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Obesity: A multifactorial disease- A review

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ABSTRACT:

Background: Obesity is a complex, multifactorial disease. It is a major public health burden with a rising prevalence worldwide. This condition is linked to a variety of genetic, environmental and behavioural factors. Obesity can lead to leptin resistance, insulin resistance and oxidative stress. These can act as potent biomarkers ofobesity,along with other inflammatory biomarkers and signs of hormone dysregulation. Epigenetic factors, such as DNA methylation, histone modifications and non-coding RNAs can contribute to the development of obesity. A relation exists between obesity and alterations in gut microbiota composition. Additionally, gut microbiota is also strongly linked to the development of obesity.

Conclusion:Obesity management stresses upon appropriate diet and lifestyle changes.Dietary changes, such as the inclusion of fibre, precision nutrition and probiotics, prebiotics or synbiotics have shown beneficial effects on health. Multidimensional preventiontechniques need to be followed to control obesity and its various consequences.

Introduction:

Obesity is a complex metabolic disorder that occurs due to excessive accumulation of fat or abnormal distribution of body fat. It can be assessed using various anthropometric methods such as: body mass index (BMI), waist circumference (WC), weight-to-height ratio, waist-to-hip ratio, body fat percentage and neck circumference [1,2,3].

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Obesity poses a global burden. WHO,in 2022, reported that 43% of adults aged 18 years and above were overweight and 16% were living with obesity[4]. In India, the scenario is no different.The National Family Health Survey in 2015-2016 (NFHS-4) recorded the prevalence of overweight or obese adults, aged 15-49 years and withaBMI of ≥ 25.0 kg/m2, to be 20.6% in women and 18.9% in men.NFHS-5 reported further, substantial increase in obesity to 24.0% in women and 22.9% in menfrom the year 2019 to 2021[5]. This shows that the prevalence of obesity is increasing world-wide and represents a growing public health concern which requires immediate attention.

BMI, the most widely used method of obesity measurement, is calculated by weight/(height)^2 ratio. Based on BMI, adults can be classified as: (i) underweight –BMI below 18.5 kg/m^2 (ii) normal weight – BMI between 18.5-24.9 kg/m^2 (iii) overweight – BMI between 25-29.9 kg/m^2 (iv) class I obese – BMI 30-34.9 kg/m^2 (v) class II obese -BMI 35-39.9 kg/m^2 (vi) class III obese - ≥ 40 kg/m^2 [6].However, BMIassessment has some limitations.It does not account for fat distribution in body and differs by ethnicity [3]. It also fails to create a linear link between BMI and critical illnesses [7,8]. BMI gives information about adiposity, whereas the waist-to-height ratio gives information about abdominal adiposity [9]. Neck circumference is a measure of subcutaneous neck fat, which is correlated with various metabolic disorders [3].

Obese individuals can be further classified into 4 categories–(i)Metabolically healthy obese (MHO) (ii) Metabolically unhealthy obese (MUO) (iii) Normal weight obese (NWO) (iv) Metabolically obese normal weight (MONW) [10].

A major reason of obesity is dysregulation of lipid metabolism [11]. In anaerobic processes, excess energy is stored as triglycerides by adipocytes, a process known as lipogenesis. In catabolic processes, stored fats are broken down into free fatty acids (FFA) and glycerol, a process called lipolysis. Imbalance in lipogenesis and lipolysis, along with persistent positive energy balance (due to increased food intake) [12], leads to structural and functional changes in adipose tissue, resulting in hyperplasia and hypertrophy.

Obesity is often marked by the intake of unbalanced diet which is rich in fats and is highly calorific. Studies show that people who lack physical activity or do irregular physical activity, have short sleep durations, smoke and are present in a stressful environment are prone to being obese. When physical activity is low, glucose utilisation for energy decreases, so more glucose is stored as glycogen and fat [1].

This metabolic disorder has various adverse health outcomes, such as: type 2 diabetes mellitus, cardiovascular diseases, dyslipidemia, hepatic diseases, osteoporosis, hypertension, obstructive sleep apnoea, multiple types of cancers, polycystic ovarian syndrome (PCOS) and many other conditions. All of these conditions not only increase the morbidity and mortality rate but also pose an economic burden due to high medical treatment costs. [12,1] The obese population often also suffer from depression, issues regarding self-esteem and body image [13].

