Chapter

Obesity: A Prerequisite for Major Chronic Illnesses

Hafeez Abiola Afolabi, Zaidi Zakaria, Salzihan Md. Salleh, Ewe Seng Ch'ng, Siti Norasikin Mohd Nafi, Ahmad Aizat Bin Abdul Aziz, Sameer Badri Al-Mhanna, Ahmad Adebayo Irekeola, Yusuf Wada and Abubakar Bishir Daku

Abstract

Obesity is rampantly soaring at an alarming rate globally and simultaneously causing an increased incidence, and predisposition to various comorbidities. obesity is body mass index of >30kg/m^2, while <18kg/m^2 is underweight. The world at large fails to recognize obesity as an inevitable disease that requires strict measures to control this modifiable risk factor. W.H.O news release reported that over one billion people globally are obese among which 650 million were adults, 340 million were adolescents, and 39 million were children. The lowest obesity prevalence was reported in Timor Leste at 3.80%, Bangladesh at 3.60%, and Vietnam at 2.10% while the highest were noted in Nauru at 61%, cook island at 55.9%, and Palau at 55.3%. obesity is the most prevailing health problem (15% globally) associated with an increased propensity for development of several medical illnesses, obesity-associated adverse outcomes causing fatal complications that are difficult to manage, and premature mortality. The obese often feel they are not socially cared for by society and are accorded limited time by physicians who don't view their health concerns from their own perspectives. Thus, making them pessimistic from low self-esteem and discrimination, body shaming, and stigmatization. They eventually develop depressive-anxiety disorder because of distrust insight.

Keywords: obesity, overweight, body mass index (BMI), comorbid, chronic diseases, obesity prevalence

1. Introduction

Obesity is exponentially rising at an alarming rate and simultaneously causing an increased incidence of substantial adverse effects, and predisposition to various comorbidities such as osteoarthritis, coronary heart disease, and hypertension [1, 2]. Globally, obesity has precipitously grown to become a pandemic in all countries due to several contributing factors from vicissitudes in human lifestyles and societal norms, in addition to the multi-factorial existing causes of obesity: genetics, dietary intake,

and lifestyle modification [3–5]. Putting all these puzzles together, obesity represents a major healthcare crisis, both medically and economically around the globe [6–8].

Identifying and categorizing obese from the non-obese or even from the overweight that is deemed borderline between the non-obese and the obese individuals involve the classification based on Body Mass Index (BMI). Obesity classifications and definitions vary but, by and large, the definition by the World Health Organization (WHO) is commonly employed as the gold standard. It defined obesity as a Body Mass Index (BMI) of 30 kg/m^2 or more, 25–29.9 kg/m^2 as overweight, and 18.5–24.5 kg/m^2 as normal BMI while less than 18 are considered underweight [9].

2. Global epidemiology of obesity

On March 4, 2022, the news release theme by the WHO [10] on "World Obesity Day 2022" was "Accelerating action to stop obesity" but will it ever come about? Well, the answer is feasible! Less likely of course because the world has yet to recognize obesity as a disease or pandemic that is inevitable if strict measures are not taken to control the modifiable risk factors that are most probable factors. As such the WHO is encouraging countries to act more to overturn this foreseeable and escapable health crisis. According to the WHO news release, greater than 1 billion people globally are obese of which 650 million were adults, 340 million were adolescents, and 39 million were children [11]. More astonishing is the fact that this number is still increasing. WHO estimates that by 2025, about 167 million people: adults and children will become less healthy because of being overweight or obese [12]. Between 2011 and 2014, the gross proportion of the prevalence of obesity among adults in the United States was 36.5%, by 2016, the figure rises more, 38.9% of US adults were obese while 7.6% were classified as severely obese [13, 14]. Between 2017 and 2018, 41.9% of women 20 years and more of the US were said to be obese [15]. In 2016, 13% of adult globally were said to be obese [16]. Overall, middle-aged individuals 40–59 years old (40.2%) and older aged individuals 60 and above (37.0%) had a greater incidence of obesity than younger adults aged 20–39 (32.3%) [17]. Overall gender predilection shows that women (38.3%) had a greater propensity for obesity than men (34.3%) [18].

Heightened prevalence of obesity and overweight are being reported in several nooks and crannies of the globe: in the Middle East region, the prevalence of obesity and overweight among people 40 years old and above between 2000 and 2006 was 21.17% but increased to 33.4% between 2014 and 2020 [19]. In Europe, obesity prevalence is estimated at 60% among adults populations [20], approximately even higher than 60% in Asia [2]. According to the [21], over half of the British population were said to be overweight between 1980 and 1995, the prevalence of obesity in Britain also doubled from 8% to 15% in 2016, in fact it was projected that by 2050, 15% and 62% of females in social class I and class V will be obese [22]. Most of South America and parts of Asia reported it at 16.4% from 2015 to 2019 in China [23]. Country-wise according to the world population review, the highest obesity prevalence was recorded in countries such as Nauru 61%, Cook Island 55.9%, Palau 55.3%, Kuwait 37.9%. In the US, obesity prevalence has risen from 19.4% in 1997 to 31.4% in 2017 [24] and 36.20% in 2022 while the least was reported in Japan 4.30%, India 3.90%, Timor Leste 3.80%, Bangladesh 3.60%, and Vietnam 2.10% (full description in **Figure 1**).

Globally, the proportion of obesity is higher in women than in men by approximately 1.6-fold, at least to a certain extent, that a female higher occurrence rate would be anticipated, owing to the biologically higher ratio of body fat in women [25].

In some societies, the rate of obesity is higher in lower socio-economic classes, this can be attributed to the greater degree of fast-food restaurants in low-income districts, the higher price of healthful diets, safety fears that inhibit walking and other outdoor activities, and greater economic woes [26–28].

