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Review on Pathophysiology and Therapeutic Approaches of Obesity

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ABSTRACT

Obesity and overweight pose a major risk for serious diet-related chronic diseases, including type 2 diabetes, cardiovascular disease, hypertension and stroke, and certain forms of cancer. The health consequences range from increased risk of premature death to Serious chronic conditions that reduce the overall quality of life. Of special concern is the increasing incidence of child obesity. Our understanding of how and why obesity occurs is incomplete; however, it involves the integration of social, behavioral, cultural, physiological, metabolic, and genetic factors. Higher body weights are also associated with increases in all-cause mortality. According to WHO around 1.7 billion adults were overweight and 400 million obese. In future there may be near about 700 million people face the problem of obesity and, under the age of 15years, 10% of the world's children's are obese. Obesity develops because of energy required to body adipose tissue that secrets triglycerol during excess food intake and releases free fatty acid FFA thus the excess fat store in the adipose tissues in the body and the mechanism of body action fails to maintain the balance of body fat that is body mass index. Selecting obesity therapy may be guided by body mass index measurements, comorbid illnesses and patient preference.

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INTRODUCTION

The prevalence of obesity is increasing in adult and children in the UK; British children are getting fatter. A similar rise in prevalence of obesity, related to increasing affluence, is reported from many developed countries throughout the world. The association between adult obesity and adverse health outcomes, including diabetes, coronary heart disease, cancer and respiratory problems, is well documented, but there is limited evidence for an association between adolescent obesity and increased risks of adult morbidity and mortality. More immediate effects of developing obesity include psychosocial outcomes, with social isolation and peer problems more common in fatter children. Overweight adolescent women have lower educational attainment, lower incomes and are less likely to marry than those not overweight. If these relationships are indeed causal, then they imply consequences for costs to the health services, and the economy. The limited data available suggest the direct medical costs might amount to 4±5% of total health care[1]. In the USA, an annual cost of US$4 billion has been attributed to loss of productivity due to obesity. The relationship between relative weight and morbidity and mortality may not be linear, but J shaped with an increased risk of adverse health outcomes reported at the lower extremes of fatness in many studies. The extent is unclear to which this relationship between underweight and an increased risk of adverse outcome reflects reverse causality, whereby smoking and pre-existing disease cause both underweight and increased morbidity and mortality.

When energy required to body adipose tissue that secrets triglycerol during excess food intake and releases free fatty acid FFA. It is recognized as an endocrine organ that synthesizes and secretes biologically active molecules called adipokines, which influence various homeostatic systems[2]. In adipose tissue, lipid storage (*i.e* lipogenesis) and utilization (*i.e* lipolysis) are regulated by several hormones and by the nutritional state. These signals activate various transcription factors (e.g., sterol regulatory element-binding protein-1c and peroxisome proliferators-activated receptor -α/γ) and enzymes (e.g., fatty acid syntheses and carnitine palmitoyltransferase-1) to maintain lipid metabolism. Consequently, an imbalance in lipid metabolism leads to changes in adipose tissue mass. Disorders in lipid metabolism not only increase plasma FFA levels but also alter the production of adipokines, which contribute to the development of obesity-related pathologies, such as insulin resistance[3].

Patients with a BMI of 25 kg/m2 or greater are classified as being overweight. Preobesity and obesity class I, II and III (extreme obesity) are defined as a BMI of 25 kg/m2 to 29 kg/m2, 30

kg/m2 to 34 kg/m2, 35 kg/m2 to 39 kg/m2, and 40 kg/m2 or greater, respectively. However, obesity-related disease risk is also increased in individuals with normal weight and BMI who have an increased waist circumference: a waist circumference of more than 102 cm (40 inches) in men and more than 88 cm (35 inches) in women, poses a significant risk. Waist circumference is an indirect measurement of visceral adiposity, which is metabolically active and responsible for the secretion of pro-inflammatory cytokines that are, in part, responsible for the pathogenesis of insulin resistance and the metabolic syndrome[4].