In obesity, there is a complex interaction among genetic, environmental, socio-economic, dietary, and behavioural factors[1]. All of these play a crucial part in the accumulation of adipose tissue. This review aims to examine various aspects of obesity.

Biochemistry and Obesity:

Obesity being a complex, multifactorial disorder requires effective management due to its association with a range of biochemical changes.

Regulation of food intake is done at the arcuate nucleus (ARC) of the hypothalamus. It contains neurons that produce 2 major types of neuropeptides: orexigenic peptides [Neuropeptide Y (NPY), Agouti-related peptides (AgRP)] and anorexigenic [pro-opiomelanocortin (POMC) and cocaine and amphetamine-regulated transcript (CART)] peptides. Orexigenic peptides increase appetite and food intake, whereas anorexigenic peptides decrease appetite and food intake. Lateral hypothalamus signalsraise the food intake andparabrachial nucleus signals lessen the food intake [14,15,16,17].

Hormones such as leptin, produced by adipose tissue, inhibit NPY/AgRP neurons and bind to the leptin receptor [18]; therefore, influence POMC production. POMC has 2 products: alpha-MSH and adrenocorticotropin (ACTH). Alpha-MSH binds to melanocortin-4 receptors (MC4R), leading to decreased appetite [19]. Gut hormones such as pancreatic

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polypeptide, GLP-1, oxyntomodulin and cholecystokinin are responsible for satiety [20], whereas ghrelin causes an increase in hunger [21]. In the obese population, leptin resistance is often observed, in which high leptin levels are present, but the brain still receives starvation signals [22].

Genetic factors such as mutations in leptin (LEP) [23], leptin receptor (LEPR) [24], POMC [25], and alpha MSH receptor [26] can cause obesity. The beta-3 adrenergic receptor (ADRB3) gene, involved in lipid metabolism and thermogenesis, harbours a mutation (Trp64Arg) associated with obesity [19]. Adiponectin gene polymorphisms are associated with obesity and insulin resistance [27]. PPAR-γ (Peroxisome Proliferator-Activated Receptor Gamma) Pro12Ala polymorphism associated with obesity risk[28].

Based on mutation, obesity can also be classified as: monogenic or polygenic. Monogenic type of obesity is rare with a single mutation. For example, mutations in the genes encoding LEPR, POMC, and MC4R result in monogenic obesity. On the other hand, polygenic obesity is more common and occurs due to multiple variations in various genes [29].

Studies also show that in the obese population, pro-inflammatory cytokines such as resistin, IL-1, IL-6, and TNF-α are elevated. The anti-inflammatory molecules, such as adiponectin, are downregulated. [12]

IL-1, IL-6, and TNF-α are cytokines that play importantpart in immune responses and inflammation. IL-1 is further classified into IL-1α and IL-1β. IL-1β is a potent pro-inflammatory cytokine, stimulated by lymphocytes, macrophages, and monocytes in response to microbial molecules [30]. IL-6 contributes to the activation of T helper cells, acute-phase reactions, the inhibition of T regulatory (Treg) cells, and the differentiation of B cells [31]. TNF-α is present in neurons and glial cells and participates in various signalling pathways that regulate cellular apoptosis. Dysregulation of these proinflammatory markers has been associated with depression and other neurological disorders [32]. Resistin is an adipokine with proposed pro-diabetogenic properties [33].

Auto-phosphorylation activates JAK2 and PI3K when leptin binds to the leptin receptor. The tyrosine residues on the cytoplasmic tail of the receptors are phosphorylated by activation of JAK2, causing the recruitment and phosphorylation of signalling molecules STAT3. STAT3 activation increases POMC and inhibits AgRP & NPY expression in neurons of ARC. There is opposite function of POMC and AgRP/ NPY peptides which mediates anorectic responses to leptin, which in turn brings about changes in satiety [34]. Overexpression of cytokine signalling protein 3 (SOCS3) may cause leptin resistance and increase obesity as it suppresses STAT3 activation and blocks leptin's function. By inducing pro-inflammatory cytokines such as TNF-α and IL-6, increased lipid intake causes hypothalamic inflammation, which increases leptin resistance and creates a positive energy balance resulting in obesity [35].

