Chapter 1

EPIDEMIOLOGY OF CANCER AND AGING

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Epidemiology provides the initial clue to causes and mechanisms of diseases. It is well known that age is a risk factor for most common cancer and that incidence and prevalence of cancer increase with age\(^1\). In this chapter we explore the epidemiology of cancer and aging, in an attempt to understand the biologic interactions of these processes. In particular, we address the following questions:

1. Does aging enhance the susceptibility of older individuals to environmental carcinogens?

2. Is aging associated with increased risk of multiple malignancies?

3. Does the clinical behavior of cancer change with age?

4. Does cancer increase the risk of death of older individuals?

In conclusion we will examine the clinical implications of these questions and propose a research agenda aimed to improve the control of cancer in the older aged person.
1. AGE AND CARCINOGENESIS

The incidence of common cancers increases with age (Figure 1). This association is universal and is observed with the aging of any population around the world. A clear explanation of this phenomenon is the time-length of carcinogenesis, a stepwise process involving the activation of cellular oncogenes, and the suppression of anti-proliferative genes (anti-oncogenes). It is reasonable to assume that the duration of carcinogenesis reflects the number of stages involved in the pathogenesis of different tumors, and that this number be highest for tumors whose incidence peaks late in life, such as adenocarcinoma of the prostate and of the large bowel, or non-melanomatous skin cancer. In the era of chemoprevention and recognition and elimination of environmental carcinogens, an alternative possibility should be considered. These interventions may cause the prolongation of one or more carcinogenic steps and, in so doing; they may delay the development of cancer. For example, the incidence of lung cancer has decreased for individuals less than 60, while it has increased for older individuals. As a result, the peak incidence of lung cancer has become more and more delayed. Interestingly, these changes have paralleled the incidence of smoking cessation in the Western population. In this case it is reasonable to assume that the length of carcinogenesis has increased as a result of a prolongation of the late carcinogenic stages, from reduced intensity of exposure to tobacco smoke. If this hypothesis is correct, one may expect to see a progressive delay in the appearance of common cancer and an increased incidence of neoplasia in advanced ages.

Figure 1. The incidence of common cancers increases with age.