

sensitivity of these tumour cells to hormonal stimulation. This treatment results in the decrease in plasma testosterone, and although the effectiveness of the therapy might suggest that this androgen or its prostatic metabolites may have some aetiological role in the development of cancer of the prostate, there is at present little evidence to incriminate any hormonal imbalance. It is of interest, however, that McNeal (1970, Proc. 3rd Tenovus Workshop, p. 23) has recently challenged the traditional view that carcinoma of the prostate originates from atrophic, androgen deprived epithelium of the gland in the elderly man. Instead, he suggests that carcinoma arises from active glandular epithelium, again directing attention to an aetiological role of prolonged androgenic stimulation.

It is obvious therefore that further studies are required of the relationship between prostatic disease and endocrine function. Investigations from these laboratories have to date failed to show any major significant differences in the levels of prolactin, LH, FSH, androstenedione, testosterone or oestradiol-17 β in plasma from normal men and those with either benign prostatic hyperplasia or carcinoma of the gland. More detailed studies in which plasma hormone levels in patients on oestrogen therapy were carefully monitored indicated differences in the endocrine response between patients and suggested that such studies may provide more relevant and interesting data.

For example, the prolactin levels in the plasma were found to be markedly elevated in certain patients after initiation of oestrogen therapy. Many earlier studies have directed attention to the possible relationship between prolactin and the prostate, and it is also known that hypophysectomy can still produce regression of prostatic carcinoma even after orchidectomy and adrenalectomy (Fergusson, 1972, in *Endocrine Therapy in Malignant Disease*, p. 237).

The eventual lack of responsiveness to oestrogen therapy in many patients, resulting in progression of the disease, has been considered due to secondary production of androgen by the adrenal. A series of Synaethen stimulation tests on patients before and during diethylstilboestrol therapy indicated an increased capacity of the adrenal to produce testosterone during treatment.

Little information exists on human

prostatic steroid receptor complexes. Such steroid receptor studies in relation to hormone action within the prostatic cell are particularly valuable, and may eventually indicate the mechanism by which oestrogens can directly influence the metabolism of the prostate.

ASSAY OF STEROID HORMONE RECEPTOR IN CANCERS, METHODOLOGY AND PERSPECTIVES. R. K. WAGNER, A. HUGHES and P. W. JUNGBLUT. Max-Planck Institute for Cell Biology, Wilhemshaven, Germany.

The assay of steroid hormone receptors is now widely used as an index of hormone dependency of certain cancers, since it is known that receptors are required for the full course of hormonal action. A variety of procedures have been employed for evaluating the spare (unused) receptor content of tissues. They make use of the high affinities of receptors for their respective steroids, which allows the measurement of labelled steroid receptor complexes, after separation from excess of free steroid and dissociation of unspecific complexes. Another requirement for accurate receptor assay is the ability to distinguish between steroid receptor complexes and those formed with specific plasma proteins, contaminating tissue extracts. All requirements are met in a single step by the very sensitive agar gel electrophoresis at low temperature, which allows *e.g.* the determination of oestrogen- and androgen receptors in mammary and prostate cancer in the presence of sex hormone binding protein (Wagner, 1972). In assessing the predictive value of quantitative receptor assays in cancers, it has to be considered that the concentration of spare receptor depends not only on the uniformity of the tissue specimen investigated but also on the hormonal status of the patient. Hormone action is always accompanied by receptor depletion and replenishment. The latter is due to *de novo* synthesis rather than to recycling of used receptor. Continuous hormone supply can thus result in low actual concentrations of spare receptor, which should not be mistaken as an indicator of "marginal" hormone dependency.