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REVIEW

Rationale for using aromatase inhibitors to manage benign prostatic hyperplasia. Experimental studies

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Today, human benign prostatic hyperplasia (BPH) is considered primarily to be a disease of the stroma, in which estrogens are thought to play a considerable causative or permissive role. The growing incidence of BPH with increasing age coincides with a shift in the androgen:estrogen ratio in favor of estrogens, not only in terms of serum hormone values, but also in the prostate itself. Furthermore, evidence has been provided for a preferential accumulation of estrogens in the stroma of human hyperplastic tissue, and the presence of an estrogen receptor satisfying the classical criteria of high affinity and low capacity has been demonstrated. Also, animal studies have emphasized the potential role of estrogens in the pathogenesis of BPH. Experimentally, stimulation of the stroma, particularly of smooth muscle, can be induced by aromatizable substrates, such as androstenedione, in the prostates of beagles and cynomolgus monkeys. These effects can be antagonized by aromatase inhibitors, such as atamestane. In addition, the increase in intraprostatic estrogen concentrations and immunohistochemically detectable estrogen receptor content induced by androstenedione in intact dogs is completely reversed by simultaneous treatment with atamestane. In conclusion, clinical data, as well as that from animal models, emphasize an important role for estrogens in the development of BPH. Estrogen deprivation might, therefore, represent a useful treatment for human BPH.