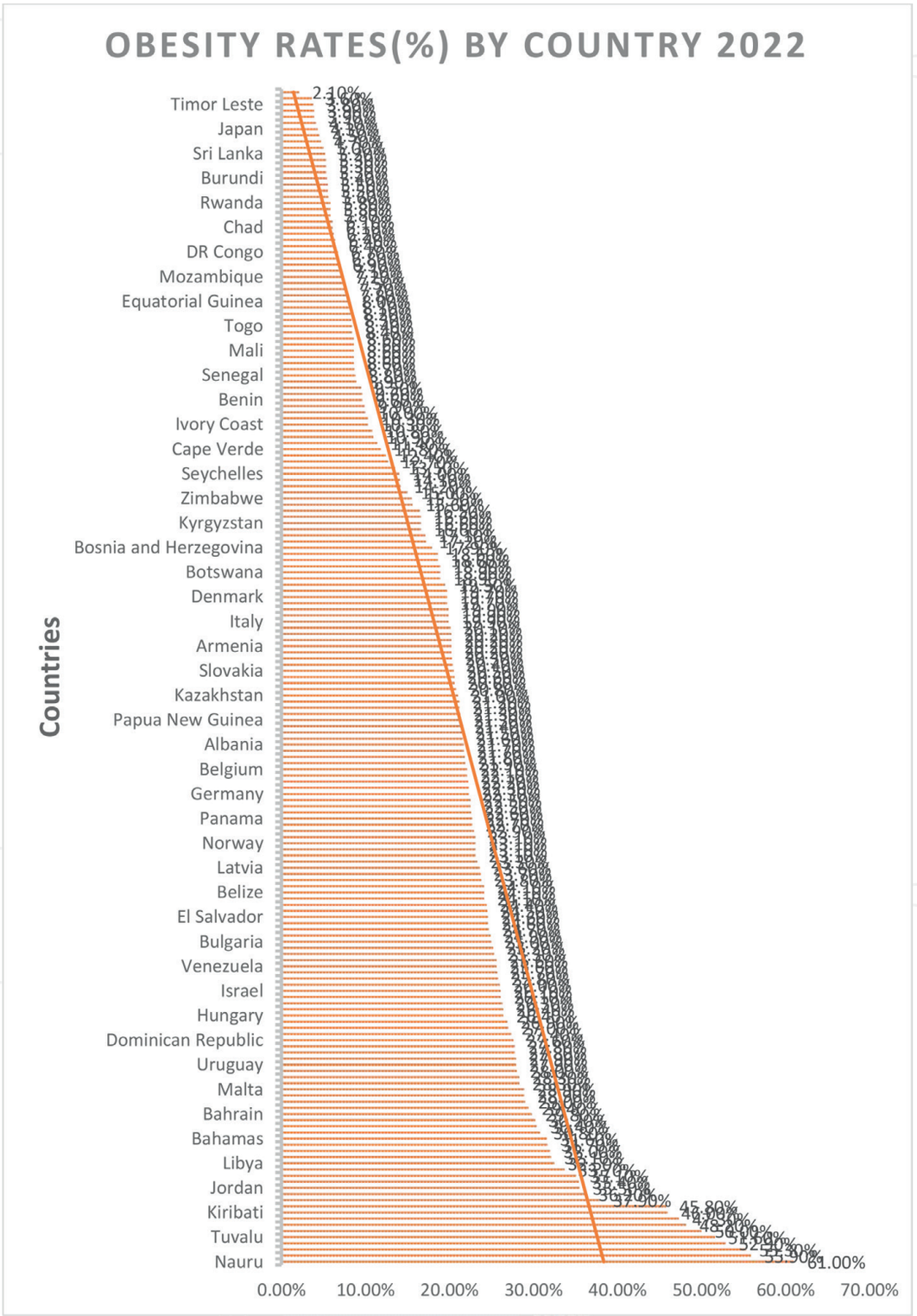

Figure 1.
Illustration of obesity prevalence by countries. (Obesity Rates by Country 2022: https://worldpopulationreview.com/country-rankings/obesity-rates-by-country).

3. Etiology of obesity

Not only does obesity affects 15% of the global populace, but it is also an underlying cause for several other chronic diseases, yet, surprisingly, it has until recently been regarded as a disease. The genesis of obesity is multi-factorial likewise the epidemic is almost certainly associated with rising sedentary daily life combined with increased desire for satiety satisfaction. However, this is not always so for every obese individual because there is substantial evidence of genetics: obesity-inherited link in the vast majority of traits.

Obesity in the form of weight gain is a continuous process resulting from the constellation of genetic, behavioral, environmental, physiological, social, and cultural factors that ultimately lead to energy imbalance and accumulation of excessive fat deposits [29, 30]. A heightened desire for taste satisfaction and abundant inexpensive, energy-dense readily available fast food increases energy consumption, because these promote feasting out of home, provides varieties of food options and large portions [31]. These eventually will lead to an imbalance between energy intake and energy output. The identification and understanding of the neuronal substrates mediating overeating (after consumption of a high-energy meal) explain the increased neural activity in the amygdala: the interstitial nucleus of the posterior limb of the anterior commissure (IPAC). The neurons in the IPAC system can be activated and switched on after eating or upon food smelling which eventually increases satiety in eating habits [32]. Being a complex system, the amygdala interface with other limbic structures to regulate emotion, learning, and behavior, through these interactions, it modulates and plays a role in the emotional-eating pattern that eventually promotes the overeating response [33, 34]. However, there are other mechanisms for increasing weight gain and many other mechanisms are still unclear.

Leptin, a neuromodulator protein formed predominantly in the white subcutaneous adipose tissue is a crucial body-weight regulator in the human feeding and satiety cycle [35, 36]. Leptin sends satiety impulses to the hypothalamus and thus decreasing food intake and fat storage while ensuring modulation of energy expenditure and carbohydrate metabolism, and finally averting excessive weight gain [37]. Most of the people living with obesity are not leptin-deficient but, they actually have a leptin-resistant condition. Thence, they have higher levels of circulating leptin. Women have higher leptin levels than their male counterparts, and these higher leptin levels have a direct relationship with potentially higher BMI in females.

Among other pathways are Hypertrophic-hypercellular obesity wherein the adipose tissues or fat cells increases in size and number and are manifested as abdominal obesity, commonly noticeable in adulthood [38, 39]. With some of these primary adipocytes-secreting pro-inflammatory products: Tumor necrosis factor–alpha TNF-α, Interleukin 6 IL-6, Monocyte chemoattractant protein–1 (MCP-1), etc. acting as active metabolites that support lipid storage, fatty acid synthesis and lipids exudate from adipocytes deposits [40, 41]. Effect of hormones, neurotransmitters, and neurogenic signals on feeding habits are also worth mentioning. Certain hormones such as endocannabinoids increases appetite, promote nutrient uptake, and promote lipogenesis. There are other varieties of gut hormones such as glucagon-like peptide-1 (GLP-1), neuropeptide YY (PYY), and cholecystokinin that act substantially to induce satiety to affect eating habits [42, 43].

The benefit here is that understanding these pathways also gives science possible therapeutic targets for obesity control. Ultimately, the occurrence or increase of obesity resides in an imbalance between energy intake and energy expenditure over a

lengthy period, therefore the etiology can be seen as surplus energy intake relative to daily energy expenditure, or as low energy expenditure relative to daily energy intake.

4. Obesity and other chronic disease pathophysiology

Obesity-related health conditions are numerous and associated with increased morbidity and greater mortality. The major obesity-related comorbidities include cardiovascular disease (majorly heart disease and stroke), type 2 diabetes, dyslipidemia, musculoskeletal disorders (osteoarthritis especially), and certain cancers (endometrial, breast, and colon) [44]. These illnesses are underlying causes for premature death and substantial health disability. Expert reviews by Chetambath et al. [45] indicated that a BMI of 25–28.9 kg/m^2 is associated with a relative risk of 1.72 for coronary heart disease the risk gradually rises with an increasing BMI; for BMIs more than 33 kg/m^2, the relative risk is 3.44. A similar trend was also reported between obesity and stroke respectively [45]. In general, obesity is reported to increase mortality rate from cardiovascular disease by 4-fold and by 2-fold for cancer-related death. Moreso severely obese (BMI ≥40) showed a 6–12-fold probability of an all-cause mortality rate, a reduced life expectancy by 20 years in men, and approximately 5 years in women [46–48]. Although there is no definitive or no cause-and-effect association that undoubtedly established obesity direct cause of these comorbidities, however, improvement of these illnesses after significant weight reduction indicates that high body mass index plays an important role in the propagation of the diseases [49, 50]. A schematic diagram is illustrated in **Figure 2**.