There is conversion of phosphatidylinositol-4,5-bisphosphate (PIP2) to phosphatidylinositol-3,4,5-triphosphate (PIP3) and activation of AKT byPI3K. Activated AKT then modulates glycogen synthase kinase 3, protein kinase C and the Forkhead-box (FOX) protein family which will control glycogen synthesis, glucose uptake, and adipogenesis [36]. Disruption of PI3K/ AKT signalling causes degradation of sortilin 1 (SORT1) which is a component of glucose transporter 4 (GLUT4) storage vesicles, and this decreases insulin sensitivity. Systemic inflammation and pro-inflammatory cytokines, such as TNF-α, IL-1, and IL-6, further increase insulin resistance by phosphorylating serine residues in the insulin receptor (IR) [37,38,39]. This results in a decrease in absorption of glucose and a high increase in blood insulin levels. High insulin levels will lead to lipolysis of triglycerides to release FFAs. These FFAs undergo β-oxidation and lead to the production of Acetyl-CoA, which enters the citric acid cycle to provide energy. Excess acetyl-CoA stimulates the liver to produce ketone bodies, especially β-hydroxybutyrate and acetoacetate, which are energy sources but can cause metabolic abnormalities [40]. Insulin resistance is widely recognised as a fundamental defect seen in obesity and type 2 diabetes. Insulin resistance is a hallmark of both obesity and type 2 diabetes, and prevailing theories suggest that type 2 diabetes ensues when pancreatic beta cells can no longer compensate for the heightened insulin demand associated with chronic insulin resistance [41].

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Oxidative stress is an event which happens due to an imbalance between antioxidants activity and free radical production. It is an important event that happens in the development of obesity. It causes stimulation of adipose tissue deposition along with increasing both adipocyte differentiation and growth.

Obesity causing oxidative stress is related to inflammation of chronic origin as adipose tissue releases cytokine such as TNF-α, IL-1β and IL-6 which can be further increase Reactive Oxygen Species (ROS) production.TNF-α not only induces oxidative stress and ROS synthesis but also affects inflammatory responses, adipose tissue apoptosis, and lipid metabolism by increasing lipogenesis and altering insulin signalling. The release of inflammatory cytokines and pro-inflammatory transcription factors such as nuclear factor kappa (NF-kb) and activated protein-1 (AP-1) is caused by ROS, which will further cause increase in production of ROS and ROS further increases the release of adhesion molecules, pro-inflammatory cytokines and growth factors including platelet derived growth factor (PDGF), connective growth factors, vascular cell adhesion molecule-1 (VCAM -1) and insulin like growth factors-1 (IGF-1). In obese population, increasing FFAs serum concentration is witnessed due to excessive fat accumulation which causes glucose metabolism impairment and initiate mitochondrial and peroxisomal oxidation. This leads to increased oxidative tissue damage, mitochondrial and DNA damage, ATP depletion and lipotoxicity. The decrease in glucose transport, insulin secretion and muscle and adipose tissue is seen due to oxidative stress. ROS produced in vicinity can also cause damage to membranes proteins, cellular structures and DNA [42]. ROS also causes mitochondrial DNA damage and dysfunction by the addition of double bonds to or removing the hydrogen atom from DNA bases [43]. Oxidative DNA damage occurs in both telomeric and non-telomeric regions of mitochondrial DNA. Bioavailability of vasodilator's (especially nitric oxide) is decreasedand endothelium contractile factors are elevated in oxidative stress which causes endothelial dysfunction.So oxidative stress leads to oxidized LDL and endothelial adhesion molecule upregulation which facilitates monocyte entry into subendothelial space. As a result, oxidative stress leads to the vascular permeability increase and enhanced adhesions of leukocyte which causes vascular injury [42].

