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**COURSE: Biochemistry**

1. What do you understand by simple obesity

This is obesity resulting when caloric intake exceeds energy expenditure.

 It is as a result of natural (nutritional) factors. It is not from chemical (e.g. medications or diseases) or genetic factors.

1. How does congenital syndrome and drug therapy affect obesity

HOW DRUG THERAPY AFFECTS OBESITY

Some drugs stimulate your appetite, and as a result, you eat more. Others may affect how your body absorbs and stores glucose, which can lead to fat deposits in the midsection of your body. Some cause calories to be burned slower by changing your body’s metabolism. Others cause shortness of breath and fatigue, making it difficult for people to exercise. Other drugs can cause you to retain water, which adds weight but not necessarily fat. Sometimes it is not the drug itself causing weight gain; however, it is the side-effects from the drug.

How much weight is gained varies from person-to-person and from drug-to-drug. Some people may gain a few pounds throughout the course of a year, while other people can gain 10, 20 or more pounds in just a few months. Because many of these medications are taken for chronic conditions, you may use them for several years with their use contributing to significant weight gain throughout time.

These common medical conditions are often treated with prescription drugs that may cause obesity:

* **schizophrenia**
* [**obsessive compulsive disorder**](https://www.drugs.com/condition/obsessive-compulsive-disorder.html) (OCD)
* [**bipolar disorder**](https://www.drugs.com/condition/bipolar-disorder.html)
* [**depression**](https://www.drugs.com/condition/depression.html)
* [**type 1 or 2 diabetes**](https://www.drugs.com/condition/diabetes-mellitus-type-ii.html)
* [**high blood pressure**](https://www.drugs.com/condition/hypertension.html)
* [**epilepsy and seizures**](https://www.drugs.com/condition/seizures.html)
* [**allergies**](https://www.drugs.com/condition/allergies.html)
* [**pain and inflammation**](https://www.drugs.com/drug-class/glucocorticoids.html)

HOW CONGENITAL SYNDROME AFFECTS OBESITY

Congenital Leptin Deficiency

Congenital leptin deficiency is a condition that causes severe obesity beginning in the first few months of life. Affected individuals are of normal weight at birth, but they are constantly hungry and quickly gain weight. Without treatment, the extreme hunger continues and leads to chronic excessive eating (hyperphagia) and obesity. Beginning in early childhood, affected individuals develop abnormal eating behaviors such as fighting with other children over food, hoarding food, and eating in secret.

People with congenital leptin deficiency also have hypogonadotropic hypogonadism, which is a condition caused by reduced production of hormones that direct sexual development. Without treatment, affected individuals experience delayed puberty or do not go through puberty, and may be unable to conceive children (infertile).

 Congenital leptin deficiency is caused by mutations in the [LEP](https://ghr.nlm.nih.gov/gene/LEP) gene. This gene provides instructions for making a hormone called [leptin](https://ghr.nlm.nih.gov/art/large/ghrelin-and-leptin.jpeg), which is involved in the regulation of body weight. Normally, the body's fat cells release leptin in proportion to their size. As fat accumulates in cells, more leptin is produced. This rise in leptin indicates that fat stores are increasing.

LEP gene mutations that cause congenital leptin deficiency lead to an absence of leptin. As a result, the signaling that triggers feelings of satiety does not occur, leading to the excessive hunger and weight gain associated with this disorder. Because hypogonadotropic hypogonadism occurs in congenital leptin deficiency, researchers suggest that leptin signaling is also involved in regulating the hormones that control sexual development. However, the specifics of this involvement and how it may be altered in congenital leptin deficiency are unknown.

Congenital leptin deficiency is a rare cause of obesity.

This condition is inherited in an [autosomal recessive pattern](https://ghr.nlm.nih.gov/art/large/autorecessive.jpeg), which means both copies of the gene in each cell have mutations. The parents of an individual with an autosomal recessive condition each carry one copy of the mutated gene, but they typically do not show signs and symptoms of the condition.