Hypertension (high blood pressure) is among the foremost prevalent comorbidity associated with obesity, and in turn is a crucial risk factor for stroke, myocardial infarction, heart failure, and chronic renal disease [51, 52]. Independent risk factors such as high body mass index and/or overweight are 65–75% linked to primary (essential) hypertension [53], with at least 72% contribution in hypertension and/or T2DM

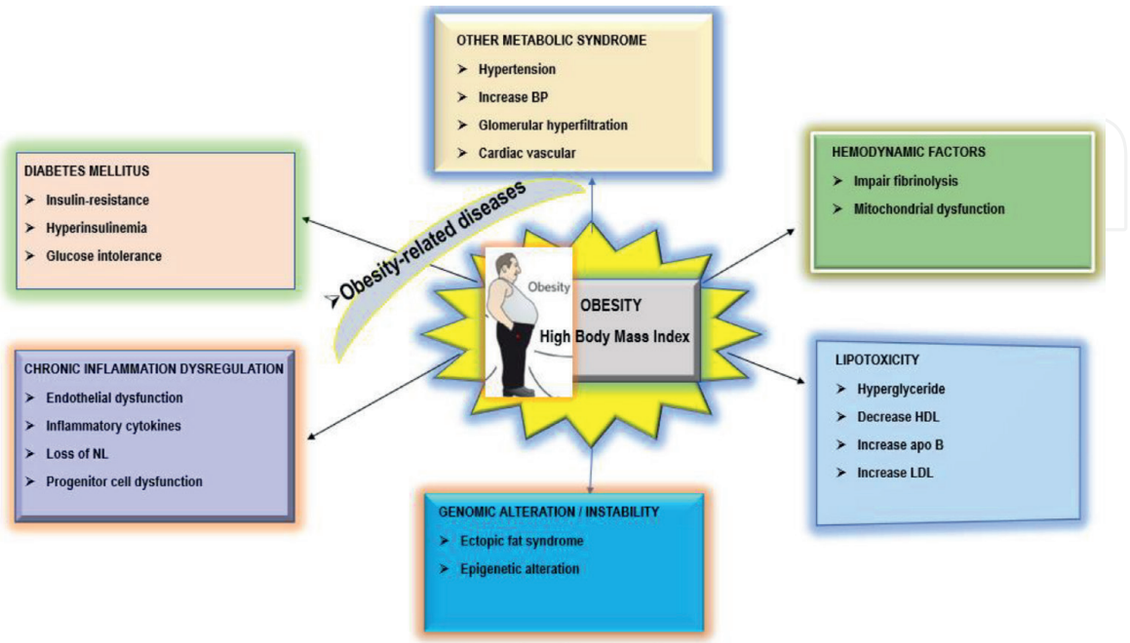

Figure 2.
Schematic diagram of obesity-related chronic disease.

diagnosed patients with end-stage renal disease, although the mechanism is yet to be fully understood [53]. Nonetheless, significant progress has been made in explaining some of the intricate relationships between renal, hormonal, and neurological system components that connect excessive adiposity with high blood pressure. Literally, the amount of blood flowing through the body increases with increased body weight, and this place additional pressure on artery walls, leading to increased arterial blood pressure (hypertension). Besides this, being overweight also increases the heart rate and thus makes blood flow within the blood vessel harder. Obesity generally reduces the parasympathetic pitch and intensifies sympathetic activity. These autonomic activity modifications cause increased heart rate, decreases heart rate variability, and reduced baroreflex sensitivity. According to the Nurses' Health Study in the United State, women who had a BMI above 32 had a four times higher mortality rate from cardiovascular disease than those who had a BMI under 19 [54–56].

Pulmonary function is also a prognosticator of various recurring illnesses even though various research outcomes on the obesity-lung function association are inconsistent. Nonetheless, a lot of research have reported central obesity as a reliable indicator of poor lung function [57]. Obesity is a serious mitigating condition that is frequently associated with respiratory problems, thus reducing the exercise-tolerance capacity of the obese perhaps due to early exhaustion. Obesity is the main culprit in obstructive sleep apnea, a condition that disrupts sleep, produces snoring and apneic episodes, and lowers oxygen saturation to levels associated with potentially fatal cardiac rhythms [58]. Due to modifications in respiratory mechanism and bronchospasm produced by gastroesophageal reflux illness, obesity makes respiratory symptoms such as dyspnea worse [59]. Obesity-lung function association by most studies has focused on the association between lung function and central obesity and central obesity indicators, however, the relationship is inversely purported while for some, the association is weak and for others, there is an independent obesity indicators-lung function interaction [59, 60]. Obesity especially central obesity restricts lung compliance function due to diaphragm-reduced movement (expansion-relaxation function) which eventually limits the respiratory functional capacity of the lungs leading to decreased lung function i.e. FVC and FEV1 in people with central obesity than when compared to the normal population [23]. An alternative possible mechanism is a reduction in the functional pulmonary capacity due to increased deposition of adipose tissues in the pulmonary compartment and respiratory-supporting tissues such as in the abdomen and surrounding viscera [61].

Obesity increases the risk of arthritis especially primary osteoarthritis (OA), a degenerative disease that causes excessive matrix degradation on the weight-bearing joints such as the hips, knees, and ankles as well as the possibility of foot discomfort and plantar fasciitis, all of which may lead to secondary limitations in physical activity and further weight gain [62]. Although osteoarthritis etiology is multi-factorial, obesity is labeled as the single most predisposing modifiable factor to OA. Increased weight-bearing condition such as obesity causes changes in the articular joint bony structures (joint space narrowing). There is subchondral ablation of the articular bony-cartilage layer of the joint leading to a greater loss of joint space on the weight-bearing joint [63], the proteoglycans level drops significantly to a critical level that makes the articular cartilage to become soften and lose its elasticity character, thus, further compromising joint surface integrity [64]. The resulting features of osteoarthritis arise i.e., pain, stiffness, and reduced movement because of the inconvenience caused by overloading (obesity). Significant weight loss is linked to a decrease in arthritic joint discomfort, and in some people, it may prevent or delay the need for joint replacement surgery [65].

Obesity has been reported to have a direct correlation with non-alcoholic fatty liver disease, increased gastric disease and hepatobiliary diseases are also associated with obesity [66]. Because of their increased biliary excretion of cholesterol, obese people are more likely to develop gallstones, cholecystitis, and biliary dyskinesia (biliary colic without cholelithiasis) [67]. Although cholesterol is eliminated and released when lowering fat storage, it may solidify in the gallbladder and raise the risk of cholelithiasis and cholecystitis. Small quantities of fat consumed every day as part of a weight reduction plan may empty the gallbladder and lower this risk. Non-alcoholic fatty liver disease caused by obesity may lead to cirrhosis and liver failure but is often preventable or mitigated by weight loss [68].

There is a strong evidence indicating that obesity is directly associated with cancer, there are prevalence evidence of obesity propensity for certain types of cancer: endometrial cancer is 7-fold and 2–4 times increased in the severely obese and obese/overweight respectively, esophageal adenocarcinoma is about 4–8 fold high in severely obese and 2.4–2.7 times more likely among obese individuals [69, 70], gastric cancer, kidney cancer and liver cancer are 2-fold higher with obesity, for others, its 1.5 fold likely for colorectal cancer,1.6-fold for gallbladder cancer, 1.2–1.4 and 0.8 times for breast postmenopausal and premenopausal cancer respectively, 1.1 times for ovarian cancer, and 1.3 times for liver cancer [69, 70].

Obesity and cancer remain a topic of investigation**.** Being overweight or obese can induce alterations in the body tissues that increases cancer propensity [71]. These alterations comprise long- *there are prevalence evidence of obesity propensity* levels of other hormones, such as insulin, insulin-like growth factor, sex hormones, as well as alteration of adipocytokine levels like leptins, adiponectin, and visfatin [72]. How obesity increase risk of cancer involves several possible mechanisms including: (1) Fat tissue-increase of estrogen and thus heightened risk of cancer of the breast, ovaries, endometrial etc., (2) hyperinsulinemia from elevated free fatty acid levels in the obese that cause insulin resistance and the eventual increased blood glucose that increases the risk hyperinsulinemia, (3) low-grade inflammation and oxidative stress that affect growth-promoting cytokines and immune modulation, and (4) intestinal flora microbiomes alteration [73].