Growth Hormone (GH) is a protein of 191 amino acids whose secretion from the pituitary is regulated by the hypothalamus. GH is involved in lipolysis primarily on visceral adipose tissues in comparison to subcutaneous adipose tissue [44].Hence, diagnosing of growth hormone deficiency syndrome (GHD) may in itself act as a biomarker. This condition is characterised by functional, metabolic and structural changes. These may include changes such as- increased visceral fat, decreased lean mass, osteopenia and/or osteoporosis, altered lipid and carbohydrate metabolism, decreased muscle strength and exercise tolerance, increased mortality due to cardiac and cerebrovascular accidents and reduced psycho-physical wellness; which overall reduce the quality of life [44]. Diagnosis of this condition, is largely based on stimulation tests of GH secretion and Insulin Tolerance Test still remains the most acceptable test [45] .

Thyroid hormone metabolism and obesity are also related. Hyperthyroidism leads to weight loss, whereas hypothyroidism leads to weight gain. TRH influence the release of TSH and this stimulates the production of T3 and T4. TSH secretion is slowed when thyroid hormone levels increase (negative feedback mechanism). Most of the T3 is obtained from the deiodination of T4. In case of obesity, TSH level may be considered as a marker of energy alteration balance. Increased TSH levels may indicate the presence of hormone resistance status. Inspite of increased TSH levels, T3 also remain elevated. This may be due to decrease expression of TSH receptors in peripheral tissues leading to down regulation of thyroid hormone receptors and therefore an increase in TSH and free T3 level. The total and free T4 level do not show changes in obese subjects. A moderate increase in TSH is associated with a slight increase in free T3, total T3 and thyroid volume respectively [46]. The slight increase of free T3 level in obese subjects could be due to a compensatory mechanism following an excess accumulation of fat mass and increased type II deiodinase activity which converts T4 to T3 required for increasing the energy expenditure [47].

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Epigenetic factors

An epigenetic change is a modification that changes gene activity without varying the DNA sequence. Genetic changes driven by epigenetic mechanisms, such as DNA methylation, histone modifications, and non-coding RNAs, can cause obesity [48].

Mechanism of methyl group addition to cytosine is referred as DNA methylation [49]. Differentially methylated CpG loci have been found in cord blood of offsprings of obese mother in comparison with offsprings of normal weight mothers [50]. Changes in DNA methylation patterns can be associated with increased risk of developing metabolic and other disorders[49]. In some studies, it has been observed that, due to restricted diets, lifestyle changes and even bariatric surgery, obesity-associated methylation changes can bereversed [15,51,52,53]. For example, in a study, after consumption of a very-low-calorie diet (VLCD), there was a change observed in the DNA methylation pattern and it started resembling the pattern of a normal-weight person [54]. Another study also reported reversal of hypermethylation with VLCD keto diet , however did not find bariatric surgery showing similar benefit[55].

Histones are basic proteins around which DNA is wrapped to form nucleosomes [56,57]. There are five families of histones: H1, H2A, H2B, H3, and H4. Post-translational modification can bring alterations in DNA packaging compactness and gene expression with processes such as histone ubiquitination, acetylation, phosphorylation and methylation[56].Such changes can alter gene expression, which may be linked to fat accumulation and the progression of obesity. For example, expression of Genes central to adipogenesis, such as CCAAT enhancer-binding protein β(C/EBPB), C/EBPA, pre-adipocyte factor- 1 (Pref-1), adipocyte protein 2 (aP2) and PPAR-γ can be influenced [58]. POMC and NPY expression can be regulated by alterations in histone acetylation, such as decreased H3K9 acetylation at the POMC and increased acetylation at the NPY gene, which have been involved in obesity induced by high-fat diet [59].

Non-coding RNAs, such as miRNAs, can act as biomarkers [60]. miRNAs are 18 to 25nucleotide long short noncoding RNA that can regulate gene expression and silence [61]. They bind to mRNA and can degrade it; hence, they can regulate gene expression [62,63]. Some studies have shown a link between miRNAs and diet, and thus miRNAs can be used as a measure to adjust diet [1]. Several miRNAs have been shown to regulate key signalling pathways involved in adipogenesis. Circulating concentrations of these miRNAs were correlated with BMI and other indicators of obesity. In addition, circulating miRNA levels were also associated with insulin resistance, early childhood obesity, metabolic syndrome in obese adolescents, neonates whose mothers were overweight and obese before pregnancy and obesity-associated inflammatory and metabolic diseases in paediatric patients [57].

