Editorial

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We can all change our diet, but none of us can change our parents. In other words, there are both modifiable and non-modifiable risk factors for prostate disease. In this issue of *Prostate Cancer and Prostatic Diseases* several new observations are made about both these important components of the aetiology of prostate cancer. Martin *et al* provide evidence that carcinogens, of the type that all of us may be exposed to in the environment, can be activated by prostate cells *in vitro*, and cause demonstrable genetic damage. Oxidative stress from excessive consumption of unsaturated fats is almost certainly an important component of the recent 'epidemic' of prostate cancer that Western societies have witnessed, but there is little evidence that lessons have yet been learned in terms of significant dietary modification.

Benign prostatic hyperplasia (BPH), by contrast, has been postulated to result from the fluctuations in androgens and estrogens that are associated with ageing. Smith *et al* report that fluctuations in estradiol can modify expression of estrogen receptors, androgen receptors and fibroblast growth factors, and thereby potentially induce the type of stromal hyperplasia that characterizes this benign neoplasm. Over the last decade alpha₁-adrenoceptor antagonists, such as doxazosin and terazosin, have been increasingly used in the treatment of this highly prevalent condition. Now Anglin *et al* suggest that these agents may also have a role in the treatment of prostate cancer by virtue of their ability to induce apoptosis possibly through activation transforming growth factor beta (TGF β) with exciting implications.

Race is a non-modifiable risk factor for prostate cancer. African-American men seem especially susceptible to the disease, but the reasons for this are still not clear. In a small but elegant study Lotan and Roehrborn report that PSA clearance rates after radical prostatectomy are no different in African-Americans than Caucasian men. However, other racial differences in PSA expression may be more significant. Egawa *et al* report that although percent free PSA levels predict the probability of a positive prostate biopsy more accurately than the total

PSA value, these probabilities are lower than those reported in Western countries.

Both BPH and prostate cancer are characterised by abnormal growth patterns, which necessitate the development of new blood vessels. This process is known as angiogenesis. Significantly therefore, in this issue Walsh *et al* report that vascular endothelial growth factor (VEGF) staining is more prevalent in BPH than prostate cancer samples. Bono *et al* suggest that microvessel density (MVD) can be used to predict the risk of disease recurrence after radical prostatectomy.

The accurate staging of clinically localized prostate cancer, either before or after treatment still poses significant clinical problems. Unfortunately the paper by Ponsky *et al* confirms that the ProstaScint™ scan, for which high hopes have been held, does not seem capable of accurately predicting the risk of lymph node metastases prior to surgery. Johnstone *et al* also highlight the uncertainty that surrounds not only the timing but also the choice of therapy in cases of PSA relapse after original definitive treatment.

In patients with metastatic disease accurate staging is no longer such a problem, but it can be difficult to obtain samples of tumour cells from the bone marrow for research purposes. Brown *et al* report the use of bone marrow trephine biopsy guided by previous bone scan to target the affected areas. The prognosis, of course, for men with metastatic prostate cancer is still poor. Koff *et al* emphasize that clinical stage and nadir PSA values can be used to accurately predict time to recurrence, but that race has no influence. Somewhat surprisingly, the presence of pulmonary metastases is reported by Nakamachi *et al* not to adversely influence survival. In a final paper Piper *et al* document the considerable economic impact of advanced prostate cancer. Of course to these we must add the incalculable emotional costs, not only to the patients, but also to their families, friends and relatives.

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