Very astonishing to know that 20% of all cancer cases are thought to be associated with excess weight gain, and obesity [74], although tumors' etiologic routes are driven by several factors and the mechanisms varies with respect to tumor-type. People living with obesity have an approximately 1.5–3.5-fold greater chance of developing certain cancer-types compared with normal-weight people [75], however, this latter statement does not imply that an overweight or obese person will definitely develop cancer, but the chances rise. Being overweight or obese raises the risk of 13 cancer-types namely: cancer of breast in post-menopausal, ovarian, esophageal, thyroid, pancreas, kidney, hepatocyte, stomach, gallbladder, myeloma, and meningioma of the brain [76]. In Europe, more than 1 in 20 cancer incidents are attributed to excessive weight in the UK [77]. Breast cancer risk is lower among those who lose weight, especially postmenopausal women. Less robust data exist for cancer patients, but observations pointing to a long history of poor outcomes for obese women with breast cancer are well detailed [76]. Even though there are different ideas about how obesity affects the outcome of different cancers, there is much evidence that exercise is advantageous for breast and colon cancer [78, 79]. According to a meta-analysis research that was published in 2002, obesity was the root cause of 11% of instances of colon cancer, 9% of cases of postmenopausal breast cancer, 39% of cases of endometrial cancer, 25% of cases of kidney cancer, and 37% of cases of esophageal cancer [80].

Although there are few studies that look at weight or changes in weight with survival after cancer diagnosis but obesity and poor outcomes for breast cancer survivors have long been reported by researchers. The majority of evidence indicates that being overweight/obese at diagnosis is the main lifestyle risk factor for having a poor prognosis for breast cancer and a poor quality of life status [81] thus, supporting the growing evidence demonstrating that gaining weight after diagnosis increases risk [82]. The importance of energy balance after breast cancer is further supported by research showing that physical activity reduces the risk of breast cancer recurrence [83] as intervention trials of diet and exercise showed longer disease-free survival among intervention groups of which lost significant weight than the control group [84].

On the other hand obesity is one of the most significant risk factors for developing stroke [85]. Both genders and several ethnic communities, including Caucasians, Chinese, and Japanese people, have shown a substantial connection between obesity and an elevated risk for ischemic stroke [86]. The American Heart Association and the American Stroke Association advise managing obesity for both primary [87] and secondary stroke prevention [88] in light of such results. The pleiotropic effects that a number of cytokines released by adipose tissue may have on vascular wall, inflammation, and insulin resistance are thought to be a plausible underlying mechanism relating obesity and stroke [89]. Adiponectin and hepatocyte growth factor are two examples of these adipokines, and low levels of adiponectin and high levels of hepatocyte growth factor have both been linked to an increased risk of stroke-related morbidity [90]. Adiponectin levels and the frequency of ischemic stroke, however, were not shown to be significantly correlated in other investigations [91]. Furthermore, in the morbid obese, higher stroke mortality was linked to both low and high adiponectin concentrations probably due to the transgene-mediated overexpression of adiponectin that causes morbid obesity due to decreased energy expenditure [92]. Additionally, according to a recent meta-analysis [93], individuals who were obese or overweight had a relative risk for ischemic stroke of 1.64 (95% CI: 1.36–1.99) and 1.22 (95% CI: 1.05–1.41), respectively, thus showing incrementally increased risk with increased BMI. Multivariate analysis of data from four cohorts involving 76,227 Chinese individuals revealed an increase of 2 kg/m^2 in baseline BMI resulting in 6.1% increase in the relative risk of total stroke [94].

5. Assessment of obesity as a comorbidity burden

Waist circumference is another clinically feasible measurement that may be used independently or in addition to body mass index BMI to assess weight-related health risk. The World Health Organization has identified sex-specific waist circumference values that signify increased health risk (≥80 cm for women, ≥94 cm for men) and substantially increased health risk (≥88 cm for women, ≥102 cm for men) [95, 96]. Waist circumference correlates well with BMI requiring only a tape measure to provides an estimate of abdominal fat. Abdominal fat is more strongly associated with health risk than fat stored in other regions of the body. Globally, the WHO obesity classification based on body mass index is the easily and generally adopted classification. Base on BMI it classifies obesity as >30 kg/m^2 for obesity, 25–29.9 kg/m^2 for overweight, 18.5–24.9 kg/m^2 for normal, and < 18.5 kg/m^2 as underweight and further classified obesity into 3 more categories of 30–34.9 kg/m^2 for obese-1, 35–39.9 kg/m^2 for obese 2 (super obese), and >40 kg/m^2 for obese 3 (morbid obesity) [97].

The World Obesity Federation (WOF) reiterated that obesity is a progressive disease process that can be chronic and relapsing in nature. Like other chronic illnesses, it's a progressive disease process where the diagnosis is based on some specific parameters such as high body mass index (BMI) value, where the higher the BMI, the more likelihood the devastating clinical consequences [22]. For example, patients with a BMI >30 reports more red flag signs such as shortness of breath and other specific disease symptoms concerning cardiopulmonary systems compared to patients within the normal BMI range [17].

6. Problem statement with obesity

The association between obesity and other life-threatening chronic diseases demands critical scrutiny, as obesity propensity for other diseases is estimated to be about 42% for both overweight and obese [11]. In the West, obesity is one of the most prevailing health problems associated with an increased tendency for development of several medical illnesses and premature loss of life [98], although this trend is fast germinating in the developing world too. Obesity predicaments caused a vast economic setback for medical facilities, and it created a massive financial meltdown for many nations, especially developing countries with poor health insurance and inadequate financial support. Besides, obesity is also linked with a reduced quality of life resulting from a number of associated diseases such as joint degenerative problems that cause pain and restrictions in carrying out daily activities and/or atherosclerosis that leads to Myocardial infarction and heart failure [98]. Obesity affects several portions of our bio-metabolic system from the heart to the liver, kidneys, joints, and reproductive system. It is associated with prevalence of multiple non-communicable diseases, such as type 2 diabetes, cardiovascular disease, hypertension, and stroke, and overall mental health in general. People living with obesity are also three times more likely to be hospitalized for infectious diseases like COVID-19 [99, 100].

At the psychosocial and economic level, obese individuals are less likely to obtain insurance, employment, promotion or enjoy personal relationships due to their quality-of-life predicament and health hindrances or even public stigmatization. Prevention especially and treatment of obesity is therefore now widely and critically recognized as the main priority for most healthcare governing bodies especially the WHO chapter of the United Nations [20]. The myriad of clinical implications of obesity make caring for obese patients a priority for most physicians, especially as mortality rises exponentially with increasing body weight [101, 102].

Psychosocial wellbeing is a measure of health or mental status in the form of quality of life. Although, the latter is a multidimensional notion that evaluates quality of life (QoL) and is associated with rising obesity level globally. QoL is an independent appraisal of both satisfactory and obnoxious features of life because the presumption is that people with higher BMI or weight are more probable to come up with an occurrence of certain mental situations [103]. Physical health in the form of phenotypic changes is a crucial contributing factor to overall quality of life [104]. Clinicians are increasingly coming to terms with the intricate association of obesity with quality of life because obesity is regarded as an important indicator and measure of quality of life. Obesity and psycho-mental disease such as anxiety-depressive disorder have a twisted and communal relationship, this is because obesity enhances the likelihood of getting a psychiatric diagnosis, and that the psycho-mental disorder may in turn further contribute to more weight gain and obesity [105]. Most of the available data

from different research on the relationship between obesity and psychological diseases focused on the major depressive disorder, where the association has been proven to be strong [105]. Although the results of different research vary, the consensus is that there is a correlation between psychopathology and obesity for the majority of common or serious mental illnesses.