Difference between obesity and overweight

The prevalence of overweight and obesity is commonly assessed by using body mass index (BMI), defined as the weight in kilograms divided by the square of the height in metres (kg/m2). A BMI over 25 kg/m2 is defined as overweight, and a BMI of over 30 kg/m2 as obese. These markers provide common benchmarks for assessment, but the risks of disease in all populations can increase progressively from lower BMI levels[5].

Adult mean BMI levels of 22-23 kg/m2 are found in Africa and Asia, while levels of 25-27 kg/m2 are prevalent across North America, Europe, and in some Latin American, North African and Pacific Island countries. BMI increases amongst middle-aged elderly people, who are at the greatest risk of health complications. In countries undergoing nutrition transition, overnutrition often co-exists with undernutrition. People with a BMI below 18.5 kg/m2 tend to be underweight.

The distribution of BMI is shifting upwards in many populations. And recent studies have shown that people who were undernourished in early life and then become obese in adulthood, tend to develop conditions such as high blood pressure, heart disease and diabetes at an earlier age and in more severe form than those who were never undernourished[6].

Assessment of Weight and Body Fat

Two measures important for assessing overweight and total body fat content are; determining body mass index (BMI) and measuring waist circumference.

Body Mass Index: The BMI, which describes relative weight for height, is significantly correlated with total body fat content. The BMI should be used to assess overweight and obesity and to monitor changes in body weight. Measurements of body weight alone can be

used to determine efficacy of weight loss therapy. BMI is calculated as weight (kg)/height squared (m2)[7]. To estimate BMI using pounds and inches, use: [weight (pounds)/height (inches) 2]x 703. Weight classifications by BMI, selected for use in this report, are shown in the table below.

	Obesity Class	BMI (kg/m^2)
Underweight		<18.5
Normal		18.5-24.9
Overweight		25.0-29.9
Obesity	I	30.0-34.9
	II	35.0-39.9
Extreme Obesity	III	40

Figure No. 1: Classification of Overweight and Obesity by BMI*

Waist Circumference: The presence of excess fat in the abdomen out of proportion to total body fat is an independent predictor of risk factors and morbidity. Waist circumference is positively correlated with abdominal fat content. It provides a clinically acceptable measurement for assessing a patient's abdominal fat content before and during weight loss treatment.

High Risk
Men: >102 cm (>40 in.)
Women: >88 cm (>35 in.)

Figure No. 2: Risk factor of wait circumference

Etiology

The etiology of obesity is multifactorial, involving a complex interaction among genetics, hormones and the environment. Though multiple candidate genes have been implicated in the pathogenesis of obesity, these findings are inconsistent. These genes include the beta-3-adrenergic receptor gene, peroxisome-proliferator-activated receptor gamma 2 gene, chromosome 10p, melanocortin-4 receptor gene and other genetic polymorphisms[7,8].

Multiple hormones are involved in the regulation and pathophysiology of obesity, including gut-related hormones, adipokines and others. Ghrelin is a circulating peptide hormone derived from the stomach. It is the only known peripherally acting orexigenic hormone and is responsible for stimulating appetite. In a double-blind cross-over study, intravenous ghrelin infusion into healthy volunteers led to a 30% increase in food intake at a buffet, with no change in gastric emptying. All other gut-derived hormones serve as anorectic agents that are

responsible for limiting food intake to achieve optimal digestion and absorption while avoiding the consequences of overfeeding, such as hyperinsulinemia and insulin resistance. These anorectic gut hormones are discussed below.

Peptide YY (PYY) is found throughout the intestine at progressively higher levels distally, with the highest levels in the colon and rectum. It is secreted by the L cells of the distal small bowel and colon. PYY is released postprandially, and signals to the hypothalamus, resulting in delayed gastric emptying, thus reducing gastric secretion. Administration of PYY before meals results in decreased food consumption[8,9].

Cholecystokinin (CCK), produced in the gallbladder, pancreas and stomach, and concentrated in the small intestine, is released in response to dietary fat. It regulates gallbladder contraction, pancreatic exocrine secretion, gastric emptying and gut motility. CCK also acts centrally by increasing satiety and decreasing appetite and acts on the satiety signal via subtype CCK-A receptors on the afferent vagal fibres to the brain, causing termination of appetite.

