NAME: NWACHUKWU CHINAZA

MATRIC NUMBER: 17/MHS03/033

DEPARTMENT: ANATOMY

COURSE CODE: BCH 308

PRIMARY OBESITY

Obesity is a multifactorial pathology that can be related to an altered nutritional behavior or secondary to genetic, hypothalamic, iatrogenic or endocrine diseases

At the base of obesity is adiposopathy defined as “pathologic adipose tissue anatomic/functional disturbances promoted by positive caloric balance in genetically and environmentally susceptible individuals that result in adverse endocrine and immune responses that may cause or worsen metabolic disease”

Therefore, we can classifiy obesity as a primary disease since the adiposopathy determines the dysregulation of the metabolic pathways. Metabolic diseases most associated with primary obesity contribute to atherosclerosis, hypertension, and dyslipidemia, and diabetes type II, hyperandrogenemia in women and hypoandrogenemia/hyperestrogenemia in men.

Obesity is necessary not only related to metabolic consequences and major chronic diseases, as it can considered a serious debilitating condition by itself. Excess of body fat may be accompanied by structural and functional abnormalities that reduced quality of life, as gastrointestinal reflux disease, gallbladder disease, osteoarthritis, obstructive sleep apnea/obesity hypoventilation syndrome, psychological and eating behavior disorders, anxiety and depression, and physical performance. Moreover, obesity has an impact on cognitive functioning and major depressive disorder (MDD), with negative effects and additive on the processing speed and executive function measurements, as highlighted by mood rating questionnaires and neuropsychological tests.

Furthermore, the excess of body fat reduces mobility, walking endurance and physical performance, accompanied by sarcopenia regardless of age but according to the inflammatory status and genetic predisposition. The most serious consequences of obesity on health are hypertension, diabetes, myocardial infarction and major cardiovascular events

HOW DRUG THERAPY AND CONGENITAL SYNDROME AFFECT SECONDARY OBESITY

Children who have unhealthy lifestyles are predisposed to develop hypertension, dyslipidemia and other complications. The epidemic of obesity is also affecting children with congenital heart disease. The aim of this study is to estimate the prevalence of obesity and describe associated risk factors, including family history in children with congenital heart disease.

Methods

A cross-sectional study with 316 children and adolescents with congenital heart disease seen in an outpatient clinic of a reference hospital. Collected sociodemographic data included family history of chronic disease, dietary habits, laboratory tests (total cholesterol, HDL and LDL/cholesterol, triglycerides, fasting glucose, CRP, hematocrit and hemoglobin), and anthropometric assessment. Anthropometric data of the caregivers was self-reported.

Results

The prevalence of excess weight was 26.9%. Altered levels of total cholesterol were observed in 46.9%, of HDL in 32.7%, LDL in 23.6% and of triglycerides levels in 20.0%. A higher frequency of family history of obesity (42.6%; p = 0.001), dyslipidemia (48.1%; p = <0.001), diabetes (47.4%; p = 0.002), hypertension (39.2%; p = 0.006) and ischemic disease (43.7%; p = 0.023), as well as significantly higher values of triglycerides (p = 0.017), glycemia (p = 0.004) and C-reactive protein (p = 0.002) were observed among patients with excess weight.

Conclusion

The presence of modifiable risk factors and the variables associated to excess weight in this population was similar to that described in the literature for children without congenital disease. As these children already present the risks associated to heart disease, it is particularly important to promote a healthy lifestyle in this group.

**Keywords:**Child, Adolescent, Congenital heart disease, Overweight, Ischemic disease

During the last three decades, there has been a considerable increase in the prevalence of obesity in children and adolescents (4–18 year-old) worldwide Children and adolescents with unhealthy lifestyles are predisposed to develop hypertension, dyslipidemia and other complications These factors, as well as physical inactivity, may track into adulthood and increase the risk of chronic diseases such as atherosclerosis The epidemic of obesity is also affecting children with congenital heart disease (CHD). More than one quarter of this population is already overweight. Two main causes have been described: physical activity restrictions and interventions for weight gain in infancy, when many lesions cause undernutrition. These interventions often include consumption of increased calories and foods with high fat and sodium content. Although nutritional requirements and physical functional capacity change as these children grow older and their heart lesions are successfully treated, the inappropriate dietary behaviors and physical inactivity are frequently maintained across childhood. The family frequently influences these unhealthy behaviors, both directly, restricting physical activity, for example, and indirectly, by setting an unhealthy model. When parents are obese, as one example, the risk of obesity in their children is increased

**THE AETIOLOGY MOLECULAR BASIS OF CANCER**

Oncogenes and tumor suppressors and the mutations that affect them are different beasts.

The same kinds of effects on cell behaviour can result from mutations in either class of genes, because most of the control mechanisms in the cell involve both inhibitory and stimulatory components. Some of the pathways important in cancer carry signals from a cell’s environment others are responsible for the cel’s internal programs such as those that control the cell cycle or cell death

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 to oncogenes, deregulation of tumour suppressor genes and DNA repair genes and ‘immortalisation. 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 synthesised and degraded in a cell cycle-dependent manner. Cyclin-CDK complexes are further tightly regulated by CDK inhibitors.

The genes that have been implicated in carcinogenesis are divided into two broad categories oncogenes and tumour suppressor genes but also include DNA repair genes Genes that promote autonomous cell growth in cancer cells are called oncogenes, and their normal cellular counterparts are called proto-oncogenes. Proto-oncogenes are physiologic regulators of cell proliferation and differentiation while oncogenes are characterised by the ability to promote cell growth in the absence of normal mitogenic signals. Their products, oncoproteins, resemble the normal products of proto-oncogenes with the exception that oncoproteins are devoid of important regulatory elements. Their production in the transformed cells becomes constitutive, that is, not dependent on growth factors or other external signals. Proto-oncogenes can be converted to oncogenes by several mechanisms including point mutation and gene amplification resulting in:Cancer development is based on the accumulation of somatic mutations over lifetime. Germ line mutations are typically not involved, but in very rare cases of inherited cancer predisposition, they are contributing to disease progression.