Comprehending the societal insights, demands, mindsets, perceptions, and preferences of individuals who are obese is essential because studies revealed societal observation and inclination more often than not have a negative perception towards people living with obesity [106, 107]. Often deem pessimistic due to low self-esteem and discrimination, body discrediting, and stigmatization. This could culminate in several adverse outcomes ranging from depression, anxiety, social phobia, declining medical support, and largely poor quality of life [106, 108]. These implanted unconstructive notions and trials encountered significantly derail or suppress their enthusiasm to manage their life situations thus, leading to a lack of devotion to weight-managing programs: lifestyle modifications, and pharmacological therapies. A detailed comprehension of the perceptions, attitudes, and preferences of the obese individuals is imperative to achieving an encouraging and societal-friendly atmosphere for the well-being of the people living with obesity.

People living with obesity are usually not satisfied with the outcomes from their healthcare provider visits especially if they feel that they were not given sufficient support from friends, or family members needed by them to achieve successful weight-reduction goals [109]. In fact, a study by Agüera et al. [110] showed that most people living with obesity feel that their physicians only accorded them limited time and do not view their health concerns from their own perspectives. These physicians are also reluctant to prescribe weight-lowering medications, they are over-aggressive in promoting strict lifestyle modification advice as the only ultimate way out. Painting pictures of them being at increased folds of developing life complications and poor quality of life.

7. Lifestyle-modifications, exercise and weight control inobesity

Early recognition and reduction of therapy barriers can conserve resources and increase the likelihood of long-term accomplishment, thereby safeguarding the patient from the medical illnesses and psychosocial, emotional and debilitative aftermath effects of excessive overweight/weight gain. Exercise is a crucial part of the behavioral therapy of obesity, along with dietary and lifestyle modifications. The components of these three therapeutic lifestyle changes or adjustment are essential initial stages in the prevention and treatment, but they are often omitted because of the complexity of their practical application [111]. Healthcare professionals that operate in an integrated team environment with a long-term horizon perspective are best able to deliver exercise along with calorie reduction, lifestyle modification, and in certain circumstances, weight loss medication and surgery, where clinical exercise physiologist play a significant role in this team [112]. The inclusion of clinical exercise physiologists in this type of programming is expected to continue to be successful considering that the prevalence of obesity in the United States and throughout the globe is not expected to decline noticeably in the near future [104]. This strategy makes it reasonable to manage, or perhaps eliminate the comorbidities associated with obesity while reducing the personal burden of obesity in a cost- and care-effective way..

As increase weight is associated with several factors from excessive food-intake to lack or inadequate daily mobile activity plus environmental and genetic factors, weight reduction and weight maintenance is undoubtedly a major task for individuals living with obesity. Both those with normal weights and those who are obese may benefit from increased physical exercise to improve their cardiovascular health status [113]. Regular bouts of aerobic exercise have been shown to lower blood pressure [114, 115], and visceral fat [114], the latter of which is linked to increased glucose tolerance and insulin sensitivity (in non-diabetic people) and glycaemic control (in type 2 diabetes patients). In another published study [116], investigators looked at 16 twin pairs with different levels of physical activity, they observed that sedentary twins had more visceral, hepatic, and intramuscular high-risk fat. The pairs with more physical activities had improved body composition and adequate metabolic parameters. Exercising 200 or more minutes per week was shown to more comparably weight loss than those who exercising less than 80 minutes per week in obese [117], such similar outcome was revealed in systematic reviews and meta-analyses published between 2010 and December 2019 [65].

In overall, a key to lessening overweight/obesity is early intervention, best even before conception. Balance and nutritious dietary intake in pregnancy, accompanied by exclusive breastfeeding for 6 months and beyond, perhaps until 2 years benefits all infant unobjectionably [118].

Therefore, public health approach to curtail overweight/obesity are crucial, but the evidence that even a modest weight loss is valuable if it is sustained makes management of obesity worthwhile and of paramount importance. A main mitigating factor to successful weight control therapy is time inadequacy, a commonly confronted barriers to obesity control [119]. Concerned individuals usually find it hard to create adequate time and space to take part in physical activity or to adopt healthy dietary routine or pattern. Because overweight/obesity being important global public health challenges, western and developing nations consider obesity as a chronic and progressive disease which demands resources and efforts as with other chronic illnesses and require lifelong management [120]. As such, there is no "quick fix" for overweight/obesity dilemma. Weight loss programs require great deal of lifelong commitment of dedicated lifestyle adjustment to achieve best weight reduction outcome, with tips on medical support and advice on how best to achieve and maintain a successful weight loss being offered by the medical experts and clinicians.

8. Conclusion

Consistently keeping an eye on one's BMI level, establishing a practical goal and engaging families and friends in the management routine and fight to lose weight are positive therapeutic steps, this is because, even losing what appears to be a modest quantity of weight, such as 3% or more of one's initial body weight and sustaining it for life-long, can significantly reduce the risk of obesity-related complications such as diabetes mellitus, osteoarthritis and cardiac diseases. If overweight/obesity is left unchecked, there is an increased risk of lifetime illnesses and disability by several folds. Additionally, overweight/obesity among the middle age is linked with poor index of quality of life and more detrimental effects in the older age group. Lastly, gender-wise, the overweight/obese women are more likely to develop depressive episodes and eating disorders, particularly the binge-eating disorder otherwise refer to as bulimia, especially if such individuals requires professional aid with their weight

reduction plan. For these challenges and obstacles to weight reduction therapy or plan to be curtailed, appraisal and treatment are very crucial for successful reducing weight and eventual obesity-related chronic diseases.

9. Recommendation

Altogether, all nations must collaborate in global efforts to establish a better and healthy food ecosystem to avail everybody access to healthy diet. Pro-active measures restricting and regulating the sales of food and drinks high in fats food and drinks to children including introduction of appropriate taxing of sugary drinks. Government should provide cities and towns secured space or tarmac for safe exercising and recreation activities. Healthy diet and lifestyle practices should be taught as courses in schools to educate the pupils as well as public adverts to help families educate their children about a healthy habit. The global governing body that oversees the general wellbeing and world obesity crisis for humanity, the WHO, should intensify its supervision of the nationwide trends on overweigh/obesity prevalence, as well as creation of standard guidelines to tackling the prevention and treatment of overweight/obesity for all nations.

10. Limitation

The article does not touch on the heterogeneity of the several functions carried out by the neuronal network formed with other brain sections as there is a crucial links between the energy intake and expenditure effects mediated by distinctive neuronal subgroups as portrayed in distinctive brain waves monitoring.

Obesity: A Prerequisite for Major Chronic Illnesses
DOI: http://dx.doi.org/10.5772/intechopen.111935

Author details

Hafeez Abiola Afolabi[1], Zaidi Zakaria[1*], Salzihan Md. Salleh[2,3], Ewe Seng Ch'ng[4], Siti Norasikin Mohd Nafi[3], Ahmad Aizat Bin Abdul Aziz[5], Sameer Badri Al-Mhanna[6], Ahmad Adebayo Irekeola[7], Yusuf Wada[7] and Abubakar Bishir Daku[8]

1 Department of General Surgery, School of Medical Sciences, Hospital Universiti Sains Malaysia, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

2 Department of Pathology, School of Medical Sciences, Hospital Universiti Sains Malaysia, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

3 Department of Pathology, School of Medical Sciences, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

4 Advanced Medical and Dental Institute, Universiti Sains Malaysia USM, Kepala Batas, Penang, Malaysia

5 Department of Human Genome Centre, School of Medical Sciences, Health Campus, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

6 Department of Physiology and Exercise, School of Medical Sciences, Health Campus, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

7 Department of Medical Microbiology and Parasitology, School of Medical Sciences, Health Campus, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

