Obesity is a real epidemic and a public health problem, defined by The Obesity Society (TOS) as a disease [10], and not only an underpinning of major chronic diseases, but a serious debilitating condition in its own right.

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 (or "sick fat") 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.

Adiposopathy is sustained by adipocyte hypertrophy, visceral adiposity and/or ectopic fat deposition and secretion of hormones, like leptin, and proinflammatory protein, like the plethora of cytokines, that in turn may lead to metabolic disease.

Therefore, we can classified obesity as a primary disease since the adiposopathy determines the dysregulation of the metabolic pathways [15].

Metabolic diseases most associated with primary obesity contribute to atherosclerosis, hypertension, dyslipidemia, diabetes type II, hyperandrogenemia in women and hypoandrogenemia/hyperestrogenemia in men.

EFFECT OF DRUG THERAPY ON SECONDARY OBESITY

Obesity is a chronic disease, and it requires chronic therapy. Hypertension, dyslipidemia, diabetes and cardiovascular diseases are leading causes of mortality in the modern world. All of them are strongly linked to obesity. 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 During the history of antiobesity drugs, many of them were withdrawn because of their side effects.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 ON SECONDARY OBESITY

The prevalence of overweight and obesity in children with congenital heart disease was similar to that described in the literature for children with non-congenital disease.In a population of patients with congenital heart disease in the U.S., researchers found a prevalence of more than 25% of obese and overweight children.However, in a study published six years ago, the excess weight rate of a population of children and adolescents in Belgium was 7.6%.
In Brazil, the high prevalence of excess weight in children and adolescents in general has been a reason for concern, because other associated risk factors for ischemic heart disease, such as hypertension, glucose intolerance, dyslipidemia, and physical inactivity have emerged .
The presence of modifiable risk factors for ischemic heart disease in this population, such as an abnormal lipid profile (high total cholesterol/LDL/triglycerides, low HDL) and excess weight may lead individuals with congenital heart disease to have a combination of risks that may persist into adulthood.These modifiable risk factors have been well discussed in the literature about children without heart disease.
The presence of chronic diseases in families of patients with congenital heart disease is an additional risk factor for ischemic disease,similarly to what occurs for healthy children/adolescents and adults in general.The presence of obesity in mothers in our study was directly related to their children's excess weight. This findings could represent both biological/ genetic characteristics and family lifestyles.In a study comparing three generations of families, there was a

strong significant relationship between the BMI of mothers and children, thus suggesting the discussion of inheritance of family patterns and lifestyle, as well as family phenotypes.In another study evaluating the role of parents in the treatment of childhood obesity, it was found that distorted maternal perception leads mothers to see their children's excess weight as normal, making it difficult for them to admit their children need treatment.
Approximately half of children and adolescents were irregularly active or sedentary. In many cases, physical activity may be limited by the parents anxiety.
Passive smoking was detected in almost half of the population studied, a rate much higher than in a survey conducted over the past decade, in which more than 25% of children lived with at least one smoking parent. Exposure to secondhand smoking in children causes higher rates of pneumonia, ear infections, sudden infant death syndrome, asthma, and other negative health effects .In addition, children's airways are more vulnerable, suffering dramatically with the effects of secondhand smoking.children exposed to smokes are liable to be smokers.
It is important to consider that factors present since the children's conception may contribute to "programming" of disease in adult life.

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 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
4. 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).

Lifestyles can affect causes of cancer
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.

Discussion of the causes of cancers necessarily involves an examination of the molecular machinery in cells that guides the basic processes of proliferation (increase in cell number by cell division), differentiation (cell specialization into different tissue types), and apoptosis (programmed cell death). Those processes are guided by two innate programs in cells, the genetic code and the epigenetic code. In cancer each of those codes ultimately becomes altered regardless of whether the disease originated with an external or internal factor. Indeed, a fundamental characteristic of a tumour cell is that it begets a tumour cell. In other words, cancer, once manifest, becomes an inherited disease of the cell and is therefore self-perpetuating. The hereditary nature of cancer at the cellular level explains why alterations have been found in both the genetic and the epigenetic codes in tumour cells. The number of alterations seen in the coded programs increases as tumours progress to more advanced stages. Their existence and accumulation also explain why principles of evolutionary theory provide insights of practical significance for cancer biology.