9.4 OBESITY

Obesity is a condition resulting from accumulation of excess body fat. The fat deposition takes place because over a period of time, people consume diets which provided much more energy than they were able to expend for their metabolism, physical activity and growth. The prevalence of obesity in developing countries has increased. This is because communities have emerged from a life style of subsistence towards a life style of affluence. In India, there has been an increased migration of the rural population to urban areas. This shift also has been a contributing factor to life style changes including significant reduction in physical activity leading to changes in weight.

Let us look at the factors that contribute to obesity.

9.4.1 Etiology

What are the causes of obesity? However simple the question may sound, the answer to it is not all that simple. We cannot deny that excess weight results from positive energy balance or consistent consumption of excess calories than the body is able to expend. This means that obesity can be corrected by balancing the intake and output of calories consumed and expended. Thus, it is not an easy task to accomplish because obesity is the net result of a complex interplay of genetic predisposition towards fat storage and a number of environmental factors that determine the weight status of an individual.

We cannot change our heredity but we can, to a certain extent, exercise control over environmental factors by carrying out suitable modifications in our life style. Indians as an ethnic group are at a disadvantage. It is a fact that for a given degree of obesity or BMI, Indians have higher body fat percent and visceral fat (fat around internal organs) than other populations which increases the risk of chronic degenerative diseases in later life. Let's enumerate the various etiological factors for obesity. We will learn about each of them in detail also.

- Genetic susceptibility
- Dietary habits
- Physical activity
- Affluence and abundant availability of food
- Psychological factors
- Hormonal imbalance
- Birth weight and childhood growth pattern

Hormonal imbalance: Certain diseases associated with secretion of hormones, e.g., hypothyroidism, hypogonadism and Cushing's syndrome exhibit obesity as one of the characteristic features. A large number of persons who are unsuccessful in reducing their weight tend to cite hormonal imbalance as causative factor for their obesity but the fact is that only a very small percentage actually suffers from it. Diagnostic tests are available which help in finding out if a person is actually suffering from hormonal imbalance.

Birth weight and childhood growth pattern: It has been shown that slow growth of the foetus in utero and during infancy is followed by accelerated weight gain in childhood. This combination of small size at birth and accelerated childhood weight gain has been found to be associated with exaggeration of adiposity, as well as, insulin resistance in later life. So can we say that small size at birth and accelerated childhood weight gain is a predictor of later obesity? Yes, we can.

Having looked at the etiological factors, next let us understand the concept of energy balance.

9.4.2 Energy Balance

Obesity is a state of positive energy balance created by consumption of calories in amount excessive to the total energy expenditure (TEE) by the body. TEE comprises the following:

a) Resting Energy Expenditure (REE) – 60-75% of TEE
b) Thermic Energy of Food (TEF) – 10% of TEE
c) Energy Expended on Physical Activity (EEPA) – 15-30% of TEE

REE is the energy required to sustain normal body functions like circulation, respiration, pumping of ions across membranes, synthesis of various compounds, maintenance of body temperature etc. The extent of this expenditure depends upon body size and composition.

TEF is the energy expended to digest, absorb and metabolize food including synthesis and storage of various nutrients.

EEPA is the most variable component of total energy expenditure and includes energy expended in voluntary exercises like in walking, cycling, swimming etc. as also that expended involuntarily e.g., in shivering and fidgeting. The sum total of REE, TEF and EEPA gives us the value for total energy expenditure(TEE).

Total Energy Expenditure	=	Resting Energy Expenditure	+	Thermic Energy of Food	+	Energy Expended in Physical Activity
(TEE)	=	(REE)	+	(TEF)	+	(EEPA)

You are aware that the total energy derived from the food that we consume can be calculated from the energy provided by protein, fat and carbohydrates present in the food. Energy provided by 1gram of protein = 4 Kcals, 1 gram of fat = 9 Kcals and 1grams of carbohydrate = 4 Kcals.

Weight status is maintained when the total energy derived from food intake equals the total energy expended by the body. We tend to lose weight when less energy is derived from food than is expended. Let us see what happens when an individual is consuming daily, say, 100 Kcal over and above the amount he is able to expend.