8 Department of Human Physiology, School of Medical Sciences, Health Campus, Universiti Sains Malaysia USM, Kubang Kerian, Kelantan, Malaysia

*Address all correspondence to: doczakaria44@gmail.com

References

[1] Agofure O. Prevalence of obesity among adults in Issele-Uku, Delta State Nigeria. Alexandria Journal of Medicine. 2018;**54**(4):463-468

[2] World Health Organization. Health at a glance: Asia/Pacific. Measuring progress towards universal health coverage: Measuring progress towards universal health coverage. sixth edition of Health at a Glance Asia/Pacific, World Health Organization OECD summit 2020. Paris: OECD Publishing; 2020. DOI: 10.1787/26b007cd-en

[3] Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: A systematic review and meta-analysis. Obesity Reviews. 2016;**17**(2):95-107

[4] Anyabolu EN. Anaemia and its associated factors in patients attending a General Out-Patient Clinic in a tertiary hospital in Southeast Nigeria. International Journal of Medical and Biomedical Research. 2017;**6**(1):10-17

[5] Van Dyck D et al. International study of objectively measured physical activity and sedentary time with body mass index and obesity: IPEN adult study. International Journal of Obesity. 2015;**39**(2):199-207

[6] Robertson C. What the harm principle says about vaccination and healthcare rationing. Journal of Law and the Biosciences. 2022;**9**(1):lsac017

[7] Caldeira TCM, Soares MM, de Sousa TM, Veiga IPA, da Silva LES, Claro RM. The prevalence of risk and protective factors for noncommunicable diseases (NCDs) among Brazilian adults with pre-obesity and obesity. Obesities. 2022;**2**(3):317-325

[8] Maturo A, Setiffi F. Obesity, physical activity, and healthism. In: Wellness, Social Policy and Public Health. Bingley, England: Emerald Publishing Limited; 2022. pp. 21-32

[9] Afolabi HA et al. The relationship between obesity and other medical comorbidities. Obesity Medicine. 2020;**17**:100164

[10] D.-G. of W. Dr Tedros. World Obesity Day 2022 – Accelerating Action to Stop Obesity. Geneva: World Health Organization; 2022. pp. 1-2. Available from: https://www.who.int/news/item/04-03-2022-world-obesity-day-2022-accelerating-action-to-stop-obesity#

[11] Haththotuwa RN, Wijeyaratne CN, Senarath U. Worldwide epidemic of obesity. In: Obesity and Obstetrics. Colombo, Sri Lanka: Elsevier; 2020. pp. 3-8

[12] De Onis M et al. The W orld H ealth O rganization's global target for reducing childhood stunting by 2025: rationale and proposed actions. Maternal & Child Nutrition. 2013;**9**:6-26

[13] Yan SC et al. International defensive medicine in neurosurgery: Comparison of Canada, South Africa, and the United States. World Neurosurgery. 2016;**95**:53-61

[14] Marynak K, Gentzke A, Wang TW, Neff L, King BA. Exposure to electronic cigarette advertising among middle and high school students—United States, 2014-2016. Morbidity and Mortality Weekly Report. 2018;**67**(10):294

[15] McCartneySA,KachikisA,HuebnerEM, Walker CL, Chandrasekaran S, Adams Waldorf KM. Obesity as a contributor to

immunopathology in pregnant and non-pregnant adults with COVID-19. American Journal of Reproductive Immunology. 2020;**84**(5):e13320

[16] Lim HJ, Xue H, Wang Y. Global trends in obesity. Handbook Eating and Drinking Interdisciplinary Perspectives. 2020;**2020**:1217-1235

[17] Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of Obesity among Adults and Youth: United States, 2011- 2014. Centers for Disease Control and Prevention CDC, United States of America USA. 2015

[18] Sultana R, Sissoho F, Kaushik VP, Raji MA. The case for early use of glucagon-like Peptide-1 receptor agonists in obstructive sleep Apnea patients with comorbid diabetes and metabolic syndrome. Life. 2022;**12**(8):1222

[19] Okati-Aliabad H, Ansari-Moghaddam A, Kargar S, Jabbari N. Prevalence of obesity and overweight among adults in the Middle East Countries from 2000 to 2020: A systematic review and meta-analysis. Journal of Obesity. 2022;**2022**:18. Article ID 8074837. DOI: 10.1155/2022/8074837

[20] World Health Organization. Regional Office for Europe. WHO European Regional Obesity Report 2022. World Health Organization. Regional Office for Europe. 2022. Available from: https://apps.who.int/iris/handle/10665/353747

[21] Seidell JC, Flegal KM. Assessing obesity: Classification and epidemiology. British Medical Bulletin. 1997;**53**(2):238-252

[22] Agha M, Agha R. The rising prevalence of obesity: Part A: Impact on public health. International Journal of Surgery and Oncology. 2017;**2**(7):e17

[23] Hou P et al. Association of Body Composition with pulmonary function in Ningxia: The China Northwest Cohort. Diabetes, Metabolic Syndrome Obesity Targets Therapy. 2022;**2022**:3243-3254

[24] Frellick M. US Adult Obesity Rate Jumped Over Last 20 Years. Medscape Logo, no. Medscape Medical News © 2018, [Online]. 2018. Available from: https://www.medscape.com/viewarticle/893957

[25] Chooi YC, Ding C, Magkos F. The epidemiology of obesity. Metabolism. 2019;**92**:6-10

[26] Cummins S, Macintyre S. Food environments and obesity—Neighbourhood or nation? International Journal of Epidemiology. 2006;**35**(1):100-104

[27] Lockyer S, Spiro A. Socio-Economic Inequalities in Childhood Obesity: Can Community Level Interventions Help to Reduce the Gap? London, United Kingdom: Wiley Online Library; 2019

[28] Van Zyl MK, Steyn NP, Marais ML. Characteristics and factors influencing fast food intake of young adult consumers in Johannesburg, South Africa. South African Journal of Clinical Nutrition. 2010;**23**(3):124-130

[29] van Hout G, van Heck G. Bariatric psychology, psychological aspects of weight loss surgery. Obesity Facts. 2009;**2**(1):10-15

[30] Hill JO, Pagliassotti MJ, Peters JC. Nongenetic determinants of obesity and body fat topography. In: The Genetics of Obesity. Florida, United States Of America USA: CRC Press; 2020. pp. 35-48

[31] Savage JS, Fisher JO, Birch LL. Parental influence on eating behavior:

Conception to adolescence. Journal of Law, Medical and Ethics. 2007;**35**(1):22-34

[32] Furlan A et al. Neurotensin neurons in the extended amygdala control dietary choice and energy homeostasis. Nature Neuroscience. 2022;**25**(11):1470-1480

[33] Kim MS et al. Prefrontal cortex and amygdala subregion morphology are associated with obesity and dietary self-control in children and adolescents. Frontiers in Human Neuroscience. 2020;**14**:563415

[34] Xia S et al. Cortical and thalamic interaction with amygdala-to-accumbens synapses. The Journal of Neuroscience. 2020;**40**(37):7119-7132

[35] Sahu B, Tikoo O, Pati B, Senapati U, Bal NC. Role of Distinct Fat Depots in Metabolic Regulation and Pathological Implications. Bhubaneswar, Odisha, India. 2022

[36] Islam T et al. Anti-inflammatory mechanisms of polyphenols in adipose tissue inflammation: Role of gut microbiota, intestinal barrier integrity and zinc homeostasis. The Journal of Nutritional Biochemistry. 2022;**2022**:109242

[37] Roji AYA, Paliwal M. Concept of Santarpana: A scientific analysis. Journal of Ayurveda Integrative Medical Science. 2021;**6**(3):84-93