Meal termination is also regulated by postprandial release of oxyntomodulin. This peptide is secreted from the intestinal cells that also secrete PYY. A single infusion of oxyntomodulin suppresses appetite and reduces food intake over a 12 h period. It is associated with a reduction in fasting ghrelin levels[10].

Glucagon-like peptide-1, which is the 6 to 29 amino acid segment of glucagon, enhances satiety and reduces food intake when administered intravenously to humans.

Several hormones, collectively referred to as adipokines, are produced by the adipocytes. The key secretory products are tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), leptin and adiponectin. The role of TNF-α in obesity has been linked to insulin resistance through the liberation of free fatty acids, reduction in adiponectin synthesis and impairment of insulin signalling. TNF-α also activates nuclear factor-kappa B, leading to a series of inflammatory changes in vascular tissue.

IL-6 is a pleiotropic circulating cytokine resulting in inflammation, impairment of host defenses and tissue injury. It is secreted by many cell types, including immune and endothelial cells, fibroblasts and adipocytes. It acts by inhibiting insulin receptor signal

transduction in hepatocytes, increasing circulating free fatty acids from adipose tissue and reducing adiponectin -secretion.

Leptin acts as a dominant long-term signal responsible for informing the brain of adipose energy reserves. Leptin is transported across the blood-brain barrier and binds to specific receptors on appetite-modulating neurons and the arcuate nucleus in the hypothalamus, inhibiting appetite. Leptin deficient mice that lack leptin receptors have been shown to be hyperphagic and obese. Furthermore, leptin deficiency reduces energy expenditure. True leptin deficiency in humans is rare; however, obese humans are, in part, leptin resistant.

Adiponectin is an adipokine derived from plasma protein. It is insulin sensitizing, anti-inflammatory and antiatherogenic. In contrast to other adipokines, adiponectin messenger RNA (mRNA) levels are reduced in adipose tissue in obese and diabetic individuals and adiponectin levels are restored to normal levels after weight loss.

Increased visceral fat results in increased levels of IL-6, TNF-α and C-reactive protein, and reduced levels of adiponectin and interleukin-10, resulting in a proinflammatory milieu that leads to both insulin resistance and endothelial dysfunction and culminating in the metabolic syndrome, diabetes and atherosclerosis[11]. Visceral adiposity modulates these key regulators of inflammation and has a proinflammatory potential equivalent to or greater than that of macrophages. Secondary causes of obesity include drugs, and neuroendocrine diseases (hypothalamic, pituitary, thyroid and adrenal).

Therapeutic management for weight loss and weight maintenance

Weight loss occurs by generating a negative energy balance, which is achieved by consuming fewer calories than energy expended. The evidence and recommendations for nonpharmacological management of obesity, including diet therapy, physical activity and behavioural therapy, as well as pharmacotherapy, and endoscopic and bariatric surgery are discussed in the present review[12].

Dietary Therapy:

In the majority of overweight and obese patients, adjustment of the diet to reduce caloric intake will be required. Dietary therapy consists, in large part, of instructing patients on how to modify their diets to achieve a decrease in caloric intake. A key element of the current

recommendation is the use of a moderate reduction in caloric intake to achieve a slow but progressive weight loss. Ideally, caloric intake should be reduced only to the level required to maintain weight at the desired level. If this level of caloric intake is achieved, excess weight will gradually disappear. In practice, somewhat greater caloric deficits are used in the period of active weight loss, but diets with very low calories are to be avoided. Finally, the composition of the diet should be modified to minimize other cardiovascular risk factors[12,13].

Low-calorie diet (LCD) (800 to 1,500 kcal/day): The LCD recommended contains a nutrient composition that will decrease other risk factors, notably, high serum cholesterol and hypertension.

Table No. 1: Low-calorie diet supplements

Nutrient	Recommended Intake
Calories	Approximately 500 to 1,000 kcal/day reduction from usual intake
Total Fat	30 percent or less of total calories
Saturated Fatty Acids	8 to 10 percent of total calories
Monounsaturated Fatty Acids	Up to 15 percent of total calories
Polyunsaturated Fatty Acids	Up to 10 percent of total calories
Cholesterol	300 mg/day
Protein	Approximately 15 percent of total calories
Carbohydrate	55 percent or more of total calories
Sodium Chloride	No more than 100 mmol per day (approximately 2.4 g of sodium or approximately 6 g of sodium chloride)
Calcium	1,000 to 1,500 mg
Fiber	20 to 30 g

Physical Activity:

An increase in physical activity is an important component of weight loss therapy since it leads to increased expenditure of energy. Increased physical activity may also inhibit food intake in overweight patients. Physical activity can also be helpful in maintaining a desirable weight. In addition, sustained physical activity has the benefit of reducing overall CHD risk beyond that produced by weight reduction alone[14].

