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17/MHS05/007

PHYSIOLOGY 300L

BCH 308

ASSIGNMENT

1. WHAT DO YOU UNDERSTAND BY PRIMARY OBESITY

2. HOW DOES DRUG THERAPY AND CONGENITAL SYNDROME AFFECT SECONDARY OBESITY

3.DISCUSS THE AETIOLOGY OF CANCER AND ITS MOLECULAR BASIS

SOLUTION

1. Primary obesity is a medical condition in which excess body fat has accumulated to an extent that it may have a negative effect on health.People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 kg/m2; the range 25–30 kg/m2 is defined as overweight.
2. Drug therapy

The goal of treatment is not only to reduce weight, but more importantly to improve the comorbid conditions associated with obesity, such as hyperglycemia, hyperlipidemia, and heart disease, as well as reduce mortality. Patients and physicians should appreciate the concept that obesity is a chronic disease that will require long-term treatment. They should also understand that the efficacy of the current medication options is limited to 5-10% body weight loss in the majority of successful patients. Thus, medication should not be viewed as a panacea for obesity treatment, but as in other chronic diseases, as a next-step treatment option for those continuing a healthy lifestyle regiment, including an increase in daily activity and a calorie-deficit diet. Pharmacotherapy can also be considered an adjunct to bariatric surgery when additional weight loss is required or to prevent weight regain after weight loss surgery.  Using pharmacotherapy for weight management is consistent with treating obesity as a chronic disease that requires a multifaceted approach including behavioral intervention, dietary change, and appropriate medical intervention.

Congenital Syndrome

Determining the etiology of pediatric obesity requires a detailed history, including age at onset and speed of weight gain, pubertal onset and rate of progression, cognitive development, and parents’ and siblings’ heights, weights, and pubertal onset. Identifying a family history of obesity or an endocrine disorder, particularly thyroid disease, is helpful. Hypothyroidism can reduce growth velocity and promote water retention and fat deposition; thus, many (but not all) hypothyroid children have an increased body mass index. Hypothalamic obesity is usually associated with a central nervous system injury or structural lesion involving the hypothalamic-pituitary region and/or the third ventricle; examples include supra/parasellar brain tumors, hypothalamic surgery, or central nervous system radiation.

1. 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 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. Some substances cause cancer primarily through their physical, rather than chemical, effects on cells. A prominent example of this is prolonged exposure to asbestos, naturally occurring mineral fibers which are a major cause of mesothelioma, which is a cancer of the serous membrane, usually the serous membrane surrounding the lungs.

Most cancer-critical genes code for components of the pathways that regulate the social behavior of cells in the body—in particular, the mechanisms by which signals from a cell's neighbors can impel it to divide, differentiate, or die. Most cancer-critical genes code for components of the pathways that regulate the social behavior of cells in the body—in particular, the mechanisms by which signals from a cell's neighbors can impel it to divide, differentiate, or die. Other signaling pathways can function to inhibit cell division, the best known example being the antigrowth effect of the TGFβ family of signaling proteins. Loss of growth inhibition through TGFβ-mediated pathways contributes to the genesis of several types of human cancers. The receptor  TGFβ-RII is found to be mutated in some cancers of the colon and Smad4—a key intracellular signal transducer in the pathway—is inactivated in cancers of the pancreas and some other tissues.

Many cancer cells proliferate inappropriately by eliminating Rb entirely, as we have already seen. Other tumors achieve the same endpoint by acquiring mutations in other components of the Rb regulatory pathway. Thus, in normal cells, a complex of cyclin D1 and the cyclin-dependent kinase Cdk4 (G1-Cdk) stimulates progression through the cell cycle by phosphorylating Rb . The p16 (INK4) protein—which is produced when cells are stressed—inhibits cell-cycle progression by preventing the formation of an active cyclin D1-Cdk4 complex. Some glioblastomas and breast cancers are found to have amplified the genes encoding Cdk4 or cyclin D1, thus favoring cell proliferation. And deletion or inactivation of the *p16* gene is common in many forms of human cancer. In cancers where it is not inactivated by mutation, this gene is often silenced by methylation of its regulatory DNA.