[38] Cleary MP, Vasselli JR, Greenwood MR. Development of obesity in Zucker obese (fafa) rat in absence of hyperphagia. American Journal of Physiology Metabolism. 1980;**238**(3):E284-E292

[39] Reuter W, Ulbricht W. Adipocyte volume and count in obesity during weight reduction. Z. Gesamte Inn. Med. 1987;**42**(13):372-373

[40] Badawi A et al. Type 2 diabetes mellitus and inflammation: Prospects for biomarkers of risk and nutritional intervention. Diabetes, Metabolism Syndrome Obesity Targets Therapy. 2010;**2010**:173-186

[41] De Luca C, Olefsky JM. Inflammation and insulin resistance. FEBS Letters. 2008;**582**(1):97-105

[42] Stanley S, Wynne K, Bloom S. Gastrointestinal satiety signals III. Glucagon-like peptide 1, oxyntomodulin, peptide YY, and pancreatic polypeptide. American Journal of Physiological and Liver Physiology. 2004;**286**(5):G693-G697

[43] Huda MSB, Wilding JPH, Pinkney JH. Gut peptides and the regulation of appetite. Obesity Reviews. 2006;**7**(2):163-182

[44] Srivastava S, Rathor R, Singh S, Kumar B, Suryakumar G. Obesity: A risk factor for COVID-19. In: Coronavirus Therapeutics–Volume I. Delhi, India: Springer; 2021. pp. 195-210

[45] Chetambath R. Obesity, obstructive sleep apnoea, and metabolic syndrome: The fatal association. The Indian Practitioner. 2021;**74**(6):35-41

[46] Ofoegbu CKP, Manganyi RM. Off-pump coronary artery bypass grafting; is it still relevant? Current Cardiology Reviews. 2022;**18**(2):64-77

[47] Finkelstein EA. How big of a problem is obesity? Surgery for Obesity and Related Diseases. 2014;**10**(4):569-570

[48] Craig BM, Tseng DS. Cost-effectiveness of gastric bypass for severe obesity. The American Journal of Medicine. 2002;**113**(6):491-498

[49] Felisbino-Mendes MS et al. The burden of non-communicable diseases

attributable to high BMI in Brazil, 1990-2017: Findings from the global burden of disease study. Population Health Metrics. 2020;**18**(1):1-13

[50] Feigl AB, Goryakin Y, Devaux M, Lerouge A, Vuik S, Cecchini M. The short-term effect of BMI, alcohol use, and related chronic conditions on labour market outcomes: A time-lag panel analysis utilizing European SHARE dataset. PLoS One. 2019;**14**(3):e0211940

[51] Benjamin EJ et al. Heart disease and stroke statistics—2018 update: A report from the American Heart Association. Circulation. 2018;**137**(12):e67-e492

[52] Virani SS et al. Heart disease and stroke statistics—2020 update: A report from the American Heart Association. Circulation. 2020;**141**(9):e139-e596

[53] Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity, kidney dysfunction and hypertension: Mechanistic links. Nature Reviews. Nephrology. 2019;**15**(6):367-385

[54] Wang Y-X et al. Associations of birth weight and later life lifestyle factors with risk of cardiovascular disease in the USA: A prospective cohort study. EClinicalMedicine. 2022;**51**:101570

[55] Pi-Sunyer FX. The obesity epidemic: Pathophysiology and consequences of obesity. Obesity Research. 2002;**10**(S12):97S-104S

[56] Willett WC et al. Weight, weight change, and coronary heart disease in women: Risk within the 'normal' weight range. JAMA. 1995;**273**(6):461-465

[57] Mafort TT, Rufino R, Costa CH, Lopes AJ. Obesity: Systemic and pulmonary complications, biochemical abnormalities, and impairment of lung function. Multidisciplinary Respiratory Medicine. 2016;**11**(1):1-11

[58] Friedlander AH, Walker LA, Friedlander IK, Felsenfeld AL. Diagnosing and comanaging patients with obstructive sleep apnea syndrome. Journal of the American Dental Association (1939). 2000;**131**(8):1178-1184

[59] Murugan AT, Sharma G. Obesity and respiratory diseases. Chronic Respiratory Disease. 2008;**5**(4):233-242

[60] McNamara K et al. Targets and indicators for chronic disease prevention in Australia. Australian Health Policy Collaboration. 2019;**2019**

[61] Salome CM, King GG, Berend N. Pulmonary physiology and pathophysiology in obesity physiology of obesity and effects on lung function. Journal of Applied Physiology. 2010;**64**:206-211

[62] Al-Mhanna SB et al. Effectiveness of physical activity on immunity markers and quality of life in cancer patient: A systematic review. PeerJ. 2022;**10**:e13664

[63] Melrose J. The importance of the knee joint meniscal fibrocartilages as stabilizing weight bearing structures providing global protection to human knee-joint tissues. Cell. 2019;**8**(4):324

[64] Pal S. Mechanical properties of biological materials. In: Design of Artificial Human Joints & Organs. Boston, USA: Springer; 2014. pp. 23-40

[65] Bellicha A et al. Effect of exercise training on weight loss, body composition changes, and weight maintenance in adults with overweight or obesity: An overview of 12 systematic reviews and 149 studies. Obesity Reviews. 2021;**22**:e13256

[66] Mutalub YB et al. Gut microbiota modulation as a novel therapeutic strategy in Cardiometabolic diseases. Food. 2022;**11**(17):2575

[67] Adams DB. Biliary dyskinesia: Does it exist? If so, how do we diagnose it? Is laparoscopic cholecystectomy effective or a sham operation? Journal of Gastrointestinal Surgery. 2013;**17**(9):1550-1552

[68] Villaça Chaves G, Pereira SE, Saboya CJ, Ramalho A. Non-alcoholic fatty liver disease and its relationship with the nutritional status of vitamin A in individuals with class III obesity. Obesity Surgery. 2008;**18**(4):378-385

[69] Finer N. Medical consequences of obesity. Medicine (Baltimore). 2011;**39**(1):18-23

[70] Tahergorabi Z, Khazaei M, Moodi M, Chamani E. From obesity to cancer: A review on proposed mechanisms. Cell Biochemistry and Function. 2016;**34**(8):533-545

[71] Rose DP, Vona-Davis L. Biochemical and molecular mechanisms for the association between obesity, chronic inflammation, and breast cancer. BioFactors. 2014;**40**(1):1-12

[72] Dev R, Bruera E, Dalal S. Insulin resistance and body composition in cancer patients. Annals of Oncology. 2018;**29**:ii18-ii26

[73] Abreu MT, Peek RM Jr. Gastrointestinal malignancy and the microbiome. Gastroenterology. 2014;**146**(6):1534-1546

[74] Wolin KY, Carson K, Colditz GA. Obesity and cancer. The Oncologist. 2010;**15**(6):556-565

[75] Pischon T, et al. General and abdominal adiposity and risk of death in Europe. The New England Journal of Medicine. 2008;**359**(20):2105-2120

[76] Karra P et al. Metabolic dysfunction and obesity-related cancer: Beyond obesity and metabolic syndrome. Obesity. 2022;**30**(7):1323-1334

[77] Bhaskaran K, Douglas I, Forbes H, Santos-Silva I, Leon DA, Smeeth L. Body-mass index and risk of 22 specific cancers: A population-based cohort study of 5· 24 million UK adults. Lancet. 2014;**384**(9945):755-765

[78] Al-Mhanna SB et al. Effects of circuit training on patients with knee osteoarthritis: A systematic review and Meta-analysis. Healthcare. 2022;**10**(10):2041

