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OBESITY AS LIFESTYLE DISEASE AND HEALTH CONCERN

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Abstract
Obesity is rapidly becoming an emerging disease in developing countries due to the increasing westernization of societies and change in the lifestyle. The etiology of obesity is said to be multifactorial, with a combination of genetic and environmental factors. Literature has been extensively reviewed to provide a broad overview of obesity. Data for this review were obtained from original articles, review articles and textbooks. Internet search engines were also employed. The years searched were from 1993 to 2008. Obesity, classified in terms of the body mass index and the waist–hip ratio, has several associated co-morbidities such as diabetes mellitus, hypertension, degenerative osteoarthritis and infertility. In Nigeria, there is limited information on obesity. A literature review on obesity is necessary to improve the knowledge about obesity in developing countries, its prevention and its management.

1. INTRODUCTION

The Japanese lifestyle has undergone remarkable changes as Japan achieved enormous economic growth. Westernization of the diet is an example of such changes. Another recent concern has been the decrease of physical activity l) One more important change that cannot be overlooked is that people have become exposed to various stresses at school, work, and/or home. These changes have given rise to various diseases, particularly those that are considered to be closely related to lifestyle. The increasing prevalence and mortality due to these diseases have become a matter of concern. (1)
"Obesity" as discussed in this article, is a lifestyle-related disease and is also a risk factor for the development of other lifestyle-related diseases, including diabetes, cardiovascular disease, and cancer. In Japan, the prevalence of obesity and the BMI* have been increasing steadily in all populations, except for women in their 20s to 30s. Prevention of obesity through modification of lifestyle is an important goal because it would also assist in the prevention of other lifestyle related diseases. In this article, risk factors for the development of obesity, particularly those related to lifestyle, are discussed (2).
Obesity is a non-communicable disease which is gaining increasing importance globally and is a rapidly emerging disease in the developed world. It is a chronic condition characterized by an accumulation of body fat. (3) Obesity is one of the most important preventable diseases in developed countries. The prevalence of the disease is increasing in both industrialized nations and in those undergoing alterations in diet and activity patterns as a consequence of adoption of the western culture. (4)

Currently, the health is a major concern in developed as well as developing nations where industrialization and globalization are increasing at a tremendous rate. Children as well as adults are being embattled by various junk food enterprises for their profits proceeding to various health issues amongst every age group. With the growth of privatization, young adults are lured by large pay-scales and in response; they are enforced by these companies to work for hours sitting at one place. This work gives rise to work-related stress which adds more to the deterioration of physical and mental health. The feeling of discontent that arises due to such stress leads to imbalanced eating habits at odd hours. Since a large number of occupations involve desk-job the lifestyle of people is at stake directing to a sedentary lifestyle. One such public health concern which is proliferating these days at an alarming rate is being overweight and then drifting towards being obese. [(5)]

The prevalence of obesity, which is defined as a body mass index (BMI) of 30 kg/m2 or higher, has been increasing dramatically. According to the Behavioural Risk Factor Surveillance System, a cross-sectional telephone survey of noninstitutionalized adults ages 18 years or older conducted by the Centres for Disease Control and Prevention, the prevalence of obesity between 1991 and 1998 increased in all 50 states in the United States, in both men and women, and across all age groups. [(6)] Notably, only 4 states had obesity rates of 15% or higher in 1991, but by 1998, 37 states had exceeded that level. The National Health and Nutrition Examination Surveys (NHANES) show that the prevalence of obesity rose gradually from 1960 to 1980, but in the period from the second survey (NHANES II: 1976 to 1980) until the third (NHANES III: 1988 to 1994), it increased markedly, from 14.5% to 22.5% (2). The increase in obesity prevalence noted in the NHANES surveys was also evident in both men and women, across all age groups, and across race-ethnic groups (Figure 1). The highest prevalence of obesity was found among women of minority groups. The combined prevalence of overweight (BMI, 25 to 29.9 kg/m2) and obesity also increased dramatically, from 46.0% to 54.4% in the period from the second to third NHANES surveys, the rates of overweight and obesity were highest among black women and Mexican-American men and women. [(7)]

