FOLASHADE JEGEDE

300 LEVEL

17/MHS01/168

MEDICINE AND SURGERY

BIOCHEMISTRY

Assignment

1) WHAT DO YOU UNDERSTAND BY PRIMARY OR SIMPLE OBESITY

2) HOW DOES CONGENITAL SYNDROME AND DRUG THERAPY AFFECTS OBESITY

3) OUTLINE THE AETIOLOGY OF CANCER AND ITS MOLECULAR BASIS.

Answers

1) SIMPLE OBESITY

Simple or primary obesity is a type of obesity due to excessive energy intake and too little consumption, also known as diet. It is a medical condition in which excess body fat has accumulated to an extent that it may have a negative effect on health. A person has traditionally been considered to be obese if they are more than 20 percent over their ideal weight. That ideal weight must take into account the person’s height, age, sex, and build. Obesity is often multifactorial, based on both genetic and behavioural factors. Accordingly, treatment of obesity usually requires more than just dietary changes. Exercise, counselling and support, and sometimes medication can supplement diet to help patients conquer weight problems. Extreme diets, on the other hand, can actually contribute to increased obesity.

2) HOW CONGENITAL SYNDROME AFFECTS OBESITY

 Some genetic syndromes associated with childhood obesity include:

* Prader-Willi syndrome
* Pseudohypoparathyroidism
* Laurence-Moon-Biedl (Bardet-Biedl) syndrome
* Cohen syndrome
* Down syndrome
* Turner syndrome

The epidemic of obesity is also affecting children with congenital heart disease. Concordance rates for obesity and type 2 diabetes mellitus are higher in monozygotic twins than in dizygotic twins, and measures of total body fat (TBF) correlate nearly as strongly in monozygotic twins reared apart as in monozygotic twins reared together. Still, genetic factors cannot explain the increased prevalence of obesity observed among American adolescents over the past generation. Lifelong exposure to glucocorticoids, such as in patients with congenital adrenal hyperplasia to control hyperandrogenism is linked to excess risk of obesity and metabolic disorders.

It is important to consider that factors present since the children’s conception may contribute to “programming” of disease in adult life. The quality of the mother’s nutrition during pregnancy may affect the fetal metabolism and the child’s taste and attitudes towards food. Along the life course, these factors interact with family habits and childhood risks to compose different health and disease pathways.

 HOW DRUG THERAPY AFFECTS OBESITY

Anti-obesity medication or weight loss medications are pharmacological agents that reduce or control weight. These medications alter one of the fundamental processes of the human body, weight regulation, by altering either appetite, or absorption of calories. Some examples are orlistat (xenical), cetilistat, lorcaserin, rimonabant, amylin (pramlinatide) and so on.

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. Treatment with anti-obesity drugs such as tetrahydrolipstatin (e.g. orlistat) and serotonin–noradrenaline reuptake inhibitors (e.g. sibutramine leads to weight loss and, more importantly, reduction in long-term weight regain.

In the United States orlistat (Xenical) is currently approved by the FDA for long-term use. It reduces intestinal fat absorption by inhibiting pancreatic lipase. Rimonabant (acomplia), a second medication, works via a specific blockade of the endocannabinoid system.

Because of potential side effects, and limited evidence of small benefits in weight reduction especially in obese children and adolescents, it is recommended that anti-obesity medications only be prescribed for obesity where it is hoped that the benefits of the treatment outweigh its risks. Medications don’t replace physical activity or healthy eating habits as a way to lose weight. Studies show that weight-loss medications work best when combined with a lifestyle program.

Some anti-obesity medications can have severe, even, lethal side effects, fen-phen being a famous example. Fen-phen was reported through the FDA to cause abnormal echocardiograms, heart valve problems, and rare valvular diseases. Another medication, orlistat, blocks absorption of dietary fats, and as a result may cause oily spotting bowel movements (steatorrhea), oily stools, stomach pain, and flatulence.

3) AETIOLOGY OF CANCER

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 and is also a cause of cancer.

 MOLECULAR BASIS

The causes of cancers necessarily involve an examination of the molecular machinery in cells that guides the basic processes of proliferation (increase in cell number by cell division), differentiation (cell specialization into different tissue types), and apoptosis, (programmed cell death). Those processes are guided by two innate programs in cells, the genetic code and epigenetic code. In cancer each of those codes ultimately becomes altered regardless of whether the disease originated with an external or internal factor. Indeed, a fundamental characteristic of a tumor cell is that it begets a tumor cell. In other words, cancer once manifest, it becomes an inherited disease of the cell and is therefore self-perpetuating. The hereditary nature of cancer at the cellular level explains why alterations have been found in both the genetic and epigenetic codes in tumor cells. The number of alterations seen in the coded programs increases as tumors progress to more advanced stages.