becomes a "food addiction," a label that has caused a great deal of clinical and scientific discussion.(2)

Obesity is caused by overeating and sedentary lifestyles. Obesity severely affects blood glucose, blood pressure, and lipid metabolism and affects insulin action on lipid-free glucose metabolism. Those are the first signs of a cluster of conditions known as the metabolic syndrome (dysglycemia, dyslipidaemia, hypertension, and procoagulation).(3) However, recent studies have shown that obesity prevalence is increasing in developing countries in the same manner as it is increasing in developed countries.(4)

The World Health Organization maintains a database of obesity prevalence rates around the globe. Despite the fact that prevalence rates differ from one country to another, the organisation estimates that over 1.7 billion people are overweight and 310 million are obese around the world. The problem of obesity is becoming more prevalent in many developing countries. In the last 20 years, obesity has tripled in the developing world, with 10% of the world's children being overweight or obese. Middle East, Pacific Islands, Southeast Asia, and china are facing the greatest challenges.(5) According to the World Health Organization (WHO), at least 400 million adults worldwide are obese and this number is expected to double by 2015. Although obesity has traditionally been associated with high calorie intake and low physical activity levels in Western, a high income country, the burden of these conditions is increasingly felt in low and middle income countries.(6)

As one of the most important, yet preventable health hazards, obesity has now reached epidemic proportions with over 25% of the US population obese, and 15% in Europe.(7) Globally, the 2002 World Health Report lists the top ten selected risk factors as percentage causes of disease burdens as measured in disability-adjusted life years (DALYs) for high- and low- mortality developing countries and for developed countries. Overweight was regarded as the fifth most serious risk factor in developed countries and low- mortality developing countries. Other risk factors, such as tobacco consumption, hypertension, and underweight, rank is high.(8)

The obesity epidemic can be considered the first wave of the NCDs (Non- communicable diseases) collectively known as -New World Syndrome, creating a tremendous socio-economic burden and health burden including type 2 diabetes, hypertension, dyslipidaemia, cardiovascular diseases, obstructive sleep apnoea, musculoskeletal disorders, and some cancers.(9) According to the World Health Organization (WHO), obesity has reached epidemic proportions worldwide with one billion overweight adults and 300 million clinically obese. According to the World Health Organization (WHO), obesity has reached epidemic proportions worldwide with one billion overweight adults and 300 million clinically obese. According to the World Health Organization (WHO),

obesity has reached epidemic proportions worldwide with one billion overweight adults and 300 million clinically obese.(10)

Etiology:

Obesity is the result of genetic, behavioural, environmental, physiological, social, and cultural factors that result in energy imbalance and promote excessive fat deposition. The relative contribution of each of these factors has been studied extensively, and although genes play an important role in the regulation of body weight, the World Health Organization Consultation on Obesity21 concluded that behavioural and environmental factors (e.g. sedentary lifestyles combined with excessive energy intake) are primarily responsible for the dramatic increase in obesity in the past two decades.(11)

According to Stunkard et al's studies of twins, the concordance rates for various degrees of overweight were twice as high for monozygotic than dizygotic male twin pairs at age 20 years.(12) In another classic study of adult adoptees, a strong correlation was found between the adoptees' weight and their biological parents' BMI, while no such relationship was found for their adoptive parents. These results highlight the importance of genes, while signifying little or no environmental contribution.(13) Maes et al estimated that genetic factors account for 50% to 90% of the variability in BMI.(14) In another study, Bouchard et al demonstrated that the amount of weight and fat gained in response to overfeeding, as well as the distribution of fat gained, were more similar between twin pairs than among them, supporting the theory that a tendency to become overweight or obese is hereditary.(15)

The regulation of energy expenditure is one of the mechanisms by which genotype affects body weight. It is estimated that approximately 40% of the variance in daily energy expenditure (excluding vigorous physical activity) is attributable to genotype.(16) Genotype-environment interactions also have been implicated in the development of obesity.(17) The development of obesity is dependent on an imbalance between energy intake and energy expenditure during an extended period of time. Energy intake in excess of energy expenditure may be causing weight gain, or low energy expenditure in comparison to daily energy intake may be the cause. In adipose tissue, excess energy is stored as triacylglycerol. The primary functions of adipocytes are to store energy when calories are in excess and to mobilize energy from this triacylglycerol reservoir when energy needs exceed intake (eg, during dieting and starvation).(18)

