Biochemistry

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Question 1: what do you understand primary or simple obesity

Simple obesity is a type of obesity resulting when caloric intake exceeds energy expenditure.

Simple obesity is characterized by a normal or increased growth rate with an acceleration of bone age maturation. When longitudinal growth slows down in the presence of obesity, a hormonal disturbance should be sought. Despite normal growth, simple obesity is characterized by a reduced GH secretion evaluated by standard provocative tests, the administration of GH-releasing hormone or spontaneous 24-hour secretion. In obese children GH secretion may be as low as in poorly growing children with classical GH deficiency. The endocrine abnormalities along the GH axis seem to involve complex mechanisms at the hypothalamic, pituitary and peripheral level. Recent data suggest that simple obesity is associated with an increase in GH clearance and a decrease in GH synthesis and secretion. It is also associated with high insulin and insulin-like growth factor I levels which may interfere in the complex endocrine interactions. In conclusion, simple obesity is characterized by normal growth in the presence of 'hyposomatotropism'.

Question 2: How does congenital syndrome and drug therapy affect obesity?

Obesity is a chronic disease, and it requires chronic therapy. There are some congenital syndromes/ disorders and drugs that cause obesity. Hypertension, dyslipidemia, diabetes and cardiovascular diseases are leading causes of mortality in the modern world. All of them are strongly linked to obesity. Some genetic disorders which are linked to obesity are downs syndrome, pohen syndrome, prader-willi syndrome, carpenter syndrome, etc.

While treating obesity, those conditions are also managed. Obese patients should always be treated through lifestyle interventions, though the results of such interventions are modest. Pharmacotherapy is a second step in the treatment of obesity, approved only when weight loss targets were not reached through lifestyle intervention. During the history of antiobesity drugs, many of them were withdrawn because of their side effects. Various guidelines recommend prescribing drug therapy for obesity through consideration of the potential benefits and limitations. Orlistat deactivates intestinal lipase and inhibits intestinal fat lipolysis. It is actually the only drug on the European market approved for the treatment of obesity. Orlistat therapy reduces weight to a modest extent, but it reduces the incidence of diabetes beyond the result achieved with lifestyle changes. Recently, some effective antiobesity drugs like sibutramine and rimonabant have been removed from the market due to their side effects. The new combination of topimarate and fentermine is approved in the US but not in Europe. The cost effectiveness of long-term pharmacotherapy of obesity is still an unresolved question.

Congenital syndrome and drug therapy are both associated with secondary obesity. Examples of FDA-approved drugs that may be considered for the long-term treatment of obesity include orlistat (Xenical, Alli), the combinations of phentermine and extended-release topiramate (Qsymia), and the fixed-dose combination of bupropion and naltrexone (Contrave)

Question 3 : Outline the aetiology of cancer and its molecular basis

Cancer is caused by accumulated damage to genes. Such changes may be due to chance or to exposure to a cancer causing substance.

The substances that cause cancer are called carcinogens. A carcinogen may be a chemical substance, such as certain molecules in tobacco smoke. The cause of cancer may be environmental agents, viral or genetic factors.

We should bear in mind, though, that in the majority of cancer cases we cannot attribute the disease to a single cause.

We can roughly divide cancer risk factors into the following groups:

1. Biological or internal factors, such as age, gender, inherited genetic defects and skin type

2. Environmental exposure, for instance to radon and UV radiation, and fine particulate matter

3. Occupational risk factors, including carcinogens such as many chemicals, radioactive materials and asbestos

Lifestyle related factors

Lifestyle-related factors that cause cancer include:

tobacco

alcohol

UV radiation in sunlight

some food-related factors, such as nitrites and poly aromatic hydrocarbons generated by barbecuing food).

Cancer causing factors related to work and living environments include:

asbestos fibres

tar and pitch

polynuclear hydrocarbons (e.g. benzopyrene)

Some metal compounds

Some plastic chemicals (e.g. Vinyl chloride)

Bacteria and viruses can cause cancer:

Helicobacter pylori (H. pylori, which causes gastritis)

HBV, HCV (hepatitis viruses that cause hepatitis)

HPV (human papilloma virus, papilloma virus, which causes changes eg. Cervical cells)

EBV (Epstein-Barr virus, the herpes virus that causes inflammation of the throat lymphoid)

Radiation can cause cancer:

ionising radiation (e.g. X-ray radiation, soil radon)

non-ionised radiation (the sun's ultraviolet radiation)

Some drugs may increase the risk of cancer:

certain antineoplastic agents

certain hormones

medicines that cause immune deficiency

In 5 – 10 per cent of breast cancer genetic predisposition plays an important role in the emergence of the disease.

Molecular basis of cancer

The molecular basis of cancer is seen in the shortening of telomeres. Normally, cells replicate and remove old cells by a process referred to as apoptosis. Cancer cells are able to escape apoptosis of the normal cell cycle by the release of telomere polymerase which lengthens the telomeres on the chromosome. Through this, apoptosis is averted and the cancer cells are immortalized. All normal cells receive signals for apoptosis and chemicals that cause cancer destroy the signals, hence cells continue to multiply uncontrollably.