NAME: OMOGBEMEH FAITH ALENOSI
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QUESTIONS

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) **PRIMARY OR SIMPLE OBESITY**:

It is not associated with clinical condition. It is characterised by a normal or increased growth rate with an acceleration of bone age maturation. It is characterised by a reduced growth hormone secretion evaluated by standard provocative tests, the administration of growth hormone releasing hormone or spontaneous 24 hour secretion. It is also associated with high insulin and insulin like growth factor I levels which may interfere in the complex endocrine interactions.

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

HOW DRUG THERAPY AFFECTS OBESITY.

- Medication treatment of obesity should be used only in patients who have health risks related to obesity. Medications should be used in patients with a BMI greater than 30 or in those with a BMI of greater than 27 who have other medical conditions (such as high blood pressure, diabetes, high blood cholesterol) that put them at risk for developing heart disease. Medications should not be used for cosmetic reasons.
- Medications should only be used as an adjunct to diet modifications and an exercise program.
- Like diet and exercise, the goal of medication treatment has to be realistic. With successful medication treatment, one can expect an initial weight loss of at least 5 pounds during the first month of treatment, and a total weight loss of 10%-15% of the initial body weight. It is also important to remember that these medications only work when they are taken. When they are discontinued, weight gain often occurs.
- The first class (category) of medication used for weight control cause symptoms that mimic the sympathetic nervous system. They cause the

body to feel "under stress" or "nervous." As a result, the major side effect of this class of medication is high blood pressure. This class of medication includes sibutramine (Meridia, which was taken off the market in the U.S. in October 2010 due to safety concerns) and phentermine (Adipex P).

- These medications also decrease appetite and create a sensation of fullness. Hunger and fullness (satiety) are regulated by brain chemicals called neurotransmitters. Examples of neurotransmitters include serotonin, norepinephrine, and dopamine. Anti-obesity medications that suppress appetite do so by increasing the level of these neurotransmitters at the junction (called synapse) between nerve endings in the brain.

HOW CONGENITAL SYNDROME AFFECTS OBESITY

Syndromic obesity corresponds to severe obesity associated with additional phenotypes (mental retardation, dysmorphic features, and organ-specific developmental abnormalities). Prader-Willi (PWS) and Bardet-Biedl (BBS) syndromes are the 2 syndromes most frequently linked to obesity, but more than 100 syndromes are now associated with obesity.

- Prader-Willi: some clinical features associated to obesity; neonatal hypotonia, mental retardation, hyperphagia, facial dysmorphy, hypogonadism, shoet stature.
- Bardet- Biedl: some clinical features associated with obesity; mental retardation, renal dystrophy or pigmentary retinathy, dysmorphic extremeities, hypogonadism, kidney anomalies.

3) **AETIOLOGY OF CANCER**

- Cancer arises from cancer within cells and external factors within the environment eg inherent mutation or mutation due to metabolism and environment.
- Aetiology of cancer is multifactorial, physical chemical, hormonal, metabolic, genetic and environment and all have a role in development of cancer.
- They all have replication of genes/mutation.
- Thus carcinogens are mutagens and vice versa.
- In summary, carcinogens whether physical or chemical cause DNA damage which leads to mutation and mutation leads to cancer
- Every normal cell has DNA repair gene mechanism that corrects defects that occur during replication.

- Carcinogens and hereditary mutations affect the repair genes and hence cancer occurs.
- About 50% of human cancer is due to mutation or deletion of this repair gene called ANTI ONCOGENS or ONCOSUPPRESSOR GENE.

MOLECULAR BASIS

- Normally, normal cells replicate and remove from the body through apoptosis, a natural process
- The molecular basis of this is seen in the shortening of the telomeres on the chromosomes on the normal cells
- Cancer cells are able to escape apoptosis of the normal cell cycle. They accomplish this by production of the envyme, TELOMERE POLYMERASE, which tends to lengthen the telomere which prolongs life. In this way cancer cells are immortalised because they escaped apoptosis
- All normal cells receive signal for apoptosis, chemicals that cause cancer destroy signals hence cells continue to multiply uncutrollably.