When continued for decades, an imbalance of even 10 surplus calories per day will lead to weight gain of 0.45 kg (1 lb) per year. Weight gain during adulthood is characterized predominantly by adipocyte hypertrophy, a process by which adipocytes can increase their volume several thousand folds to accommodate large increases in lipid storage. In an evolutionary context, the ability to store excess energy in adipose tissue was essential for survival, because energy could be drawn from this storage depot in times of famine.(19) Diet also impacts leptin secretion; short-term food restriction leads to lower concentrations of leptin,(20) while resuming normal eating habits increases the concentrations.(21) Obesity is largely influenced by dietary patterns. Despite an increased focus on nutrition, a heightened awareness of the energy and fat content of foods, and the availability of various reduced-fat, fat-free, and sugar-free foods and beverages, obesity continues to increase.(19) A family history of obesity as well as genetic factors contributes to obesity development. Through the mechanism of thrifty genes, humans have evolved developing the ability to deposit fat and thus utilize it during periods of energy deficit.(22)

Childhood obesity is broadly classified as exogenous or endogenous, based on etiology. Exogenous obesity is caused by a chronic imbalance between energy intake and expenditure, whereas endogenous obesity is caused by genetic, syndromic, and endocrine factors, metabolic programming, as seen in children born small or large for gestational age, infants with diabetes, and those with rapid or excessive growth in the first few years of life, becomes exacerbated by lifestyle and dietary factors leading to obesity.(23) Parenting styles and behaviours, as well as parents' diets and physical activity patterns have a very strong influence on children's food and exercise choices of the child. There is increased risk of childhood obesity with maternal diabetes, smoking and obesity, underlining the role of foetal environment. Infant feeding style, sleep duration, rate of postnatal weight gain and age of adiposity rebound are also associated with obesity later.(24)

Hypothyroidism with elevated TSH and low T4 levels is typically associated with mild weight gain (an increase in BMI by 1-2 kg/m2). BMI gain is caused by decreased resting energy expenditure, fluid retention, and diminished linear growth Subclinical hypothyroidism is observed in roughly 10% of overweight/ obese children, but is considered as a consequence rather than cause of obesity and does not require treatment with thyroxin.(25) ROHHADNET (Rapid Onset obesity, hypothalamic dysfunction, and Hypoventilation, Autonomic Dysregulation and Neuroendocrine tumours) syndrome: This syndrome cause of childhood obesity manifests as accelerated weight gain and decelerated linear growth between 2 and 4 years of age, coupled with

autonomic dysfunction and hypoventilation. It is associated with varying degree of involvement of the hypothalamic-pituitary axis, including GH deficiency with low IGF-I in some, glucocorticoid deficiency or excess, hypo gonadotropic hypogonadism, hyperprolactinemia, hypothyroidism abnormalities of water and sodium homeostasis, adrenal tumours etc.(26)

Presently, obesity is epidemic but It is essential to identify underlying aetiology, modify lifestyle factors, assess current and future obesity related complications, and assess the patient's or family's readiness to make behavioural changes.(27)

Incidence and prevalence of obesity

Globally, the prevalence of chronic, non-communicable diseases is increasing at an alarming rate. Each year, more than 18 million people are affected by cardiovascular disease, which is caused by diabetes and hypertension. Propelling the upsurge in cases of diabetes and hypertension is the growing prevalence of overweight and obesity — which have, during the past decade, joined underweight, malnutrition, and infectious diseases as major health problems threatening the developing world.(5)

More than 1.1 billion adults worldwide are overweight, and 312 million of them are obese. Furthermore, 155 million children around the world are overweight or obese, says the International Obesity Task Force. This task force and the World Health Organization (WHO) have revised the definition of obesity to adjust for ethnic differences, and this broader definition may reflect an even higher prevalence — with 1.7 billion people classified as overweight worldwide.(5)

In the world, India is the second most populous country with 1.2 billion people, experiencing a rapid epidemiological transition at the moment. Under nutrition due to poverty which dominated in the past, is being rapidly replaced by obesity associated with affluence.(5) The National Family Health Survey-3 (NFHS-3) reported that in India, obesity (BMI ≥ 25 kg/m2) was more prevalent in urban areas and higher socioeconomic groups than in rural areas, especially among women (Men- urban:15.9 vs. rural: 5.6%; Women- urban: 23.5 vs. rural: 7.2%).(28) According to the NFHS-3 data, in the three States studied, the percentage of women who were obese (BMI≥ 25kg/ m2) was highest in Tamil Nadu (24.4%), followed by Maharashtra (18.1%) and Jharkhand (5.9%) and a similar order was reported among men in the three States with 19.8, 15.9 and 5.3 per cent being obese respectively.(28)

The Jaipur Heart Watch studies (I-IV) conducted in India in rural and urban areas reported that generalized and abdominal obesity were significantly higher among the urban compared to rural population.(29) Based on a study conducted among middle-aged women in four urban and five rural locations in the northern (Haryana), central (Jaipur), western (Pune), eastern (Kolkata), and southern (Kochi, Gandhigram) regions of India, age-adjusted prevalence of obesity in urban vs rural was reported to be45.6 vs. 22.5 per cent and abdominal obesity to be 44.3 vs. 13.0 per cent, respectively.(30)