BMI values in the United States population do not exhibit a normal distribution; rather, there is an enormous burden of higher BMI values that reflect the large percentage of people who are overweight or obese. In NHANES III, the BMI distribution included 16.0% with values of 25 to 27 kg/m 2, 18.6% with values of 27 to 30 kg/m 2, 16.1% with values of 30 to 34.9 kg/m2 (class I obesity), 5.1% with values of 35 to 39.9 kg/m2 (class II obesity), and 2.9% with values in excess of 40 kg/m2 (class III obesity). [(8)]

Importantly, BMI values are strongly correlated with total-body fat content, indicating that the degree of obesity can be calculated simply from height and weight measurements. [(9)]

2.CAUSES OR MECHANISMS OF OBESITY

Obesity is a disease that has rapidly escalated over the past several decades and is caused by environmental, humoral, and genetic factors, likely working in combination. The environmental factors contributing to the increase in obesity include but are not limited to decreased physical activity; increased television watching times and sedentary lifestyle. [(10)] increased food consumption, particularly of energy-dense, high-calorie, palatable food served in increasing portion sizes and the use of medications with weight gain as a side effect. [(11)] However, despite most

individuals being exposed to these environmental factors, not all of people become obese, suggesting differing genetic mechanisms that predispose certain individuals to developing obesity. Many genes have been identified as potentially contributing to obesity, possibly acting in combination; studies with twins have shown relatively high heritability for eating behaviours (53%–84%). [12] [13]

One of the most well-studied is the fat mass and obesity-associated gene, which exerts modest effects on its own and seems to be modified by lifestyle. Relatively few individuals have monogenic forms of obesity, although up to 200 types of single gene mutations have been found to cause obesity [14]. There are relatively few well-known monogenic mutations that explain no more than 10% of extreme obesity cases, such as mutations in leptin or the leptin receptor and the melanocortin-4 receptor [15]. Syndromic forms of obesity also make up a relatively small amount of clinical cases and are related to genetic disorders that include a distinct set of clinical phenotypes and also demonstrate obesity. For instance, some of the most common forms include WAGR (Wilms tumor, aniridia, genitourinary anomalies, and mental retardation), Prader-Willi, Bardet Biedl, and Cohen syndromes. Apart from genetics, certain other neuroendocrine causal factors for obesity include but are not limited to hypothyroidism, Cushing disease, pseudohy poparathyroidism, growth hormone deficiency, hypothalamic causes, and PCOS. Early referral to an endocrinologist and intervention is useful in patients suspected to have an underlying neuroendocrine or genetic cause for obesity. More recently, studies have also implicated epigenetic factors, such as changes in DNA methylation, microRNA expression, and noncoding microRNAs, as contributing to obesity [16]. Unlike with genetics, epigenetics are susceptible to change throughout the lifespan and with lifestyle modifications through diet and physical activity. As research continues to grow in these areas, one begins to understand the complex gene-environment interactions that contribute to obesity and how these may be targeted as treatment [17].

To understand obesity further, one must examine the central nervous system (CNS) circuitry that controls appetite and how this may become dysregulated through the Obesity as a Disease 3 gene-environment changes previously discussed. Although the oldest research focuses on changes in the homeostatic CNS control of eating in the hypothalamus, more recent research points to other networks, such as reward, emotion or memory, attention, and cognitive control as playing a more potent role in the control of appetite in humans [18]. The hypothalamus regulates homeostatic energy intake and expenditure, integrating hormonal signals from the periphery and communicating them to the rest of the CNS. For instance, leptin is secreted by adipose tissue and circulates proportionally to the amount of body fat mass, also responding to acute changes in energy deprivation [19]. At low levels of body fat, leptin circulates at lower levels and communicates with neurons in the hypothalamus to increase energy intake and decrease energy expenditure. in obesity, the opposite occurs and leptin circulates at high levels; however, leptin does not decrease energy intake and increase energy expenditure due to leptin resistance or tolerance, demonstrating a resistance to the homeostatic control of eating [20]. Thus, homeostatic control of energy intake is more critical in states of starvation. Increasing evidence demonstrates that other peripheral molecules may act on several areas in the brain, such as glucagon-like peptide 1 (GLP-1) and its analogues, which have been shown to act on the attention and reward networks [21]. Other molecules that are secreted by the periphery and may act in the brain have not yet been studied in humans, including amylin, pancreatic hormones, myokines such as irisin, and others. These may prove to be potential targets

