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* **Bch308 assignment**
* **Title: Diabetes, obesity and cancer**
* **17/sci03/007**

Question :

1. What do you understand by primary obesity
2. How does drug therapy and congenital syndrome affect secondary obesity
3. Discuss the A-etiology of cancer and it’s molecular

 **Answer:**

1. **Primary obesity:** it’s not associated with variable condition such as endocrine disorders, hypothalamic disorders and some congenital conditions.

**2. . How does drug therapy affect secondary obesity** : drug therapy like amphetamines , thyroxin and dinitrophenol can be used to treat or manage secondary obesity

**B. how does congenital syndrome affect**

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 huger continues and leads to 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 obesity: Congenital syndrome 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.

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**3.The A-etiology of cancer and it’s molecular basis :**

Cancer is a disease caused by genetic changes leading to uncontrolled cell growth and tumor formation. The basic cause of sporadic (non-familial) cancers is DNA damage[citation needed] and genomic instability. A minority of cancers are due to inherited genetic mutations.Most cancers are related to environmental, lifestyle, or behavioral exposures. Cancer is generally not contagious in humans, though it can be caused by oncoviruses and cancer bacteria. The term "environmental", as used by cancer researchers, refers to everything outside the body that interacts with humans.The environment is not limited to the biophysical environment (e.g. exposure to factors such as air pollution or sunlight), but also includes lifestyle and behavioral factors

Particular substances, known as carcinogens, have been linked to specific types of cancer. Common examples of non-radioactive carcinogens are inhaled asbestos, certain dioxins, and tobacco smoke. Although the public generally associates carcinogenicity with synthetic chemicals, it is equally likely to arise in both natural and synthetic substances

**Molecular basis of cancer** : 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-rms, and Ras genes, can be activated by point mutations that lead to amino-acid substitution in critical portions of the protein.