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Editorial

One especially helpful model for the ever-growing international problem of prostate cancer is to consider it as a 'continuum' which encompasses pre-malignancy, locally invasive disease, metastatic cancer and eventually hormone refractory disease. Overall, our ability to stem the rising tide of death and disability from this disease will depend on our ability to understand the molecular mechanisms that underlie this progression. At present, although we are capable of meaningful interventions at almost every stage of the disease, our treatments are ultimately unsuccessful unless we can diagnose and treat the cancer at its earlier stages.

In this issue of *Prostate Cancer and Prostatic Diseases* a number of new insights are provided to help us understand this continuum. In terms of pre-malignancy, Slater *et al* provide an innovative report of the use of P2X immunostaining for the detection of malignancy before the usual diagnostic markers of cancer are present. Green *et al* confirm that prostatic intraepithelial neoplasia (PIN) in radical prostatectomy specimens is associated with prostate cancer in a linear fashion, a finding which emphasises the need for clinicians to follow up carefully and consider rebiopsy of the prostate inpatients with PIN reported on their original diagnostic evaluation. The question as to how these type of observations might translate into effective chemoprevention for our patients is discussed by Liu *et al*.

The thorny question of what initiates the changes that lead to the progression of prostate cancer is also examined. Mydlo *et al* report that both serum testosterone and body mass index (ie obesity) are positively associated with the risk of prostate cancer. Since the incidence of obesity is increasing in most societies internationally, the latter finding is of particular concern.

Angiogenesis is now well established as a pivotal event in prostate cancer development and progression. The ability to secrete angiogenic cytokines is a critical initiating step, resulting in vascular hypermeability, capillary basement membrane breakdown, and endothelial cell migration and proliferation. Dall'Era *et al* raise the important question as to whether cell cultures derived from epithelial cells secrete angiogenic cytokines in as representative a manner as mixed epithelial and stromal cell lines.

The only means we have of achieving a long-term cure for prostate cancer involves either surgery or radiotherapy. A report from Soulie *et al* in France confirms that there are marked regional variations in the utilisation of surgery, probably resulting from varying preferences of both physicians and patients. Hormonal therapy is the most commonly employed therapy for prostate cancer and a paper in this issue confirms the safety and efficacy of the antiandrogen nilutamide in a post-marketing surveillance setting. The recent evidence that another antiandrogen, bicalutamide, can significantly delay PSA progression and the time to development of metastases, alluded to in our 'Hot News' section, seems likely to increase the usage of this agent in an earlier setting in the cancer continuum than has been the case up to now. Antiandrogens, because they maintain serum testosterone levels, may cause fewer problems with osteoporosis than LHRH analogues.

Finally, we must not neglect the more common but much less hazardous condition of benign prostatic hyperplasia (BPH), which has an important impact on quality of life and still keeps urologists busy all over the world. Vallancien *et al* report that there is a shift in preference in France towards the newer alpha-1 adrenoceptor blockers and that these medicines are associated with a lower switch rate than either plant extractors or 5 alpha-reductase inhibitors.

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