for therapy. Aside from the homeostatic system, other neural systems may be more potent in terms of regulating appetite and obesity. The reward system, in particular, has been suggested to be at the root of obesity [22] [23]. Food is naturally rewarding and this system may be altered in patients with obesity, leading to and/or exacerbating weight gain. The 2 primary theories are that either a hyporesponsivity to rewards leads individuals to seek highly rewarding, high-fat or high-calorie foods, or there is a hyperresponsivity to food cues that leads individuals to increasingly eat highly palatable foods. These theories are supported by the observed lower availability of rewarding dopamine D2 receptors in individuals with obesity [24] [25]. and the heightened activity of brain areas responding to reward, such as the orbitofrontal cortex and nucleus accumbens, to visual food cues [26]. Emotions are also potent regulators of appetite because depressed mood and anxiety are comorbidities of obesity and related to central obesity in particular [27]. Indeed, stress is also known to cause changes in appetite that can lead to the development of obesity. Memory, regulated by the hippocampus, may also influence eating, and impaired functioning of the hippocampus leads to increased food intake and obesity, which in turn leads to further impairment of the hippocampus [28]. Thus, reward, emotion, and memory all may influence eating and the development of obesity. Higher CNS centers, such as those controlling attention and cognitive control, are also altered in obesity. Individuals with obesity and even normal weight individuals who later gain weight show more attention to food cues and attentional bias toward eating when sated [29]. Attention, controlled primarily by the parietal and occipital visual cortices, is generally increased for items of salience and in obesity these areas demonstrate increased activation to highly palatable food cues. Cognitive areas in the prefrontal cortex exert control inappropriate behaviors, such as eating when full or eating unhealthy foods. 78 Individuals with obesity and normal weight individuals who later gain weight have shown impaired inhibitory control toward food cues [30]. and even when performing tasks not related to food. Cognitive control may also suppress reward-related responses and, in the case of obesity in which cognitive control is impaired, this may enhance the activation of the reward system [31]. Altogether, the control of eating in the human brain is complex and involves several cortical and subcortical networks. Inflammatory links between obesity and insulin resistance (IR) or metabolic syndrome were suggested when increased tumor necrosis factor (TNF)-a expression was found in the adipose tissue of obese humans and rodents almost 20 years ago. Further research demonstrated involvement of multiple inflammatory pathways and increased cytokine levels in the mechanism of obesity and obesity-related IR [32]. Along the same lines, De Souza and colleagues98 found that rats subjected to long term high-fat diet (HFD) had increased activation of Jun N-terminal kinase (JnK) and Nuclear factor kappa B (NF-kB) inflammatory pathways resulting in increased cytokines (interleukin [IL]-6, TNFa, and IL-1b) in the mediobasal hypothalamic region. They further demonstrated that this inflammation led to significant impairment in insulin and leptin signaling pathways. These results have since been replicated by other investigators with consistent finds in mice and also other nonhuman primates. At the cellular level, HFD-induced inflammation involves reactive gliosis of the hypothalamus in rats. Reactive gliosis, which involves recruitment, proliferation, and morphologic transformation of astrocytes and microglia, is observed as early as 24 hours after starting an HFD diet in rats and resolves after 4 weeks of returning to a normal chow diet. However, prolonged HFD diet has shown to result in more significant and irreversible changes in hypothalamus, including gliosis, loss of synapsis in proopiomelanocortin (POMC) neurons, and reduction of neurogenesis in the hypothalamic region, leading to structural changes in the blood

brain barrier. Although studies in rodents have provided some critical insights, they may not fully capture the complexity of the human CNS and obesity [33].