Gut microbiota

Intestinal tract microorganisms make up the gut microbiota. The gut-brain axis connects the gut microbiota to the central nervous system. This enables the information to be exchanged with the hypothalamus, pituitary and adrenal glands [64]. This allows gut microbiota to utilise energy from food, store energy and influence gene expression [65].

Multiple intestinal hormones, metabolites and neurotransmitters released by the gut microbiota can alter central appetite [66]. For example, *Lactobacillus* and *Bifidobacterium* produce lactate, which prolongs satiety after a meal [67]. Enteroendocrine cells release anorexigenic hormones such as peptide YY (PYY) and glucogen-like peptide 1 (GLP-1) that can modulate appetite by binding to their receptors locally distributed in enteric neurons, vagal afferents, hypothalamus and brain stem [68-71].

Gut induced liver fat synthesises is done by increasing glucose absorption and activating transcription factors (ChREBP, SREBP-1) along with regulating lipoprotein lipase and ANGPTL4 factors, which are involved in lipid storage [72]. Certain bacteria, such as *Lactobacillus paracasei*, can reduce fat accumulation through PPAR-α/γ pathways [72,73].

In the obese population, specific changes in gut microbiota composition are observed. Some studies have shown that there is a higher number of*Firmicutes* and fewer *Bacteroidetes*in obese subjects [74].

The vitamins, short-chain fatty acids, health-promoting components (such as antioxidants and anti-inflammatory products) generated by the gut microbiota

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along with also toxic compounds (like carcinogens, neurotoxins and immunotoxins) can enter the circulation, and causes changes in gene expression, and influence immune and metabolic pathways [75,76,72].

Gut microbiota ferments indigestible carbohydrates and release SCFAs [77]. These SCFAs regulate lipid metabolism. Even though SCFAs have an effect against diet-induced obesity, overproduction increases energy availability and can promote obesity [72].

Lipopolysaccharide (LPS) is an endotoxin released by Gram-negative bacteria. Gut dysbiosis in obesity can contribute to an excess population of Gram-negative bacteria in the obese population (for example, as *Veillonella),* which can cause more LPS concentration, which in turn can trigger chronic inflammation [78]. By activating the TLR4/MyD88/IRAK4 signalling pathway, LPS disrupts the gut barrier and then translocates to the systemic circulation [79]. Translocation becomes easier due to the decrease of *Akkermansia muciniphila,* which maintains gut barrier integrity [80]. At the same time, SCFAs released by gut microbiota have anti-inflammation properties (especially butyrate, which upregulates PPAR-γ, represses LPS-induced NF-kB activation, induces IL-18 release and promotes regulatory T cells and IL-10-producing T cells differentiation) [81-85].

Gut microbiota relies on diet. It has been observed that the gut microbiota of individuals consuming high fibre is more balanced, and immune mechanisms, such as IgA secretion and the synthesis of antimicrobial peptides, help maintain the mucosal barrier [86-88]. In obese individuals, a diet consisting of high caloric intake and low fibre content can disrupt immune cell function, aggravating gut dysbiosis and increasing inflammation through mechanisms such as NF-κB signalling and TLR-4 activation [87-90].

Nutrition and Obesity:

Obesity can be managed using appropriate diet, lifestyle modifications, behaviour therapy, pharmacotherapy and bariatric surgery [28].

In one study, it was noted that the Indian diet usually consists of excess cereals and a low intake of protein (both animal- and plant-based) and fruits and vegetables (especially in rural households). The diet also includes consuming more unhealthy saturated fats, such as vanaspati. Hence, the Indian diet can be considered unhealthy [91].

It is advised to create a diet that includes plant-based foods such as vegetables, legumes, and fruits, as they are fibre-rich, have a low glycaemic index, are low in calories and are high in water content, thereby providing satiety [92]. Consumption of fish and dairy products should be in moderation and lower consumption of saturated (such as red meat) and trans-unsaturated fats isrecommended [44,93].