Strategies to Increase Physical Activity: Extremely obese persons may need to start with simple exercises that can gradually be intensified. The practitioner must decide whether exercise testing for cardiopulmonary disease is needed before embarking on a new physical

activity regimen. This decision should be based on a patient's age, symptoms, and concomitant risk factors.

✓ Initial activities may be walking or swimming at a slow pace.

✓ With time, depending on progress, the amount of weight loss and functional capacity, the patient may engage in more strenuous activities. Some of these include fitness walking, cycling, rowing, cross-country skiing, aerobic dancing, and rope jumping.

✓ Jogging provides a high-intensity aerobic exercise but can lead to orthopedic injury. If jogging is desired, the patient's ability to do this must first be assessed. The availability of a safe environment for the jogger is also a necessity.

✓ Competitive sports, such as tennis and volleyball, can provide an enjoyable form of physical activity for many, but again, care must be taken to avoid injury, especially in older people.

Behavior Therapy:

The goal of behavior therapy is to alter the eating and activity habits of an obese patient. Behavioral strategies to reinforce changes in diet and physical activity can produce a weight loss in obese adults in the range of 10 percent of baseline weight over 4 months to 1 year. Unless a patient acquires a new set of eating and physical activity habits, long-term weight reduction is unlikely to succeed. The acquisition of new habits is particularly important for long-term weight maintenance at a lower weight. Most patients return to baseline weights in the absence of continued intervention[15].

Self-monitoring of both eating habits and physical activity: Objectifying one's own behavior through observation and recording is a key step in behavior therapy. Patients should be taught to record the amount and types of food they eat, the caloric values, and nutrient composition. Keeping a record of the frequency, intensity, and type of physical activity likewise will add insight to personal behavior.

Stress management: Stress can trigger dysfunctional eating patterns, and stress management can defuse situations leading to overeating. Coping strategies, meditation, and relaxation techniques all have been successfully employed to reduce stress.

Stimulus control: Identifying stimuli that may encourage incidental eating enables individuals to limit their exposure to high-risk situations. Examples of stimulus control strategies include learning to shop carefully for healthy foods, keeping high-calorie foods out of the house, limiting the times and places of eating, and consciously avoiding situations in which overeating occurs.

Problem solving: Self-corrections of problem areas related to eating and physical activity. Approaches to problem solving include identifying weight-related problems, generating or brainstorming possible solutions and choosing one, planning and implementing the healthier alternative, and evaluating the outcome of possible changes in behavior[16].

Contingency management: Behavior can be changed by use of rewards for specific actions, such as increasing time spent walking or reducing consumption of specific foods Rewards can come from either the professional team or from the patients themselves. For example, self-rewards can be monetary or social and should be encouraged.

Cognitive restructuring: Unrealistic goals and inaccurate beliefs about weight loss and body image need to be modified to help change self-defeating thoughts and feelings that undermine weight loss efforts. Rational responses designed to replace negative thoughts are encouraged.

Social support: A strong system of social support can facilitate weight reduction. Family members, friends, or colleagues can assist an individual in maintaining motivation and providing positive reinforcement.

Treatment of Obese Individuals with Binge Eating Disorder:

If a patient suffers from binge eating disorder (BED), consideration can be given to referring the patient to a health professional who specializes in BED treatment. Behavioral approaches to BED associated with obesity have been derived from cognitive behavior therapy (CBT) used to treat bulimia nervosa.

Combined Therapy:

To achieve the greatest likelihood of success from weight loss therapy, the combination of dietary therapy with an LCD, increased physical activity, and behavior therapy will be required. Inclusion of behavior therapy and increased physical activity in a weight loss regimen will provide the best opportunity for weight loss, and hopefully for long-term weight

control. In order to achieve weight loss, such a regimen should be maintained for at least 6 months before considering pharmacotherapy[17].

