NAME: LAWAL AMINAT TEMITOPE

MATRIC NO.: 17/MHS01/177

DEPARTMENT: MEDICINE AND SURGERY

LEVEL: 300

ASSIGNMENT TITLE: DIABETES, OBESITY AND CANCER

COURSE CODE: BCH 313

**QUESTION 1:**

**PRIMARY OR SIMPLE OBESITY**

Obesity represents one of the major public health issue associated with increased morbidity and mortality. Obesity increases the risk of contracting diseases such as arterial hypertension, dyslipidemia, type 2 diabetes mellitus, coronary heart disease, cerebral vasculopathy, sleep apnea syndrome, e.t.c.

Primary obesity has been defined simply as a state of excess adipose tissue in the body. Simple obesity occurs when caloric intake exceeds energy expenditure. It has been known to affect 5 to 30% of the adult population depending on the standards used and the population studied. The prevalence has been reported to be higher in the urban poor and lower in the affluent.

Simple obesity in children is influenced by selected environmental factors such as parents’ level of education, familial inclination to obesity and health habits. Simple obesity in children aged 3-15 years is connected with familial and environmental factors, including incorrect eating habits.

**QUESTION 2:**

**HOW CONGENITAL SYNDROME AND DRUG THERAPY AFFECTS OBESITY**

Drug therapy can cause obesity in some people and this is called medicine-related obesity. Medicine-related obesity is not uncommon, especially with certain types of medicines. For example, many steroids can cause weight gain. Medicines that treat mental health problems such as depression and schizophrenia can also cause weight gain.

Some drugs when taken, lead to increased stimulation of appetite. This leads to eating more and gaining extra weight. Some drugs can also affect the body’s metabolism. This causes the body to burn calories at a slower rate. Other drugs can also affect how the body stores and absorbs sugar and other nutrients.

Some drugs can also cause tiredness or shortness of breath and this lead to decreased exercise which can cause weight gain.

Medicines that can cause weight gain include; medicines for diabetes such as insulin, thiazolidinediones, anti-psychotic medicines, anti-depressants, epilepsy medicines, steroid hormone medicines like prednisone or birth control pills, blood-pressure reducing medicines, e.t.c.

Not all medicines of these types cause weight gain. For example, metformin which is a diabetes medicine can lead to weight loss. Topiramate, a medicine used for seizures and migraines, also can help a person lose weight.

Some congenital syndromes can also cause obesity. Congenital anomaly syndromes such as Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, Albright hereditary osteodystrophy, Borjeson-Forssman-Lehmann syndrome and some other rare disorders can lead to obesity. Hypothalamic-pituitary axis abnormalities are thought to be a possible causative mechanism in some of these disorders.

**QUESTION 3:**

**AETIOLOGY OF CANCER AND ITS MOLECULAR BASIS**

**AETIOLOGY OF CANCER**

All cancers are multifactorial in origin. They include genetic, hormonal, metabolic, physical, chemical and environmental factors. Most human cancers are spontaneous.

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. All cancers usually originate from one aberrant cell which goes on to multiply and produce a tumor mass. Most cancers are related to environmental, lifestyle or behavioral exposures. Cancer can be caused by oncoviruses and some cancer bacteria.

Common environmental factors that contribute to cancer death include exposure to different chemical and physical agents such as tobacco, environmental pollutants, infections and radiations. These factors act, at least partly, by altering the function of genes within cells.

Aging has been repeatedly and consistently regarded as an important aspect to consider when evaluating the risk factors for the development of particular cancers. Many molecular and cellular changes involved in the development of cancer accumulate during the aging process and eventually manifest as cancer.

Hereditary cancers are primarily caused by an inherited genetic defect. A cancer syndrome or family cancer syndrome is a genetic disorder in which inherited gebetic mutations in one or more genes predispose the affected individuals to the development of cancers and may also cause the early onset of these cancers.

Many different lifestyle factors contribute to increasing cancer risks. Diet and obesity are related to approximately 30-35% of cancer deaths. Studies have shown that red or processed meat increases the risk of developing breast cancer, prostate cancer and pancreatic cancer. A high-salt diet is linked to gastric cancer.

Cancer is the second most common cause of death in developed countries, second only to cardiovascular diseases.

**MOLECULAR BASIS OF CANCER**

Cancer is a disease of uncontrolled growth and proliferation whereby cells have escaped the body’s normal growth control mechanisms and have gained the ability to divide indefinitely. It is a multi-step process that requires the accumulation of many genetic changes over time. These genetic alterations involve activation of proto-oncogenes, deregulation of tumour suppressor genes and DNA repair genes.

In normal cells, proliferation and progression through the cell cycle is strictly regulated by groups of proteins that interact with each other in a specific sequence of events. Checkpoints ascertain that individual stages of the cell cycle are completed correctly and ensure that incompletely replicated DNA is not passed onto daughter cells. Core to this control system are cyclin-dependent kinases (CDKs). CDKs are ‘master protein kinases’ that drive progression through the different phases of the cell cycle by phosphorylating and activating other downstream kinases. CDK activity is dependent on the presence of activating subunits called cyclins which are synthesized and degraded in cell cycle-dependent manner. Cyclin-CDK complexes are further tightly regulated by CDK inhibitors.

Genetic mutations are responsible for the generation of cancer cells and are thus present in all cancers. These mutations alter the quantity or function of protein products that regulate cell growth and division in DNA repair. Two major categories of mutated genes are oncogenes and tumor suppressor genes.

Oncogenes are abnormal forms of normal genes (proto-oncogenes) that regulate various aspects of cell growth and differentiation. Mutation of these genes may result in direct and continuous stimulation of the pathways that control cellular growth and division, cellular metabolism, DNA repair, angiogenesis, and other physiologic processes.

Genes such as TP53, BRCA1, and BRCA2 play a role in normal cell division and DNA repair and are critical for detecting inappropriate growth signals or DNA damage in cells. If these genes, as a result of inherited or acquired mutations, become unable to function, the system for monitoring DNA integration becomes inefficient, cells with spontaneous genetic mutations persist and proliferate, and tumors result.

The important regulatory protein, p53, prevents replication of damaged DNA in normal cells and promotes cell death (apoptosis) in cells with abnormal DNA to survive and divide. TP53 mutations are passed to daughter cells, conferring a high probability of replicating error-prone DNA, and neoplastic transformation results. TP53 is defective in many human cancers. BRCA1 and BRCA2 mutations that decrease function increase risk of breast and ovarian cancer.

Most epithelial cancers likely result from a sequence of mutations that lead to neoplastic conversion. Further genetic changes may be required for metastasis.