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QUESTION 1: WHAT DO YOU UNDERSTAND BY PRIMARY OR SIMPLE OBESITY

ANSWER: Simply put, primary/simple obesity is a state of excess adipose tissues in the body. It is characterized by the expansion of fat mass, adipocyte size increase, and to a lesser extent cell proliferation (hyperplasia). In cadavers, it is present when fat content exceeds 35 percent od body weight in males and 30 percentage of body weight in females. This is much harder to conclude in living humans seeing as you have to put the lean and fat masses into consideration as well as contribution of bone structure.

QUESTION 2: HOW DOES CONGENITAL SYNDROME AND DRUG THERAPY AFFECT OBESITY

ANSWER: Obesity results in the desensitization of peripheral tissues to insulin. The amount of insulin receptors are decreased thereby elevating plasma insulin levels. A congenital deficiency of leptin can result in obesity seeing as leptin suppresses appetite and overall regulates energy.

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 gene. This gene provides instructions for making a hormone called leptin, 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. Leptin attaches (binds) to and activates a protein called the leptin receptor, fitting into the receptor like a key into a lock. The leptin receptor protein is found on the surface of cells in many organs and tissues of the body including a part of the brain called the hypothalamus. The hypothalamus controls hunger and thirst as well as other functions such as sleep, moods, and body temperature. It also regulates the release of many hormones that have functions throughout the body. In the hypothalamus, the binding of leptin to its receptor triggers a series of chemical signals that affect hunger and help produce a feeling of fullness (satiety). LEP gene mutations that cause congenital leptin deficiency lead to an absence of leptin. As a result, the signaling that triggers feelings of satiety do not occur, leading to the excessive hunger and weight gain associated with this disorder. This condition is inherited in an autosomal recessive pattern, 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.

DIGESTIVE INHIBITORS

A strategy in the treatment of obesity is to use digestive inhibitors that interfere with the breakdown, digestion and absorption of dietary fat in the gastrointestinal tract. A reduction in fat is recommended in most weight loss diets; however, patient compliance with these diets is generally poor. Therefore, digestive inhibitors may have a role in creating the negative energy balance necessary for subsequent weight loss. Gastric and pancreatic lipases aid in the digestion of dietary triglycerides by forming them into free fatty acids that are then absorbed at the brush border of the small intestine. Inhibition of these enzymes leads to inhibition of the digestion of dietary triglycerides and decreased cholesterol absorption, and may decrease absorption of lipid-soluble vitamins (A, D, E and K).24 Orlistat (Xenical), the first lipase inhibitor labeled by the FDA for treatment of obesity, is a potent and irreversible inhibitor of gastric and pancreatic lipases, preventing the absorption of about 30 percent of dietary fat. Orlistat is indicated for use in patients with a BMI of at least 30 kg per m2 or in patients with hypertension, diabetes or dyslipidemia who have a BMI of greater than 27 kg per m2. Based on orlistat's mechanism of action, side effects would be more significant in patients eating a high-fat diet. Gastrointestinal side effects included flatus with discharge, oily spotting and oily stool, fecal urgency, fecal incontinence and abdominal pain.

HORMONAL MANIPULATION

The gastrointestinal tract and central nervous system contain several peptides and hormones that regulate feeding behavior. For example, cholecystokinin and serotonin act to decrease appetite and food intake. Conversely, neuropeptide Y increases food intake and decreases energy expenditure. Leptin may limit food intake, decrease plasma insulin and increase energy expenditure. Therefore, agonists and antagonists of these hormones and peptides are currently under investigation for the treatment of obesity.

QUESTION 3: OUTLINE THE AETIOLOGY OF CANCER AND ITS MOLECULAR BASIS

ANSWER: Cancer is a disease caused by genetic changes leading to uncontrolled cell growth and tumor formation. All cancers are multifactorial in origin, meaning they include genetic, hormone, metabolic, physical, chemical and environmental factors. Most human cancers are spontaneous and they usually originate from one aberrant cell, which goes on to multiply and produce a tumor mass. One mutation occurs out of 106 cell divisions. By the time a person reaches adulthood, about 1026 cell divisions have occurred. Thanks to the immune system, these aberrant cells are usually destroyed. As age advances, the number of mutations accumulate, hence the statistical probability of the incidence of cancer is increased. This is why cancer is often referred to as a disease of old age, especially after 60 years.

Carcinogenesis has several risk factors. The major ones are: chemical carcinogens (tobacco, asbestos) radiation (UV, ionising) and oncogenic virus; while others include diet, chronic infections (such as human papilloma virus, hepatitis B and C viruses, etc) and genetic disposition.

Chemical carcinogens work based on their ability to race intracellular macromolexukes, especially DNA and RNA to induce malfunction in the cells. Their primary target are the onxogenes and tumour suppressor genes. The chemical carcinogenes are brought divided into complete (initiator and promoter) and incomplete (initiator only) whose actions can be direct or indirect. Pro-carcinogens with ubiquitqry enzymes cause carcinogenesis to occur at site of entrance (e.g. bronchial carcinoma) and those with orgasm specific enzymes will cause carcinogens far away (e.g. aromatic amines, first in liver, then moves to kidney). Chemical carcinogens include asbestos, aflatoxin, vinyl chloride, etc.

In radiation carcinogenesis, all types of shortvawe radiation, especially ionising radiation, can cause cancer. Principally, the effects of ionising radiation and ultraviolet rays are distinguished. Radiation energy results in chromosome breakage, translocation and point mutations, changes the protein structure, inactivates enzymes and destroys membranes. UV rays cause cell death, inactivates enzymes, inhibits cell division, inducts mutation and activates tumour suppressor cells. Its most common types are squamous cell carcinoma (epidermis), malignant melanoma (melanocytes) and basocellular carcinoma. The ionising radiation includes alpha rays, beta rays and gamma rays in increasing level of carcinogenic effect.

The oncogenic RNA viruses are referred to as retroviruses since they contain a reverse transcriptase (in the infected cell, a virus DNA is synthetised by the virus RNA which will be incorporated into the host genome). They can either be acute transforming or slow transforming viruses. It can cause type I and III of human T-cell leukaemia. The oncogenic DNA viruses are the main responsibles for the development of malignant tumours in humans. Some viruses are particularly characteristic for certain diseases. The mechanisms of the neoplastic effect of DNA viruses are manifold: some of them, like Human Papilloma Virus (HPV), include transforming sequences (oncogenes) which will be incorporated into the host genome, others have an indirect effect. HPV gene sequences can be detected in some oropharyngeal carcinomas, particularly those of the tonsils and the larynx. Other oncogenic DNA viruses include herpes virus and hep-a-DNA virus.