Pharmacotherapy:

It is important to remember that the major role of medications should be to help patients stay on a diet and physical activity plan while losing weight. Medication cannot be expected to continue to be effective in weight loss or weight maintenance once it has been stopped. Therefore, an initial trial period of several weeks with a given drug or combination of drugs may help determine their efficacy in a given patient. If a patient does not respond to a drug with reasonable weight loss, the physician should reassess the patient to determine adherence to the medication regimen and adjunctive therapies or consider the need for dosage adjustment. If the patient continues to be unresponsive to the medication, or serious adverse effects occur, the physician should consider its discontinuation. Medications are to be used in conjunction with lifestyle modification (i.e. dietary interventions, behavioral therapy, and increased physical activity).

Pharmacotherapy is recommended for individuals with a BMI >30 kg/m2 or a waist circumference >35 inches (women) or 40 inches (men) and for patients with a BMI >27 kg/m2 with the presence of an additional comorbid condition or more than one risk factor for 'weight-related' disease such as hypercholesterolemia, diabetes, hypertension[18,19].

Table No. 2: Pharmacological drugs for Treatment of Obesity

Sr. No.	Medication	Action	Dose
1	Benzphetamine (Didrex)	Noradrenergic	25-50mg 1-3 times/day
2	Phendimetrazine (Bontril, Prelu-2, Melfiat –105 Unicelles)	Noradrenergic	17.5-70mg 2-3 times/day before meals or 105mg/d in the morning
3	Phentermine (Adipex-P)	Noradrenergic	18.75-37.5mg/d once daily before breakfast
4	Phentermine resin (Ionamin)	Noradrenergic	15-30mg/d once daily before breakfast or 10-14 hr before bedtime
5	Diethylpropion (Tenuate, Tenuate Dospan)	Noradrenergic	25mg 3 times/day before meals or 75mg sustained release/d (mid-Morning)
6	Sibutramine (Meridia)	Mixed noradrenergic and serotonergic	10-15mg/day once daily with or without food
7	Orlistat (Xenical)	Lipase inhibitor-reduces nutrient absorption	120mg 3 times/day with or within 1 hour after fat containing meals, plus a daily multivitamin (spaced at least two hours from the medication)
8	Ephedra, herbal products (ma huang), chromium, caffeine (Dexatrim, Acutrim – multiple components)		
9	Other investigational agents: Bupropion (Wellbutrin), topiramate (Topamax), metformin (Glucophage), Fluoxetine (Prozac); Rimonabant, Amphetamines.		

Surgery for Weight Loss:

Surgery is one option for weight reduction for some patients with severe and resistant obesity. The aim of surgery is to modify the gastrointestinal tract to reduce net food intake. Most authorities agree that weight loss surgery should be reserved for patients with severe obesity, in whom efforts at other therapy have failed, and who are suffering from the complications of obesity[20].

CONCLUSION

The medical evaluation should entail a complete history (eating patterns, behavioural patterns, physical activity, weight history, attempts at weight loss, and obesity-related risk factors and complications) and physical examination (including a BMI and waist circumference measurement), as well as appropriate laboratory and diagnostic testing.

Traditionally various therapies were developed for obesity management but recently the new types of targets are improved to reduce the risk of obesity that is the greline, leptin, amyline, MC4 etc. The present review concluded that etiological factors of obesity and detailed description of therapeutic approaches of obesity.