3.CLASSIFICATION OF OBESITY

Obesity can be classified into central or peripheral obesity. In central obesity, otherwise called "android" obesity, the distribution of fat is commonly on the upper part of the trunk (the chest and abdomen) and is more common in the males. Android obesity is more clearly associated with disordered lipid and glucose metabolism and diseases like diabetes mellitus, gout, atherosclerosis, osteoarthritis, cardiovascular disease especially hypertension, and some cancers. However, in the peripheral or "gynecoid" type of obesity, the distribution of fat is mainly on the hip and thighs and is more common in females. Before the menopause, lipid assimilation is favored in the abdominal and femoral depots. However, after the menopause, these differences in fat metabolism between the abdominal and femoral sites disappear. The differences seen in the metabolism of fat suggest that female hormones are responsible before the menopause [34]

There are various measures of obesity, and the BMI is a very useful and common one. It is a mathematical formula that is highly correlated with the body fat. BMI is calculated as weight in kilograms divided by the square of the height in meters [35]. The BMI takes into account both frame size and body composition and is considered to provide a realistically achievable range of healthy weights and is a predictor of dangers associated with obesity. A BMI less than 18.5 kg/m2 is underweight. Normal BMI ranges between 18.5 and 24.9 kg/m2. A BMI greater than or equal to 25 kg/m2 is overweight. Obesity is defined as having a BMI of 30 kg/ m2 or more. There are three grades of obesity grade 1, which is a BMI equal to or greater than 30 kg\m2; grade II, which is moderate obesity with a BMI of 35–39.9 kg/m2; and grade III, which is extreme or morbid obesity, and the BMI is greater than or equal to 40 kg/m2 [36].

Waist–hip ratio (WHR) is another means of knowing if a person is obese and is calculated by using the ratio of waist circumference to hip circumference. Waist and hip measurements are simple and their expression as a ratio (WHR) makes it possible to do studies on a large number of obese individuals. Using this method of calculation of WHR, extreme gynecoid distribution has a WHR of about 0.5, whilst WHR is 1.0 in the android type. Another classification of WHR is 0.76–0.80 which is normal, 0.81–0.86 which is moderate obesity, while above 0.86 is severe obesity [37].

4. MANAGEMENT OF OBESITY

It is important to emphasize to the individual that management of obesity is lifelong. Health education and counseling have a remarkable role in encouraging adoption of lifestyle modifications. Health education should be offered to the individual about the etiology and treatment options for obesity. Referral may be necessary to other members of the multidisciplinary team, such as the physiotherapist, occupational therapist, the social worker, the dietician, and the clinical psychologist [38].

5. PATHOPHYSIOLOGY OF OBESITY

Ageing is associated with important changes in body composition and metabolism. Between age 20 and 70, there is a progressive decrease of fat-free mass (mainly muscle) of 40% with a relatively greater decrease in peripheral fat-free mass than in central fat-free mass, whereas fat mass rises with age. After age 70, fat-free mass and fat mass decrease in parallel. The fat distribution changes with age such that there is an increase in visceral fat that is more marked in women than in men. Also, fat is increasingly deposited in skeletal muscle and in the liver. The higher visceral fat is the main determinant of impaired glucose tolerance in the elderly. Increased intramuscular and intrahepatic fat contribute to impaired insulin action through locally released free fatty acids. Increased pancreatic fat with declining b-cell function also plays a role [(39).]

Because of the loss of skeletal muscle, the basal metabolic rate declines with 2% to 3% per decade after age 20, with 4% per decade after age 50, corresponding to approximately 150 kcal/d (630 kJ/d), and overall with 30% between age 20 and 70. This, together with a decreased intensity and duration of physical activity as well as decreased postprandial energy expenditure due to a decreased fat oxidation, accounts for the decreased energy expenditure with ageing [(40).]