Milk has an anti-obesity effect. It has been found that higher calcium intake is associated with lower body weight and helps prevent some cardiovascular disease risk factors [44].

In the obese population, a pro-inflammatory state is induced by adipocyte hypertrophy and due to high levels of hormones such as leptin, T cell function is disturbed, leading to suppression of immune responses [94]. Omega-3 fatty acids and fibres are given to obese patients and are known as immunonutrients, as they help reduce inflammation [95].

The way an individual reacts to a diet varies from person to person [28]. Every individual responds differently to the same drugs and nutrients [96]. Some studies say that this variability among individuals could be due to genetic differences [28]. Because of this differential effect of nutrients, there is a rise in the concept of precision nutrition, which is personalised for each person to achieve effective results .

Precision nutrition is based on an individual's genetic, epigenetic factors, gut microbiota, metabolic profiles, behavioural patterns (diet, physical activity, sleep) and obesity phenotypic traits (body weight, BMI, waist circumference, central and regional adiposity) [64].

Genomic data is analysed using machine learning and Omics tools. Various genes associated with appetite and obesity have been identified, such as: fat mass and obesity related (FTO), melanocortin 4 receptor 4 (MC4R), leptin (LEP), leptin receptor (LEPR), brain-derived neurotrophic factor (BDNF), tumour necrosis factor alpha (TNF-α), interleukin (IL), Beta-2 Adrenergic Receptor (ADRB2), PPAR-γ etc. [28]. Genetic variations and patterns are studied. The impact of nutrients on geneexpression can be studied using nutrigenomics [97, 98] for example,in overweight/obese

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individuals, FTO gene expression depleted during the long fasting periods of Ramadan [99].

Metabolomics aims to study the metabolome using biological samples (such as urine and blood) and to identify biomarkers of food intake (BFI). BFI can be a metabolically active compound present in the sample. By obtaining data on metabolite concentrations over time, metabolomics can be used for dietary monitoring and to identify dietary patterns [100,28].

Changes in the microbiota profile can directly influence metabolism by affecting energy utilisation from the diet and energy storage; hence, it should be given importance when creating a nutritional plan, as it can cause weight loss or gain [101]. Administration of probiotics, prebiotics, synbiotics, or faecal transplants can be used to replenish the gut microbiota [102-106]. *Lactobacillus* and *Bifidobacterium* are traditional probiotics often used [72]. *Akkermansia muciniphila* is connected with weight loss and can enhance metabolic functions [106]. Some studies suggest that the family *Christensenellaceae* has been linked to weight loss, with higher relative abundance correlating inversely with host BMI [107]. Breastfeeding causes the passage ofmicrobial DNA from the parent to the offspring[108]. The microbiome is also known as a regulator of epigenetic factors, as it can produce microbial metabolites that mediate epigenetic processes, such as short-chain fatty acids (propionate, butyrate, and acetate), trimethylamine-N-oxide (TMAO), folate, and some B vitamins [109,110].

Physical activity is really important for managing obesity leading to good health . Physical activity can influence miRNA expression, which can control gene expression [111,112]. In recent studies, it has been found that physical activity can regulate appetite by altering the lactate produced during glycolysis [113,114].

Sleeping influences appetite. In this new era, due to various factors such as increased electronic use, long working shifts and a more stressful environment, sleep duration has decreased [115]. Sleep deprivation alters hormones that regulate appetite, such as ghrelin, leptin, and insulin [116]. It has been recommended that adults sleep 7-9 hours per night to support optimal health [117].

Conclusion:

Obesity is a huge burden on mankind. It can be managed by changing lifestyle and behavioural habits of an individual. To promote a healthier lifestyle, individuals should reduce fast food consumption, limit alcohol intake, refrain from smoking, decrease sedentary behaviour and increase physical activity. Health camps can be organised to create awareness and educate people about obesity, its complications and effective strategies for prevention and control. It is possible that with timely intervention and long-term lifestyle changes,obesity and negative health consequences associated with it can be avoided.

This study is aligned with UN Sustainable Development Goal No.3 (Good Health and Well-being) implementation of Govt. of India.

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