REFERENCES

1. Scotland's obesity strategy (2010) Preventing overweight and obesity in Scotland, Published by the Scottish Government.
2. Katsilambros N. New developments in obesity. Eur J Intern Med 2000;11: 65-74.
3. Chao CY, Shih CC, Wang CJ, Wu JS, Lu FH. (2012) Low socioeconomic status may increase the risk of central obesity in incoming university students in Taiwan. Obes Res Clin Pract 2012; 8: e212-e219.
4. Couch SC, Deckelbaum RJ Obesity and Pregnancy, Nutrition and Health: Handbook of Nutrition and Pregnancy.
5. Beechy L, Galpern J, Petrone A, Das SK. Assessment tools in obesity - psychological measures, diet, activity, and body composition. Physiol Behav 2012; 107: 154-171.
6. Hinney A, Vogel CI, Hebebrand J. From monogenic to polygenic obesity: recent advances. Eur Child Adolesc Psychiatry 2010; 19: 297-310.
7. Sabin MA, Werther GA, Kiess W. Genetics of obesity and overgrowth syndromes. Best Pract Res Clin Endocrinol Metab2011; 25: 207-220.
8. Saboo B, Zaveri H. Recent update in management of obesity and overweight patients: standardized extract of Caralluma imbriata safe and effective therapy, IJCCI.2011; 2: 5-9.
9. Isoldi KK, Aronne LJ. The challenge of treating obesity: The endocannabinoid system as a potential target. J Am Diet Assoc 2008; 108: 823-831.
10. Scheen AJ, Paquot N. Use of cannabinoid CB1 receptor antagonists for the treatment of metabolic disorders. Best Pract Res Clin Endocrinol Metab 2009; 23: 103-116.
11. Blackburn G. Effect of degree of weight loss on health benefits. Obes Res 1995;3(Suppl 2):211S-6S.
12. Clement K, Vaisse C, Manning BS. Genetic variation in the beta 3-adrenergic receptor and an increased capacity to gain weight in patients with morbid obesity. N Engl J Med 1995;333:352-4.
13. Ristow M, Muller-Wieland D, Pfeiffer A, Krone W, Kahn CR. Obesity associated with a mutation in a genetic regulator of adipocyte differentiation. N Engl J Med 1998;339:953-9.
14. Nordmann AJ, Nordmann A, Briel M. Effects of lowcarbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: A meta-analysis of randomized controlled trials. Arch Intern Med 2006;166:285-93.
15. Howard BV, Manson JE, Stefanick ML, *et al*, Low-fat dietary pattern and weight change over 7 years: The Women's Health Initiative Dietary Modification Trial. JAMA 2006;295:39-49.
16. Pirozzo S, Summerbell C, Cameron C, Glasziou P. Advice on low-fat diets for obesity. Cochrane Database Syst Rev 2002:CD003640.
17. Jackson AS, Stanforth PR, Gagnon J, Rankinen T, Leon AS, Rao DC, Skinner JS, Bouchard C, Wilmore JH2002 The effect of sex, age and race on estimating percentage body fat from body mass index: The Heritage Family Study. Int J Obes Relat Metab Disord 26:789-796.
18. Schwartz MW, Seeley RJ, Zeltser LM, Drewnowski A, Ravussin E, Redman LM, Leibel RL. Obesity pathogenesis: an Endocrine Society scientific statement. Endocr Rev. 2017;38(4):267–296.
19. Hu, F, Malik VS. Sugar-sweetened beverages and risk of obesity and type 2 diabetes: epidemiologic evidence. Physiol Behav 2010; 100(1); 47-54.
20. McConahy, KL. Food portions are positively related to energy intake and body weight in early childhood. J Pediatr 2002; 140;340-347.

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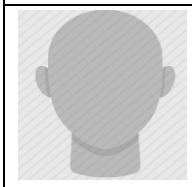	***A. Kavitha*** *B.Pharmacy,* *Sankar Reddy Institute of Pharmaceutical sciences, Salakalveedu(v), Bestavaripeta(M), Prakasam(Dist) Andha pradesh pin – 523370*
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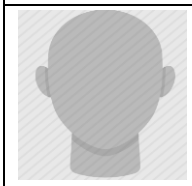
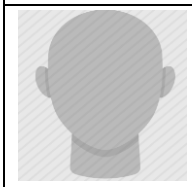
	***B. Veeresh*** *B.Pharmacy,* *Sankar Reddy Institute of Pharmaceutical sciences, Salakalveedu(v), Bestavaripeta(M), Prakasam(Dist) Andha pradesh pin – 523370*
	***K.Manohar Yadav*** *B.Pharmacy,* *Sankar Reddy Institute of Pharmaceutical sciences, Salakalveedu(v), Bestavaripeta(M), Prakasam(Dist) Andha pradesh pin – 523370*