Extra calories ingested/day = 100 Kcal

Extra calories ingested/month = 3000 Kcal

Now 1kg adipose tissue represents = 7700 Kcal (1 gm adipose tissue = 7.7 Kcal)

Weight gain/month = 3000 ÷ 7700 = 0.38 kg approximately

Weight gain/year = 4.56 kg

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You will appreciate that if this continues for a period of, say, five years, theoretically, even before the person realizes, he is transformed into a grossly obese individual. It is hard to believe that as little as one extra chapatti or two teaspoons of butter everyday will result in about 20 kg weight gain over a period of five years. Even though in effect, weight is not deposited in as direct proportion as this. Let us see why. With the increase in energy intake, energy output is affected in a number of ways. Firstly, as the quantity of food ingested is increased, thermogenic effect of food would also increase amounting to about 10% of the excess intake. Secondly, the energy stored would increase both the fat and the fat- free mass resulting in an increase in metabolic rate. This adaptation of metabolic rate which tends to oppose fluctuation in weight does not permit weight gain in direct proportion to increase in caloric intake.

The thermodynamics of weight loss is a bit less complicated. As opposed to the great metabolic cost involved in storage of excess dietary calories as fat, protein or glycogen, hardly any metabolic cost is involved in mobilization of these stores.

Plateau effect: You must have noticed that when people start following weight reducing diets, they lose weight rapidly in the beginning, then a little slowly and finally a plateau is reached when they no longer lose weight. Initially, glycogen stores (sugar stored in liver) are mobilized which is accompanied by a corresponding loss of water. Then, as weight is lost, it results in loss of extra muscle which was developed to support the extra adipose tissue. Loss of lean body mass reduces the BMR rapidly so that on a given diet, the energy deficit is reduced and the rate of weight loss slows down. Weight loss stops at this point unless a change is made either in nutritional intake or physical activity. This fact has been hypothesized as "set-point theory".

Weight cycling: There are a number of obese people who keep loosing and gaining weight a number of times in their lives. This is called the *Yo-yo effect.* Every time they regain lost weight, it takes longer to lose the same amount of weight and also less time to regain it. This frequent losing and gaining of weight is associated with health risks related to normal functioning of the heart. Psychologically also repeated weight gain is quite demoralizing for the obese individual. Withstanding, any amount of intentional weight loss results in significant reduction in all cause, cardiovascular and cancer mortality.

Adipose tissue: At this point, it will not be irrelevant to consider how exactly does an increase in the fat depot take place. For understanding obesity better, it is important for you to know that fat is stored as triglyceride in fat depots made up of adipose tissue. A normal adult woman has about 20% to 25% of her body weight as fat while in men appropriate body fatness is 12% to 15% of body weight. When we put on weight, there is an increase in the adipose tissue. This may either be a result of hypertrophy or hyperplasia of adipocytes (fat cells) or a combination of the two processes. Hypertrophy means increase in the size of adipocytes already present in the body while an increase in their number is known as hyperplasia. As an adult we put on weight mostly by hypertrophy of fat cells although in some forms of obesity hyperplasia may also be there. Hyperplasia basically occurs during infancy and adolescence as a part of growth process. Fat cell size decreases when we lose weight for any reason but weight loss does not involve a decrease in the number of adipocytes.

Brown fat and white adipose tissue (WAT): There are two kinds of adipose tissue. Brown Fat is located around the shoulder blades and kidneys, constituting 1-2% of body weight. It is highly vascular which is the reason for its brown colour. It is capable of producing a large amount of heat for cold adaptation by burning of excess energy. It is a site for conversion of thyroid hormone, thyroxin, to its biologically active form. White adipose tissue acts as a cushion to protect abdominal organs and is the fat that accumulates under the skin. Earlier, it was thought that WAT is passive and acts only as a fat storage depot. WAT, in fact, is a smart tissue and has a number of functions to perform. It has now been realized that WAT is an endocrine organ, which besides some other factors, secretes a hormone leptin. Leptin seems to have a role to play in

① Assessment of Obesity.

pg. 226.

② Types: (pg 229)

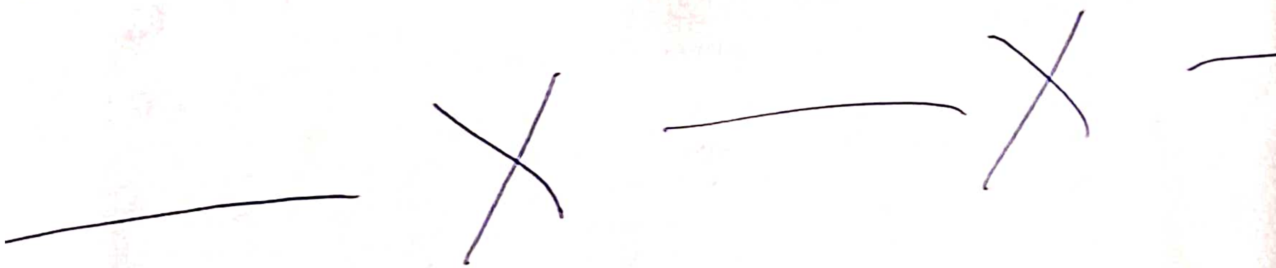
③ Metabolic Aberrations & Clinical Manifestation.