Both obesity and ageing are characterized by a low-grade inflammatory state and by endocrine changes. Central and visceral obesity is more proinflammatory than global obesity [(41).] The low-grade inflammatory state is associated with decreased lean body mass, reduced immune function, cognitive decline, insulin resistance, and several correlates of metabolic control and insulin resistance such as increased levels of tumor necrosis factor (TNF)-a, interleukin (IL)-6, and C-reactive protein (CRP). TNF-a and IL-6 have catabolic effects on muscle mass and are involved in sarcopenia, a steady and involuntary loss of skeletal muscle mass with ageing resulting in decreased physical performance, mobility disability, and frailty [(42)(43).]

Endocrine changes related to obesity in the elderly include changes in gonadal steroids and thyroid hormones. Decreased growth hormone and insulin-like growth factor-1(IGF-1), leptin and insulin resistance, and downregulation of ghrelin are also present. The changes in hormones that occur with normal ageing seem to be exaggerated in the presence of abdominal obesity and insulin resistance [(44).]

6. OBESITY AND LIFESTYLE

6.1. Diet

The diet has considered as an important risk factor for obesity. With respect to the frequency of meals, it has been reported that the prevalence of obesity rises as the frequency of meals decreases.3) However, this issue does not yet seem to have been settled because some other studies have come to the opposite conclusion. In large-scale surveys performed in various parts of Europe and the United States, it has been demonstrated that the body weight increases as the total dietary calorie intake increases.

In large-scale surveys such as the National Health and Nutrition Examination Survey (NHANES) performed in the United States and other surveys performed in Germany, Scotland, and Denmark, the BMI or the amount of subcutaneous fat was higher in the high-fat diet group than in the low-fat diet group. In regional surveys performed in Tennessee and North California in the United States

and in Finland (Odds ratio (OR=1.7), the weight gain of the high-fat diet group was significantly greater than that of the low-fat diet group [45].

According to several large-scale surveys, including the European prospective investigation and the American Cancer Prevention Study (ACPS) II, the consumption of a large amount of meat results in weight gain. An investigation by Kahn et *al*. showed that the risk of obesity in relation to increased consumption of meat was estimated by an OR = 1.46) In contrast, the mean BMI of vegetarians is low. A survey performed on 10,000 subjects in Norway and the ACPS II (OR=0.81) have suggested that it is possible to reduce the BMI by eating a large amount of fruit and vegetables [46].

6.2.Exercise

A beneficial effect of physical activity on obesity has been demonstrated in many studies.

A study performed on 3,132 individuals at seven health centers delineated the association between exercise and obesity in the Japanese. This study showed that the prevalence of obesity was lower among individuals who were in the habit of performing exercise, and the risk of obesity in this group was low (OR=0.48) [47].

Many studies have shown that the prevalence of obesity, the mean BMI, or the body weight decrease as the amount of exercise increases. Among persons in their 20s from the Coronary Artery Risk Development in Young Adults (CARDIA) study, there was a significant association between an increase of exercise over 2 years and weight loss. The risk of weight gain was decreased by jogging (OR=0.57 in men) and aerobics (OR=0.59 in men), but was not significantly reduced by playing a team sport or tennis [48].

6.3.Stress

The direct association between stress and obesity is not so strong, but some reports have supported a direct influence of mental stress on the development of obesity. The CARDIA study, a higher Cook-Medley hostility score was significantly correlated with a higher waist-to-hip ratio. In the NHANES I study, people who gained weight were less happy than those who neither gained nor lost weight (OR for unhappiness:1.54 for obese vs 2.03 for non-obese). In many reports, however, stress was concluded to have no influence on the degree of obesity. Because the methods used to assess stress have varied among studies, direct comparison is difficult. In addition, the influence of other relevant factors, such as dietary habits, cannot be ignored [49].

