INTRODUCTION

Prostate cancer (CaP) is the second leading cause of cancer death in US males, and it accounts for almost 30% of all cancers diagnosed in men. During the 1990s, the CaP incidence rate rapidly increased, which has been mainly attributed to heightened screening using prostate-specific antigen (PSA). Established risk factors for CaP are primarily nonmodifiable; these include older age; race, with greatest risk being among African Americans followed by Caucasians; and a family history of CaP. Possible modifiable risk factors are now beginning to emerge, including those that increase risk, such as intake of saturated fat and calcium, and those that decrease risk, such as selenium, vitamin E, and tomato products, which contain the carotenoid lycopene. Androgens, vitamin D, and insulin-like growth factor-1 (IGF-1) have been explored recently for their relationship with CaP risk; these hormonal systems may be influenced by external factors, such as diet, as well as genetics.

In autopsy studies, the prevalence of latent prostate tumors (i.e., microscopic foci of well-differentiated cancer cells) is fairly high in young as well as older men. Prostate tumors vary widely in their growth rate and ability to metastasize. Moreover, some risk
factors, for example, short androgen receptor (AR) gene CAG repeats and possibly calcium intake and cigarette smoking, are more strongly associated with aggressive CaP (i.e., those of high stage or poor histologic grade at diagnosis) than to the development of CaP. These observations suggest that it is important also to identify those factors that act on tumor progression resulting in invasion, metastasis, and death. Such factors may overlap with or may be distinct from those factors that promote tumor development.

In this chapter, we describe the epidemiology of and risk factors for CaP. We focus in particular on suspected dietary and lifestyle contributors, possible genetic variation in the population resulting in differential, but modest, susceptibility to this disease, and those dietary, lifestyle, and genetic factors that may account for the observed racial/ethnic variation in CaP risk internationally and in the United States.

**DESCRIPTIVE EPIDEMIOLOGY**

**International Rates**

CaP incidence rates vary almost 70-fold around the world (1). The lowest rates are observed in the Far East and on the Indian subcontinent, whereas the highest rates occur in the Western Europe, Australia, and North America. Adjusting the rates to a common age standard (younger than that observed in the United States), the incidence rate for CaP was approx 1/100,000 men annually in China compared to 62 and 82/100,000 for US whites and African Americans, respectively, in the late 1980s (1). Some of the highest CaP rates are found on Caribbean islands, including Trinidad and Tobago (2) and Jamaica (3), where the populations are of African descent. Notably, rates in African countries, such as Nigeria, appear to be substantially lower (4). Mortality rates vary greatly among nations as well; in Japan 4/100,000 men die of CaP/yr, whereas in Canada, France, Germany, the United Kingdom, and the United States, the mortality rate ranges from 16 to 18/100,000 men annually (5). A gradient in the CaP mortality rate exists between Northern Europe, where the rates are more than 20/100,000 men annually, and Southern Europe, where the rates are half that (5).

Some of the disparity in CaP incidence rates among countries is likely the result of differences in medical practice leading to differential rates of detection of subclinical tumors. The frequency of these latent tumors does not appear to vary dramatically among populations (6), including in autopsy series of black populations in West Africa and in the United States (7). Migration studies show that men of Asian heritage living in the US are at lower risk for CaP than white Americans, but at greater risk of the disease than men of similar ancestries living in Asia (8,9). Japanese immigrants living in Los Angeles County, CA, have CaP rates more comparable to individuals with similar ancestry, but who were born in the US than to men in Japan (10), and these rate differences do not appear to be entirely owing to differences in detection of early stage tumors between the United States and Japan (11). The changing CaP rates among migrants, despite a common prevalence of early stage lesions, suggest that endogenous or exogenous factors affecting tumor growth and progression, rather than initiation, may vary among countries as well.

**United States Rates**

Using data collected by the National Cancer Institute’s Surveillance, Epidemiology, and End Results Program, the American Cancer Society estimates that 29% of all new cancer diagnoses among US men in 1999 will be CaP, amounting to 179,300 cases (2).