④ ANROID OBESITY.

Is a case in obese individual in which the body's extra fat gets distributed over the ~~old~~ abdominal region of the body because of which the person's body's shape seems to be apple shaped. [Causes - Hormonal disorders/Fluctuations]

⑤ GYNACOID OBESITY.

Overweight with a fat distribution generally characteristic of a woman, with the largest accumulation around the hips.



reducing appetite or increasing satiety and also in regulation of the energy balance. A deficiency of leptin, therefore, is conducive to obesity. Adipocytes in WAT also have a number of hormone receptors on their cell surfaces. That is why individuals with abdominal obesity are prone to developing insulin resistance which initially causes impaired glucose tolerance and ultimately may cause Diabetes mellitus.

Let us learn about the metabolic aberrations and clinical manifestations of obesity next.

9.4.3 Metabolic Aberrations and Clinical Manifestations

The state of obesity brings about certain alterations in the normal body processes which are enumerated herewith and highlighted in Figure 9.1.

Deranged lipid profile: Lipids, as you are already aware, are important dietary constituents that include fats, steroids, phospholipids and glycolipids. A number of vitamins and essential fatty acids are associated with them. In obese individuals, the lipid profile is usually deranged. The triglyceride values are generally high and HDL cholesterol is low. Both triglycerides and HDL cholesterol are synthesized from products of digestion of dietary fats. With weight reduction, both these levels come back to normal.

Insulin resistance: Insulin resistance is a condition in which your body cells cannot utilize insulin efficiently although sufficient amounts are secreted by the pancreas. Obesity is a contributing factor towards insulin resistance. Because sufficient insulin is being produced but the body cells are not able to use it, the blood insulin levels become high (hyperinsulinaemia). This affects the utilization of glucose leading to high fasting blood sugar levels and abnormal glucose tolerance. In addition, levels of plasma glucagon (a hormone produced by pancreas having an effect opposite to that of insulin), free fatty acids and uric acid also are found to be elevated in obese individuals. All these altered biochemical parameters get back to normal as weight loss is affected.

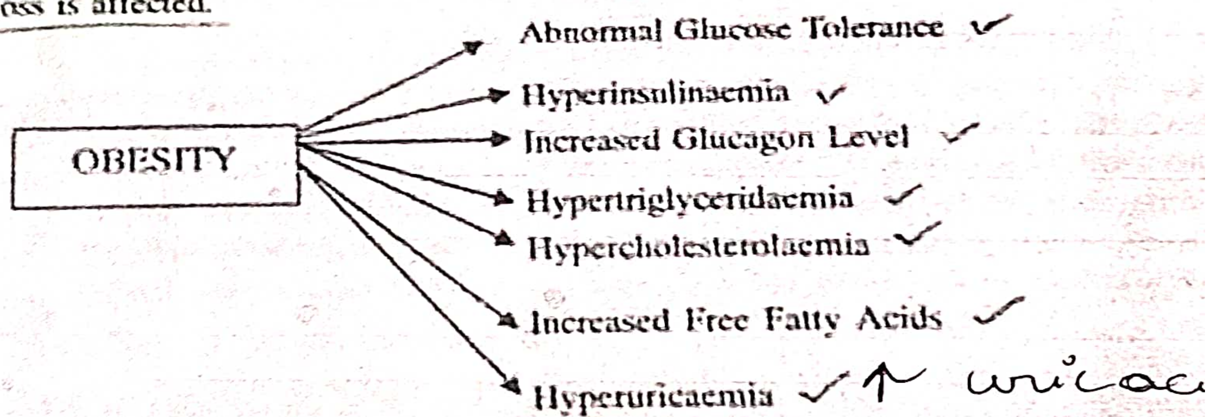


Figure 9.1: Altered biochemical parameters in obesity

The clinical manifestations are highlighted next.

Clinical manifestations: You must have observed that your overweight friends a[nd] colleagues seem to have less energy which makes them an easy prey for fatig[ue]. They are also less agile and more likely to fall because of imbalance. They ha[ve a] tendency to have high blood pressure and dyspnoea (breathlessness on exerti[on]). Many of them may have increased susceptibility to developing skin disorders suc[h as] heat rash, intertrigo (superficial inflammation of two skin surfaces that are in co[ntact] with each other such as between thighs), candidiasis (a fungal infection) and acanth[osis] nigricans (dark, warty growths in skin folds like groin, armpits and mouth).

What are the consequences of obesity? Let us read and find out.