6.4.Smoking

Many epidemiological studies performed in Europe and the United States appear to indicate that smoking reduces obesity. In all of these surveys, including a health survey performed in Ontario on 20,306 subjects, NHANES I and III, the CARDIA study, and a study on 1,911 pairs of monozygotic twins, current smokers were the leanest whereas ex-smokers the most obese. However, a 10-year follow-up study performed in the United States showed that the OR of smokers for an increase of BMI was 0.8, indicating no significant difference between smokers and ex-smokers. In Australia, the recent increase of BMI has been reported as not being attributable to the decreasing prevalence of smoking. Body weight appears to increase for several years after ceasing to smoke. Despite this, anti-smoking campaigns are still useful if the risk of smoking with respect to the development of

cardiovascular disease and cancer is considered. It appears necessary to provide appropriate measures for the prevention of weight gain when smokers are trying to quit the habit [50].

6.5.Alcohol

With respect to the association between alcohol intake and obesity, many large-scale studies have been performed, including the health study in Ontario, NHANES I, the study on monozygotic twins, and the ACPS. In NHANES I, the OR for weight gain by heavy drinkers (defined as intake of alcohol twice daily or more) was 0.9 when the risk for those who did not drink was set at 1 and there was no significant difference. In the study on monozygotic twins cited above (see "Smoking"), when the risk of obesity in subjects who did not drink was set at 1, the OR for heavy drinkers (alcohol intake of 0.99 ounces or more per day) was 1.43 and the OR for non-drinkers was 2.14, showing no significant difference [50].

6.6.Childhood obesity

There have been many reports that obese children are at high risk of becoming obese adults. The risk has been estimated as very high, with OR values of 2.0 to 6.7. Thus, prevention and management of childhood obesity is considered to be one of the mainstays for primary prevention of adult obesity. To prevent adult obesity, children should be encouraged to lead a healthy lifestyle. Obesity and hyperlipidaemia are likely to be the two major risk factors for lifestyle-related diseases at school age [51].

7.HEALTH CONCERN OF OBESITY

It is far from clear that which measure of adiposity best predicts the impact of obesity on health outcomes in the elderly. BMI, which correlates with body fat in the young and middle-aged, can either underestimate the degree of fatness in older people because of changes in body composition or overestimate it because of loss of height from vertebral compression and kyphosis. So, the relationship between BMI and disease risk is less close in the elderly than in younger people. Moreover, the effect of ageing on body fat distribution (increased omental and mesenteric fat and intramuscular and intrahepatic fat deposition) increases risks of insulin resistance. Waist circumference, which correlates highly with total fat and intraabdominal fat might better predict adverse health effects of obesity in the elderly but there are insufficient data to define appropriate cutoff values for the elderly [52].

The National Institute of Health (NIH) guidelines, published in 1998, suggested that a 70-year-old person with a weight of 64 kg and a height of 1.6 m (BMI 25 kg/m2) and one of the mentioned risk factors (such as established coronary heart disease, hypertension, impaired glucose tolerance, dyslipidemia, etc.) would be a candidate for weight loss. This was questioned by Heiat et *al*22 in 2001 and by Janssen and Mark in 2007. Heiat et *al*22 reviewed 13 articles that reported the association between BMI and all cause and cardiovascular mortality in nonhospitalized subjects older than 65 years with a follow-up of more than 3 years. They did not find support for overweight conferring an excess mortality risk and found a small relative mortality risk in higher BMI ranges (RR of 1.15 to 1.34 with BMI 28 to 29 kg/m2 and a RR of 1.31 to 2.0 with BMI 30 to 35 kg/m2). The relation between BMI and all-cause mortality was described as a U-shaped curve with a large