Prader Willi Syndrome

Prader–Willi syndrome (PWS) is a [genetic disorder](https://en.m.wikipedia.org/wiki/Genetic_disorder) due to loss of function of specific genes.[3] In [newborns](https://en.m.wikipedia.org/wiki/Newborn), symptoms include [weak muscles](https://en.m.wikipedia.org/wiki/Hypotonia), poor feeding, and slow development.[3] Beginning in childhood, the person becomes constantly hungry, which often leads to [obesity](https://en.m.wikipedia.org/wiki/Obesity) and [type 2 diabetes](https://en.m.wikipedia.org/wiki/Type_2_diabetes).[3]

PWS is not generally [inherited](https://en.m.wikipedia.org/wiki/Heredity), but instead the genetic changes happen during the formation of the [egg](https://en.m.wikipedia.org/wiki/Egg), [sperm](https://en.m.wikipedia.org/wiki/Sperm), or in early development.

1. Outline the aetiology of cancer and its molecular basis

Cancer refers to any one of a large number of diseases characterized by the development of abnormal cells that divide uncontrollably and have the ability to infiltrate and destroy normal body tissue.

Cancer is caused by changes (mutations) to the DNA within cells. The DNA inside a cell is packaged into a large number of individual genes, each of which contains a set of instructions telling the cell what functions to perform, as well as how to grow and divide. Errors in the instructions can cause the cell to stop its normal function and may allow a cell to become cancerous.

**What do gene mutations do?**

A gene mutation can instruct a healthy cell to:

* **Allow rapid growth.** A gene mutation can tell a cell to grow and divide more rapidly. This creates many new cells that all have that same mutation.
* **Fail to stop uncontrolled cell growth.** Normal cells know when to stop growing so that you have just the right number of each type of cell. Cancer cells lose the controls (tumor suppressor genes) that tell them when to stop growing. A mutation in a tumor suppressor gene allows cancer cells to continue growing and accumulating.
* **Make mistakes when repairing DNA errors.** DNA repair genes look for errors in a cell's DNA and make corrections. A mutation in a DNA repair gene may mean that other errors aren't corrected, leading cells to become cancerous.

These mutations are the most common ones found in cancer. But many other gene mutations can contribute to causing cancer.

**What causes gene mutations?**

Gene mutations can occur for several reasons, for instance:

* **Gene mutations you're born with.** You may be born with a genetic mutation that you inherited from your parents. This type of mutation accounts for a small percentage of cancers.
* **Gene mutations that occur after birth.** Most gene mutations occur after you're born and aren't inherited. A number of forces can cause gene mutations, such as smoking, radiation, viruses, cancer-causing chemicals (carcinogens), obesity, hormones, chronic inflammation and a lack of exercise.

Gene mutations occur frequently during normal cell growth. However, cells contain a mechanism that recognizes when a mistake occurs and repairs the mistake. Occasionally, a mistake is missed. This could cause a cell to become cancerous.

**How do gene mutations interact with each other?**

The gene mutations you're born with and those that you acquire throughout your life work together to cause cancer.

For instance, if you've inherited a genetic mutation that predisposes you to cancer, that doesn't mean you're certain to get cancer. Instead, you may need one or more other gene mutations to cause cancer. Your inherited gene mutation could make you more likely than other people to develop cancer when exposed to a certain cancer-causing substance.

It's not clear just how many mutations must accumulate for cancer to form. It's likely that this varies among cancer types.

Molecular Basis

Cancer is a group of diseases characterized by an autonomous proliferation of neoplastic cells which have a number of alterations, including mutations and genetic instability. Cellular functions are controlled by proteins, and because these proteins are encoded by DNA organized into genes, molecular studies have shown that cancer is a paradigm of acquired genetic disease. The process of protein production involves a cascade of several different steps, each with its attendant enzymes, which are also encoded by DNA and regulated by other proteins. Most steps in the process can be affected, eventually leading to an alteration in the amount or structure of proteins, which in turn affects cellular function. However, whereas cellular function may be altered by disturbance of one gene, malignant transformation is thought to require two or more abnormalities occurring in the same cell. Although there are mechanisms responsible for DNA maintenance and repair, the basic structure of DNA and the order of the nucleotide bases can be mutated. These mutations can be inherited or can occur sporadically, and can be present in all cells or only in the tumor cells. At the nucleotide level, these mutations can be substitutions, additions or deletions. Several of the oncogenes discussed below, including the p53, c-fms, and Ras genes, can be activated by point mutations that lead to amino acid substitution in critical portions of the protein. This article examines the current concepts relating to cellular mechanism that underlie the molecular alterations that characterize the development of cancer.