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1. **Primary obesity** is a [medical condition](https://en.wikipedia.org/wiki/Medical_condition) in which excess [body fat](https://en.wikipedia.org/wiki/Adipose_tissue) has accumulated to an extent that it may have a negative effect on health. People are generally considered obese when their [body mass index](https://en.wikipedia.org/wiki/Body_mass_index) (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 [kg](https://en.wikipedia.org/wiki/Kilogram)/[m2](https://en.wikipedia.org/wiki/Square_metre); the range 25–30 [kg](https://en.wikipedia.org/wiki/Kilogram)/[m2](https://en.wikipedia.org/wiki/Square_metre) is defined as [overweight](https://en.wikipedia.org/wiki/Overweight). Primary **obesity** is most commonly caused by a combination of excessive food intake, lack of physical activity, and genetic susceptibility.
2. CONGENITAL SYNDROME: Congenital leptin deficiency is a form of monogenic obesity characterised by severe early-onset obesity. Congenital leptin deficiency is a condition that causes severe obesity beginning in the first few months of life. Affected individuals are of normal weight at birth, but they are constantly hungry and quickly gain weight. Without treatment, the extreme hunger continues and leads to chronic excessive eating (hyperphagia) and obesity. Beginning in early childhood, affected individuals develop abnormal eating behaviors such as fighting with other children over food, hoarding food, and eating in secret.

DRUG THERAPY: **Drug**-**induced** weight gain is a serious side effect of many commonly used **drugs** leading to noncompliance with therapy and to exacerbation of comorbid conditions related to **obesity.** Some medicines might stimulate your appetite. This **causes** you to eat more **and** gain extra weight. Some medicines might affect your body's metabolism. This **causes** your body to burn calories at a slower rate

1. ETIOLOGY OF CANCER: Cancer is a pathologic accumulation of clonally expanded cells derived from a common precursor. The fundamental cause of all cancers is genetic damage, which is usually acquired but is sometimes congenital. In general, the genetic dysregulation that gives rise to uncontrolled cell proliferation results from activation of growthpromoting oncogenes and/or deletion/inactivation of growth-inhibiting tumor suppressor genes. Additional contributions to carcinogenesis come from genes that regulate programmed cell death (apoptosis) and genes involved in DNA repair. The most widely accepted theory of cancer development is the Knudson “2-hit” hypothesis, which posits that a mutation in one predisposing gene is necessary but not sufficient for malignancy and that only after development of a second mutation will invasive cancer develop (Fig.  2.1) [1]. In addition, it has become increasingly apparent that cancer cells must evade host immune responses and this can sometimes be exploited to treat cancer. In this chapter, we discuss the major categories of carcinogens and their role in the etiology of cancers, with an emphasis on common ophthalmic cancers. Carcinogenic Agents Although a clearly defined cause is not apparent in the majority of de novo malignancies, it is well documented that carcinogenic agents contribute to many human cancers. These fall into several broad groups, which can be categorized as infective (e.g., viruses), chemical (including occupational, environmental, and therapeutic), electromagnetic radiation (i.e., ultraviolet [UV], X-ray, gamma ray), and immunosuppressive (HIV, immunosuppressive medications). These are shown in Table 2.1. Chemical Carcinogens Several hundred chemicals have been shown to be carcinogenic in humans. Harm from these chemical carcinogens can be a result of occupational (asbestos, aniline dyes), environmental (alcohol, tobacco), or iatrogenic (chemotherapy) exposure. While many carcinogens are directly mutagenic to DNA, most carcinogens undergo activation after exposure to reactive metabolites that are responsible for genetic damage. Environmental Exposure Of the known environmental carcinogens, perhaps the most well-documented agent known to cause cancer is asbestos. This is a naturally occurring mineral that has a broad range of industrial and commercial applications, but aerosolized fibers of asbestos are known to lodge in small airways, causing tissue damage and significantly predisposing exposed individuals to both lung cancer as well as mesothelioma [2]. A number of other agents have been associated with elevated risks of various cancers through epidemiologic studies. For example, several agents have been implicated in lung cancer, including heavy metals [arsenic, cadmium, chromium, and nickel, as well as BCME (bischloromethl ether)] [3]. Aromatic amines such as 4-AMP, 4-aminobiphenyl, naphthylamine, and benzidine are associated with an increased risk of bladder cancer [3]. Aflatoxin and vinyl chloride are associated with liver cancers. An exhaustive list of known environmental carcinogens is beyond the scope of this text but should be considered when obtaining the occupational and social history of patients with newly diagnosed cancer. Behavioral Exposure Tobacco Tobacco is responsible for over 30% of the cancer deaths and represents the single most common cause of preventable cancer. Cigarettes as well as smokeless tobacco are associated with cancers of THE lung.
2. MOLECULAR BASIS OF CANCER: Cancer is a group of diseases characterized by an autonomous proliferation of neoplastic cells which have a number of alterations, including mutations and genetic instability. Cellular functions are controlled by proteins, and because these proteins are encoded by DNA organized into genes, molecular studies have shown that cancer is a paradigm of acquired genetic disease. The process of protein production involves a cascade of several different steps, each with its attendant enzymes, which are also encoded by DNA and regulated by other proteins. Most steps in the process can be affected, eventually leading to an alteration in the amount or structure of proteins, which in turn affects cellular function. However, whereas cellular function may be altered by disturbance of one gene, malignant transformation is thought to require two or more abnormalities occurring in the same cell. Although there are mechanisms responsible for DNA maintenance and repair, the basic structure of DNA and the order of the nucleotide bases can be mutated. These mutations can be inherited or can occur sporadically, and can be present in all cells or only in the tumor cells. At the nucleotide level, these mutations can be substitutions, additions or deletions. Several of the oncogenes discussed below, including the p53, c-fms, and Ras genes, can be activated by point mutations that lead to aminoacid substitution in critical portions of the protein.