Introduction

Prostate cancer is a major healthcare problem worldwide, especially in the industrialized countries of the Western world. Prostate cancer has become the most common type of cancer among men and is the second leading cause of years of life lost from cancer in males [58]. Incidence estimates for the year 2000 indicate prostate cancer newly affected 542,990 men worldwide that year, 204,313 of whom have since died. Early detection and treatment of prostate cancer could theoretically reduce the burden of this potentially disabling and deadly disease. However, because no conclusive, direct evidence demonstrates that early detection and treatment improve length or quality of life, the value of prostate cancer screening remains controversial.

Prostate cancer is primarily a disease of elderly men. About 85% of all cases of prostate cancer are diagnosed in men older than 65 years, and 90% of deaths due to this disease are in men over age 65. Prostate cancer is diagnosed in very few men younger than 50 years (<0.1% of all patients with prostate cancer) [2].

There are large differences in the incidence of prostate cancer worldwide. Incidence is very high in North America and northern Europe (peaking at 63 per 100,000 white men and 102 per 100,000 African-Americans in the U.S.), but much lower in Asia (10 per 100,000 men in Japan) [18]. The lifetime risk for clinical prostate cancer among men in the United States is approximately 10%; approximately 3% die of this disease [43]. Despite these differences, the microfocal incidence of prostate cancer on autopsy is similar worldwide. Autopsy and cystoprostatectomy studies have shown a prostate cancer prevalence of 30% to 80%, depending on age, based on histological examination. For men over age 50 years, the weighted average of prostate cancer prevalence based on autopsy studies is 30% [13]. Approximately 30% of these cancers are believed to be clinically significant [48]. Thus, the risk for a 50-year-old man with a 25-year life expectancy of having microscopic cancer is approximately 30%; of having clinically evident disease, 10%; and of dying of prostate cancer, 3% [60]. The disparity between the 30% with microscopic cancer and the 3% lifetime risk of death shows the difficulty in distinguishing cancer as an indolent disease. Patients with very aggressive tumors, (i.e., high Gleason grade) have a significant risk of dying of prostate cancer. Patients with relatively non-aggressive prostate cancer have a smaller risk of dying of the disease [39, 49].

Epidemiology and Regional Variation

Prostate cancer is one of the few malignancies for which the incidence varies widely across different parts of the world. Hsing and colleagues classified 15 countries according to their level of prostate cancer risk. High-risk countries included the United States, Canada, Sweden, Australia, and France. Medium-risk countries included most of Asia [27]. The same group of investigators also examined trends in the incidence from 1973 to 1992. From 1998 to 1992, when prostate-specific antigen (PSA) testing became widespread, the incidence in the high-risk countries ranged from 48 to 137 per 100,000 person-years, while the incidence in low-risk countries ranged from 2.3 to 9.8 per 100,000 person-years. In general, prostate cancer incidence rose in all countries during these years, with the increment increasing by between 16.2% and 113.3% over the period [27].
The large geographical difference in the clinical incidence of prostate cancer, coupled with the marked discrepancies between the incidence of latent microfocal disease and clinical disease, raises the concept that environmental factors may play an important role in the prevention and/or progression of the disease. In the majority of men with pre-existing microfocal disease, the growth is stimulated. It seems likely that these differences are only rarely due to genetic factors [36].

Among the environmental factors that are supposed to be critical in the development of prostate cancer, nutrition is suspected to play a major role. Dietary habits vary greatly across the world. There is increasing evidence to suggest that several elements of the diet may play an important role in the prevention and/or progression of prostate cancer (Table 5.1) [53]. To date there have been 14 well-performed, case-control studies involving 4,797 prostate cancer patients and 5,779 control subjects [18]. Eleven of these studies have demonstrated a positive association between increased dietary fat or specific fatty foods and a higher risk of prostate cancer with an odds ratio (OR) of 1.3–3.4. Epidemiological studies have suggested that vitamin E may also influence the development of prostate cancer [25]. However, the preventative effect of dietary components has not been definitely demonstrated in any specifically designed prostate cancer-focused studies that would withstand rigid scientific scrutiny.

### Screening and Early Detection

There is a worldwide attempt to improve the terrible outcome of prostate cancer. We think that prostate cancer, when detected in a localized stage, can be cured or the survival rate and the patients’ quality of life can be improved. If we diagnose prostate cancer in a late stage, it means an incurable status. Two approaches are accepted to achieve this goal at present: early detection and systematic screening. The first means evaluation on patients’ request or as part of any other medical examination. The second is a planned examination of the affected population. The same clinical examinations are used in both methods.

These are the digital rectal examination (DRE), the serum PSA level measurement, and transrectal ultrasound.

The latest has not been used for years because of the very low specificity, invasiveness, and high cost. DRE has low sensitivity alone, so it is not recommended for screening, but together with (PSA) testing, it improves the detection rate. PSA is a glycoprotein with serine protease activity produced primarily by epithelial cells lining the acini and ducts of the prostate gland. PSA is secreted into the lumina of the prostatic ducts and is present in high concentrations in seminal fluid. Plasma concentrations are normally low but are increased by conditions that disrupt normal prostate structure and function (i.e., inflammation, infection, hyperplasia, prostate cancer). Androgens regulate expression of the PSA gene. Men who have regular PSA tests have a much higher chance of finding out that they have prostate cancer compared to men who do not have PSA tests. With the use of an effective testing procedure, systematic screening shows a temporary but significant increase in the incidence, because we diagnose those patients whom we would otherwise diagnose clinically at a later time. Thus, lead-time is produced, which can last from 4 to 10 years. After the second or third screening round, lowering of the incidence is to be expected. This was seen in the United States’ statistics very well [56]. The decreasing of mortality is expected only years after that. The cause of the lead time and the aggressive early treatment produce additional survival time, for which the