[79] Calle EE, Thun MJ. Obesity and cancer. Oncogene. 2004;**23**(38): 6365-6378

[80] Kruger J, Bowles HR, Jones DA, Ainsworth BE, Kohl HW. Health-related quality of life, BMI and physical activity among US adults (⩾ 18 years): National Physical Activity and weight loss survey, 2002. International Journal of Obesity. 2007;**31**(2):321-327

[81] Dieli-Conwright CM et al. Effects of aerobic and resistance exercise on metabolic syndrome, sarcopenic obesity, and circulating biomarkers in overweight or obese survivors of breast cancer: A randomized controlled trial. Journal of Clinical Oncology. 2018;**36**(9):875

[82] Kroenke CH, Chen WY, Rosner B, Holmes MD. Weight, weight gain, and survival after breast cancer diagnosis. Journal of Clinical Oncology. 2005;**23**(7):1370-1378

[83] Holmes MD, Chen WY, Feskanich D, Kroenke CH, Colditz GA. Physical activity and survival after

breast cancer diagnosis. JAMA. 2005;**293**(20):2479-2486

[84] Chlebowski RT et al. Dietary fat reduction and breast cancer outcome: Interim efficacy results from the Women's intervention nutrition study. Journal of the National Cancer Institute. 2006;**98**(24):1767-1776

[85] Quiñones-Ossa GA, Lobo C, Garcia-Ballestas E, Florez WA, Moscote-Salazar LR, Agrawal A. Obesity and stroke: Does the paradox apply for stroke? Neurointervention. 2021;**16**(1):9-19

[86] Yatsuya H et al. Body mass index and risk of stroke and myocardial infarction in a relatively lean population: meta-analysis of 16 Japanese cohorts using individual data. Circulation. Cardiovascular Quality and Outcomes. 2010;**3**(5):498-505

[87] Goldstein LB et al. Guidelines for the primary prevention of stroke: A guideline for healthcare professionals from the American Heart Association/ American Stroke Association. Stroke. 2011;**42**(2):517-584

[88] Field T, Benavente O. Secondary prevention of stroke. Stroke Cerebrovascular Diseases. 2014;**2014**:163

[89] Rexrode KM. Emerging risk factors in women. Stroke. 2010;**41**(10):S9-S11

[90] Rajpathak SN et al. Hepatocyte growth factor and the risk of ischemic stroke developing among postmenopausal women: Results from the Women's Health Initiative. Stroke. 2010;**41**(5):857-862

[91] Ogorodnikova AD et al. High-molecular-weight adiponectin and incident ischemic stroke in postmenopausal women: A Women's Health Initiative Study. Stroke. 2010;**41**(7):1376-1381

[92] Nagasawa H, Yokota C, Toyoda K, Ito A, Minematsu K. High level of plasma adiponectin in acute stroke patients is associated with stroke mortality. Journal of the Neurological Sciences. 2011;**304**(1-2):102-106

[93] Strazzullo P, D'Elia L, Cairella G, Garbagnati F, Cappuccio FP, Scalfi L. Excess body weight and incidence of stroke: meta-analysis of prospective studies with 2 million participants. Stroke. 2010;**41**(5):e418-e426

[94] Wu Y et al. Determinants of developing stroke among low-income, rural residents: A 27-year population-based, prospective cohort study in northern China. Frontiers in Neurology. 2019;**10**:57

[95] Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: Evidence in support of current National Institutes of Health guidelines. Archives of Internal Medicine. 2002;**162**(18):2074-2079

[96] Lean MEJ, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. BMJ. 1995;**311**(6998):158-161

[97] Edelstein AI, Lovecchio F, Delagrammaticas DE, Fitz DW, Hardt KD, Manning DW. The impact of metabolic syndrome on 30-day complications following total joint arthroplasty. The Journal of Arthroplasty. 2017;**32**(2):362-366

[98] De Lorenzo A, Gratteri S, Gualtieri P, Cammarano A, Bertucci P, Di Renzo L. Why primary obesity is a disease? Journal of Translational Medicine. 2019;**17**(1):1-13

[99] Lighter J et al. Obesity in patients younger than 60 years is a risk factor for Covid-19 hospital admission. Clinical Infectious Diseases. 2020;**71**(15):896-897

[100] Popkin BM et al. Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships. Obesity Reviews. 2020;**21**(11):e13128

[101] Afolabi HA, Zakaria ZB, Hashim MNM, Vinayak CR, Shokri ABA. Body Mass Index and predisposition of patients to knee osteoarthritis. Obesity Medicine. 2019;**16**:100143

[102] Mitsakos AT, Irish W, DeMaria EJ, Pories WJ, Altieri MS. Body mass index and risk of mortality in patients undergoing bariatric surgery. Surgical Endoscopy. 2022;**2022**:1-9

[103] Todisco P, Donini LM. Eating disorders and obesity (ED&O) in the COVID-19 storm. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity. 2020;**26**(3):747-750

[104] Wadden TA, Webb VL, Moran CH, Bailer BA. Lifestyle modification for obesity: New developments in diet, physical activity, and behavior therapy. Circulation. 2012;**125**(9):1157-1170

[105] Kasen S, Cohen P, Chen H, Must A. Obesity and psychopathology in women: A three decade prospective study. International Journal of Obesity. 2008;**32**(3):558-566

[106] Schetz M et al. Obesity in the critically ill: A narrative review. Intensive Care Medicine. 2019;**45**(6):757-769

[107] Puhl RM, Himmelstein MS, Pearl RL. Weight stigma as a psychosocial contributor to obesity. The American Psychologist. 2020;**75**(2):274

[108] Abouzed M et al. Relation of depression and anxiety disorders in choosing obesity management in obese patients. International Journal of Preventive Medicine. 2022;**13**(10):13-136

[109] Pagoto S et al. Tweeting it off: Characteristics of adults who tweet about a weight loss attempt. Journal of American Medical Informatics Association. 2014;**21**(6):1032-1037

[110] Agüera Z, Lozano-Madrid M, Mallorquí-Bagué N, Jiménez-Murcia S, Menchón JM, Fernández-Aranda F. A review of binge eating disorder and obesity. Neuropsychiatrie. 2021;**35**(2):57-67

[111] Wadden TA, Butryn ML, Wilson C. Lifestyle modification for the management of obesity. Gastroenterology. 2007;**132**(6):2226-2238

[112] Wadden TA, Tronieri JS, Butryn ML. Lifestyle modification approaches for the treatment of obesity in adults. The American Psychologist. 2020;**75**(2):235

[113] Haskell WL et al. Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Circulation. 2007;**116**(9):1081

[114] Ross R et al. Exercise-induced reduction in obesity and insulin resistance in women: A randomized controlled trial. Obesity Research. 2004;**12**(5):789-798

[115] Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: A meta-analysis of randomized, controlled trials. Annals of Internal Medicine. 2002;**136**(7):493-503

[116] Leskinen T et al. Leisure-time physical activity and high-risk fat: A longitudinal population-based twin study. International Journal of Obesity. 2009;**33**(11):1211-1218

[117] Jakicic JM, Winters C, Lang W, Wing RR. Effects of intermittent exercise

and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: A randomized trial. JAMA. 1999;**282**(16):1554-1560

[118] Butte NF, Lopez-Alarcon MG, Garza C. Nutrient Adequacy of Exclusive Breastfeeding for the Term Infant during the First Six Months of Life. Texas, USA: World Health Organization; 2002

[119] Teixeira PJ et al. Exercise motivation, eating, and body image variables as predictors of weight control. Medical Science Sports Exercise. 2006;**38**(1):179-188

[120] WH Organization. Obesity: Preventing and Managing the Global Epidemic. Aberdeen, Scotland; 2000