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**QUESTION ONE: What do you understand by primary or simple obesity.**

**Answer:**

Primary obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health. It is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist- hip ratio and total cardiovascular risk factors. In primary obesity, excess accumulation of fat is as a result of low insulin production of the body or reduced response to the insulin hormone without any underlying condition. Any underlying condition that leads to the patient to gain weight and have disorders in the weight regulating system of the body is categorized as secondary obesity e.g endocrine conditions, hypothalamic conditions and congenital disorders. Primary obesity is an exogenous obesity.

**QUESTION TWO: How does congenital syndrome and drug therapy affects obesity.**

**Answer:**

Effect of Congenital syndrome on Obesity

Congenital diseases, also known as birth defects are conditions present at birth regardless of the cause. Examples are fragile X syndrome, Down syndrome, Prader willi syndrome and so on. These diseases exhibit certain characteristics which are known as symptoms. Some congenital diseases will present obesity as a symptom. An example is Prader Willi syndrome, children with this syndrome present a metabolic rate that is lower than normal, and mental conditions such as a constant sense of hunger among other symptoms. Metabolism or metabolic rate is defined as the series of chemical reaction in a living organism that create and break down energy necessary for life. A high metabolism means that more calories are needed to maintain weight. However a person with low metabolism will burn fewer calories at rest and during activity and therefore has to eat less to avoid becoming overweight. In Prader Willi syndrome, one of the symptoms is a constant sense of hunger which will result in compulsive overeating. Because of their slow metabolic rate, they become overweight. Although this is true for Prader Willi syndrome, it is not the same for all congenital diseases that present obesity as a symptom. Some congenital diseases and the reason they present obesity are given below:

* Prader Willi syndrome: people with this disorder become obese as a result of a mental disorder that will result to overeating which together with their slow metabolic rate leads to obesity.
* Lawrence-Moon-Bardet syndrome: people with this disorder become obese as a result of leptin resistance in fat cells (leptin is responsible for decrease in the number of adipocytes). Because the adipocytes do not respond to leptin, they continue to increase in size and leads to obesity.
* Down syndrome: obesity in Down syndrome occurs due to the same reason as that of Lawrence-Moon-Bardet syndrome.
* Pseudohypoparathyroidism: people with this disorder because obese as a result of leptin deficiency.
* Turner syndrome: people with this disorder become obese as a result of insulin resistance. Excess insulin, due to insulin resistance, can lead to weight gain and eventually obesity.
* Cohen Syndrome: people with this disorder become obese as a result of increased response of adipocytes to insulin.

Effects of Drug therapy on Obesity

Medications such as antidepressants, antipsychotic, diabetes medications and generally drugs in class known as thiazolidinediones (TZDs) can lead to weight gain and an increase in fat. This eventually will result in obesity. As in the case of congenital diseases, how these drugs will produce obesity is different. Thiazolidinediones are oral anti-diabetic drugs that act as insulin sensitizers. TZDs improve glycemic control and insulin sensitivity in patients with type 2 diabetes, despite their potential to cause weight gain. Studies have attempted to elucidate the mechanisms behind the apparent paradox of TZDs improving insulin sensitivity while causing weight gain. Data indicate that with TZD treatment, there is a favourable shift in fat distribution from visceral to subcutaneous adipose depots that are associated with improvements in hepatic and peripheral tissue sensitivity to insulin. Although weight gain may occur with TZD therapy, it is not inevitable. Experts do not fully understand why antidepressant leads to weight gain in some people. One theory is that both metabolism and hunger level may be affected. Antidepressants interfere with serotonin, the neurotransmitter that regulates anxiety and mood while also controlling appetite. In particular, these changes may increase cravings for carbohydrate-rich foods, such as bread, pasta, and desserts. Also, depression itself may cause weight gain in some people and weight loss in others. When people are depressed, their appetites are affected. In some people, this may make them hungrier while others lose their appetite. It may be the case that when antidepressants take effects, a person’s usual appetite returns and this has an impact on their weight.

**QUESTION THREE: Outline the aetiology of cancer and its molecular basis**

**Answer:**

Aetiology of cancer

Cancer arises from factors within the cells and external factors from within the environment (inherited mutations or mutations due to metabolism and the environment). Thus, the aetiology of cancer is multifactoral. Physical, chemical, hormonal, metabolic, genetic and environmental factors all have a role in the development of cancer. All these factors cause replication of gene during replication and the gene mutation causes cancer. Carcinogens are mutagens and vice-versa. Thus carcinogens whether physical or chemical causes DNA damage which leads to mutation and mutation causes cancer. All cancer arises from one aberrant cell, which goes on to multiply and produce a tumour mass. One mutation occurs out of 10⁶ cell divisions. By the time a person reaches adulthood, so many amount of cell divisions has occurred. Thanks to the surveillance by the immune system, these aberrant cells are usually destroyed. As age advances, the number of mutations accumulate, hence the statistical probability of cancer is increased. Every normal cell has DNA repair gene mechanism that corrects defects that occurs during gene replication. Carcinogens affects the repair gene mechanisms ( anti-onco gene and onco suppressor gene) and cancer results.

Molecular basis of Cancer

Normally, normal cells replicate and are removed from the system when the need arises by the process called apoptosis. The molecular basis of this is seen in the shortening of the telomeres in the chromosome of the cell. However cancer cells are able to escape apoptosis of the normal cell cycle. They accomplish these by the production of the enzyme telomere polymerase which tend to lengthen the telomeres on the chromosomes and hence cell life is programmed and the cancer cells becomes immortalized.