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Assignment

1. What do you understand by primary or simple obesity?

Simple Obesity also known as primary obesity is that which is caused by excessive intake of energy in form of food with too little body expenditure. It is also known as diet-induced obesity and it has the largest proportion (95%) in obesity types.

1. How does congenital syndrome and drug therapy affect obesity?

Congenital syndromes can occur before or after birth.

Cushing syndrome is caused by very high levels of glucocorticoids (cortisol). Cortisol stimulates fat and carbohydrate metabolism for fast energy and stimulates insulin release and maintenance of blood sugar levels. It is produced during bodily. Excessive cortisol leads to a 2-5-fold increase in central adipose tissue.

 Prader-Willi syndrome is a syndrome caused by micro-deletion on the long hand of chromosome 15 paternally. It leads to obesity because of a chronic imbalance between energy intake and expenditure due to hyperphagia (an abnormally great desire for food), muscular hypotonia resulting in decreased physical activity and reduced metabolic rate.

Also, in relation to drug therapy, antidepressants interfere with serotonin which is a neurotransmitter that regulates anxiety and mood while also controlling appetite. These changes may increase cravings for carbohydrate-rich foods such as bread, desserts et.c.. Depression can lead to fatigue and inactivity and a lack of physical activity can cause weight gain. Prolonged use of antidepressants like tricyclic antidepressants and atypical antidepressants can cause obesity.

Prolonged use of blood pressure medications such as metoprolol, atenolol et.c could cause continuous weight gain although doctors aren’t sure exactly why beta blockers cause weight gain. It could be that the beta blockers slow metabolism.

Intake of insulin for the treatment to diabetes (type 1 diabetes) could lead to obesity as the person’s body may absorb too much glucose from food resulting in weight gain. Although the insulin helps regulate their glucose levels, it also promotes fat storage in the body

1. Aetiology of Cancer and its molecular basis.

Growth of all body cells is controlled. When the growth is uncontrolled it leads to malignancy hence, Cancer is a malignant growth. Malignant growth is also called tumour. Generally, cancers are named according to the organ affected. Cancer cells are carried to different body parts by circulation where they develop further. However, they are classified based on the three embryonic germ layers from which tissue or organ is derived.

1. Carcinomas- they are cancer of cells derived from either ectoderm or endoderm. Example-Squamous cell carcinoma
2. Sarcomas- they are cancers of tissues of mesodermal origin. Generally, cancers of bones, cartilage, muscle, connective tissue are all Sarcomas.

Cancer Genes

Oncogenes are genes responsible for the development of cancer. Protooncogenes are precursors of oncogenes which are converted to oncogenes by activation.

The products of oncogenes disturb the normal cell growth control mechanism leading to cancer. Usually products of oncogenes are protein kinases that phosphorylate tyrosine residues of proteins. (Tyrosine kinase). Thus, various factors that cause cancer may all act through their effects on proto-oncogene.

CARCINOGENESIS

Carcinogenesis is the development of cancer. Agents which induce cancer are called CARCINOGENS. The types of carcinogens are listed below:

1. Physical Carcinogens: the physical carcinogenic agent is radiant energy both ultraviolet light and ionizing rays (α-rays, β-rays and γ-rays). The main source of ultraviolet light is sunlight. Excessive exposure to these agents can cause various forms of skin cancer.

MODE OF ACTION OF THE PHYSICAL CARCINOGENS

Ultraviolet light and ionizing radiation differ in their mode of action. •

UV rays damage the DNA by formation of pyrimidine dimmers in DNA or by formation of apurinic or apyrimidine sites in DNA.

While ionizing radiations cause the formation of highly reactive free radicals, that can interact with DNA leading to molecular damage.

1. Chemical Carcinogens: they are divided into two groups based on their mode of action
2. Initiators of carcinogenesis- the initiators are further divided into two- Direct acting and Indirect acting/Procarcinogens. The direct acting carcinogens do not require metabolism but the procarcinogens require enzyme catalysed reactions to convert them to carcinogens.
3. Promoters of Carcinogenesis- they are chemical substances which are not necessarily carcinogenic in nature but they help to proliferate cells further e.g phenols

MODE OF ACTON OF CHEMICAL CARCINOGENS

Chemical carcinogens are usually deficient in electrons i.e they are free radicals. These free radical carcinogens can covalently bind to purines, pyrimidines and phosphodiester bonds of DNA causing unrepairable damage. These unrepaired damages generate mutations in DNA and mutation in DNA may lead to cancer.

1. Biologic Carcinogens: they are chiefly viruses, parasites and bacteria. These viruses could have DNA or RNA in their genome. The DNA viruses are classified into five subgroups: a. Papoviruses b. Herpes viruses c. Adenoviruses d. Pox viruses e. Hepadna viruses.

The RNA viruses are retroviruses that contain the enzyme reverse transcriptase although not all retroviruses are oncogenic. Examples of RNA viruses are: Rous Sarcoma Virus, Leukemia Sarcoma Virus.

MODE OF ACTION OF BIOLOGIC CARCINOGENS

**Mode of action of DNA oncogenic virus**-The DNA virus infects the host cell. Then, DNA virus binds tightly to host cell DNA and causes alterations in gene expression of host cell DNA and thus causes cancer by altering the types of protein made in cell. Viral oncoproteins bind to tumor supressors and inactivate them.

**Mode of action of RNA oncogenic virus**-The RNA viruses use RNA as the genome. The RNA gets copied by reverse transcriptase to produce single strand of viral DNA. Single strand of viral DNA is then copied to form another strand of complementary DNA, resulting in double stranded viral DNA or provirus. The provirus is then integrated into the DNA of the host cell genome and may transform the cell into cancer cell.