flat bottom and a right curve that started to rise from a BMI>31 to 32 kg/m2. Janssen and Mark23 performed a meta-analysis of 28 articles on the association of BMI and all-cause mortality in subjects 65 years and older with a follow-up of at least 1 year. They found that a BMI in the overweight range was not associated with an increased risk and that a BMI in the moderately obese range carried a modest increased risk [risk estimate 1.10 (1.06/1.13)], which was marginally higher for women [risk estimate 1.18(1.13/1.24) vs. 1.10 (1.02/1.18) in men]. So, the threshold value at which BMI confers mortality risk is higher in the elderly than for younger adults. Longitudinal studies published after these meta-analyses have confirmed this [53][54].
These findings suggesting that a higher BMI value predicts a lower relative mortality in older adults (the obesity paradox) should not mask the harm from obesity to the elderly. Although the RR of mortality and decreased survival seem to decrease at ages above 59, the absolute mortality risk increases with increasing BMI till age 75 [55]. Additional confounding factors contribute to underestimating the health risks of obesity in the elderly and the obesity paradox. These include the survival effect (the presence of "resistant" survivors in whom the relation between BMI and mortality is lost), competing mortalities, relatively shortened life expectancy in the old age, and the importance of age of onset and duration of obesity, as those who became obese in old age may die before the adverse effects of obesity become apparent. Also, smoking, weight change (weight gain and weight loss may be more detrimental than stable weight), and unintentional weight loss may confound the estimation of health risks. The underlying disease (reverse causation), physical activity and cardiorespiratory fitness (a lean unfit may have a higher mortality than an obese fit subject), fat distribution (unknown in many studies), and length of follow-up (as with shorter duration no association between obesity and mortality is evident) also play a role. Thus, the inverse relation between BMI and mortality (the obesity paradox) observed in a recent study of US veterans aged 40 to 70 years at entry could be explained by some of the abovementioned factors: reverse causation, veteran effect (becoming obese after discharge from the service), survival effect, and being a healthy obese.28 Furthermore, the high fitness state despite a high BMI may have biased the generalizability of these findings [56].

Medical complications of obesity in the elderly are mainly concentrated around the metabolic syndrome (with glucose intolerance, hypertension, dyslipidemia, and cardiovascular disease). The metabolic syndrome peaks at age 50 to 70 in men and age 60 to 80 in women with an odds ratio (OR) of 5.8 in 65-year-old men and 4.9 in 65-year-old women compared to 20- to 34-year-old subjects [57]. The metabolic syndrome is a recognized risk factor for stroke, but is also related to subclinical ischemic brain lesions, placing the subjects at risk for future cognitive impairment.32 A recent meta-analysis suggests the existence of a significant U-shaped association between BMI and Alzheimer disease and vascular dementia [58]. Obesity also increases the risk of heart failure, and estimates suggest that having a BMIZ30 kg/m2 doubles the risk.35 Waist circumference and percentage body fat also predicted this risk. Other obesity-related disorders are (osteo)arthritis (with an OR of 4.8 for men and 4.0 for women), pulmonary dysfunction including the obesity hypoventilation syndrome and obstructive sleep apnea syndrome, certain cancer types, reduced cognitive skills, sexual dysfunction, and urinary incontinence. Obesity may also contribute to cataract formation and the progression of age-related macular degeneration [59][60].

The obese elderly are also likely to have functional limitations because of decreased muscle mass and strength and increased joint dysfunction, disabilities of activities of daily living, frailty, chronic pain, and impaired quality of life [61]. Unintentional injuries such as sprains and strains occur more often. Obesity is an important risk for frailty either through increased levels of inflammatory markers or through sarcopenia (OR 3.5 in 70- to 79-y-old women) [62].
Obesity can have beneficial effects (through endocrine effects of insulin, leptin, and oestrogens on stimulating bone growth and inhibiting bone remodeling) that can result in preserved or higher bone mineral density, a lower risk of osteoporosis and hip fractures, and a cushioning effect of fat around the trochanter that can provide protection against hip fracture during a fall [63].

8. CONCLUSION

Genetic factors and social factors, such as inadequate availability of facilities for exercise and recreation, development of transport that reduces the distance that people have to walk, and instant availability of food over 24 hours whatever people want to eat, are other causes of obesity that are not discussed in this article.

The value of preventing obesity can be considered in relation to the following three factors. First, it can prevent the development of so-called lifestyle-related diseases. Second, a reduction of medical costs can be achieved by such prevention. Third, improvement in the quality of life can be achieved, both for individuals and at the population level.
To prevent obesity, individual and population-based health education needs to be provided so that people can obtain the basic knowledge necessary to establish a healthy lifestyle that does not lead to obesity.

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