Classification and assessment

Diagnosis of obesity, in clinical practice, three simple measures of obesity are commonly used; BMI, waist circumference (WC), and waist-to-hip circumference (WHR). A method of determining thinness and fatness is BMI, a ratio of weight in kilograms divided by height in meters squared (kg/m2). It has been correlated to morbidity and mortality risk in various populations.(31) There are two easy-to-use criteria for determining abdominal obesity; WC (waist circumference) and WHR (waist-to-hip circumference). Though BMI, WC or WHR correlate well with each other, it is also believed that combined use of these parameters of generalized and abdominal obesity may be better in identifying people at risk of CVD than either of them alone.(32)

The body mass index (BMI) is the most commonly used method for diagnosing obesity in primary care and subspecialty settings. Population- based studies have proven the metabolic consequences of having a BMI≥25kg/m2 and the mortality risk of a BMI ≥30kg/m2.(33)(34) BMI (kg/m2) is an easily obtained and relatively reliable measurement for overweight and obesity, rather than using complicated age- and height-specific criteria. If weight is measured in pounds and height in inches, the BMI is calculated as [weight/ height2] 703. This index gives body mass corrected for height for a wide range of heights and is a good approximate estimate of the fat content of the body. It is important to consider whether indices of overweight predict body composition because it is body composition (i.e., adiposity) rather than excess body mass that represents the greatest health risk.(35)

According to WHO and latest guidelines for Western populations, obesity is defined as a BMI ≥30 kg/m2. This classification is based on the higher risk of mortality associated with a BMI of 30 or higher. In the same guidelines, overweight is classified as BMI 25.0 to <30 kg/m2 , normal weight ranges from a BMI of 18.5 to <25 kg/m2 and a BMI below 18.5 kg/m2 is considered underweight.(36)

Autonomic nervous system and obesity

The autonomic nervous system (ANS) plays a major role in the integrated regulation of food intake, involving satiety signals and energy expenditure: thus ANS Dysregulation might results in body weight gain. Conversely, obesity might trigger alterations in the sympathetic regulation of cardiovascular function, thus favouring the development of cardiovascular complications and events Body weight is regulated by a complex homeostatic system, whose main components are the modulation of hunger and satiety and the modulation of energy expenditure and energy storage in the adipose tissue. As a result, the body weight is maintained by a homeostatic system that relies on a network of signals conveying information from the periphery to the central nervous system (CNS), where these signals are integrated and contribute to long-term and short-term body weight regulation.(37)

The sympathetic nervous system plays a pivotal role in regulating metabolic control. Recent research indicates that excess weight gain, especially when associated with increased visceral adiposity, leads to increased SNS activation and that this activity contributes to the development of hypertension in obese humans. The increase in arterial pressure caused by excess weight gain may be mediated, at least in part, by increased SNS activity, but the reason for this link between obesity and renal sympathetic activation remains unclear. Among the factors proposed to contribute to increased SNS activity in obesity are hyperleptinemia, activation of the central nervous system (CNS) melanocortin system, hypoadipo nectinemia, hypoghrelinemia, hyperinsulinemia or insulin resistance, increased angiotensin II levels, and baroreceptor dysfunction. Melanocortin-4 receptor (MC4R) is a key melanocortin receptor involved in appetite regulation, metabolism, and cardiovascular function.(38)

Activation of the baroreflex controls SNS activity in acute fluctuations of blood pressure, but its role in the long-term control of blood pressure is less clear In obese humans, baroreflex function is impaired, and the degree of impairment appears to be associated with body fat distribution: those with abdominal obesity have greater reduction in baroreflex sensitivity than those with peripheral obesity.(39)

A major function of the sympathetic nervous system is the regulation of resting metabolism and the initiation of thermogenesis as a response to physiologically relevant stimuli, namely changes in energy states, food intake, carbohydrate consumption, hyperinsulinemia and exposure to cold. Activation of sympathetic nerves

innervating the liver, pancreas, skeletal muscle, and adipose tissue can also elicit acute catabolic responses (i.e., glycogenolysis and lipolysis). The link between obesity and sympathetic over activity is further strengthened by the observation that weight loss in obese individuals causes a marked decrease in muscle sympathetic nerve activity and increase in muscle sympathetic nerve activity following weight gain.(40)

Circadian rhythm and obesity

Circadian rhythms are driven by an endogenous timekeeping system (i.e., the circadian clock) that has a periodicity of about 24 hours.(41)(42) These circadian clocks control many aspects of the organism physiology and they are influenced by external stimuli such as light, food and temperature.(43) Circadian rhythms/clocks have the primary function of anticipating changes in the external environment, which allows them to maintain homeostasis and provide adaptable physiological responses to the fluctuating environment. For example, gene expression, transcription factors, signalling pathways, hormone secretion, energy metabolism, growth, and behaviour are rhythmically coordinated by the circadian system.(44)(45)

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