



## Editorial

At the beginning of this century prostate cancer was rare. As the millennium draws to its close prostate cancer has become the most commonly diagnosed malignancy in men and the second commonest cause of cancer death after lung neoplasms. How close are we to understanding the causes of this dramatic shift in disease prevalence? Population demographics provide one answer. During the past one hundred years life expectancy of men has risen from 49 y to 73.4 y. Therefore many more men are living long enough for prostate cancer to manifest itself. Alterations in lifestyle and environment have also been important, with an increased amount of fat in our diet and more industrial pollution.

From this second issue of the second volume of *Prostate Cancer and Prostatic Diseases* some insight into the molecular mechanisms of prostate cancer may be gained. The transgenic adenocarcinoma of the mouse prostate (TRAMP) model reported by Gingrich *et al* in which both the p53 and Rb tumour suppressor genes have been deleted turns out to be an excellent model in which to study the stochastic molecular progression of prostate cancer from prostatic intraepithelial neoplasia (PIN) through to androgen independent metastatic cancer.

The signaling pathways that stimulate growth proliferation and tumourigenesis in the prostate are also considered. In an elegant review Tatoud looks at the evidence for a central role of insulin-like growth factors (IGF) and their recently described family of receptors and binding proteins. Even poorly differentiated prostate cancer cells continue to elaborate and secrete prostatic specific antigen (PSA). In an original article, Vaisanen *et al* describe the characterisation and processing of PSA and human glandular kallikrein by LNCAP cells.

It is PSA testing that first raises the suspicion of prostate cancer in most cases these days, but transrectal biopsy is required to confirm the diagnosis. In a prospective, but not randomised study, Larsson *et al* provide evidence for the cost-effectiveness of combined two-dosage oral ciprofloxacin and metronidazole as prophylaxis against urinary tract infection and septicaemia. This convenient regime should probably become standard clinical practice.

Also in this issue are two articles concerning various therapeutic options for patients with lower urinary tract symptoms due to benign prostatic hyperplasia (BPH). Medical therapy is fast becoming first line treatment for uncomplicated BPH, but which patients should receive alpha blockers and who should be treated with finasteride? As discussed in the review article, those with larger prostates respond better to 5 alpha-reductase inhibitors and there is now good evidence that long term therapy with finasteride reduces the risk of acute urinary retention and the need for surgery.

Not every patient wants to take tablets long term for BPH-related symptoms. Some prefer a 'one-off' approach, but are still reluctant to consider transurethral resection of the prostate (TURP). In their article, d'Ancona *et al* report the results of high energy thermotherapy for the treatment of BPH. The results suggest that this minimally invasive approach may constitute an important halfway house between medical therapy and traditional transurethral surgery.

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