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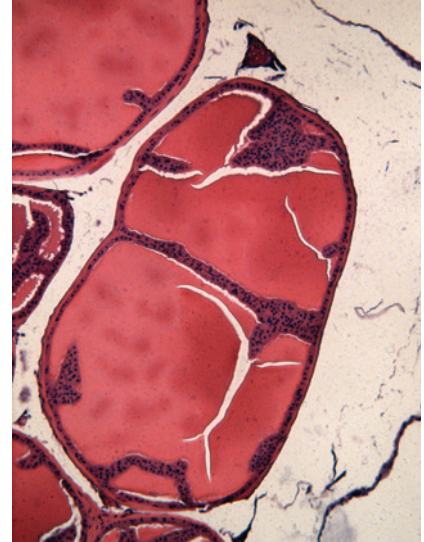
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ABOUT THE COVER

Most prostate cancers are slow growing, with the first histological signs appearing in young men and progressing over a lifetime. Because it progresses slowly, prostate cancer is a candidate for primary prevention through lifestyle interventions that include diet. The role of vitamin D in prostate cancer is controversial. Cell and animal studies show that vitamin D-mediated activation of gene transcription through the vitamin D receptor (VDR) antagonizes prostate cancer cell biology and slows tumor formation, but human population studies correlating vitamin D status to prostate cancer risk or mortality are inconsistent. Fleet and colleagues (beginning on page 343) tested whether disrupting vitamin D signaling by tissue-specific VDR deletion or with diets that affect vitamin D metabolite levels could increase prostate cancer development in TgAPT₁₂₁ mice. The cover image shows an H&E stained anterior prostate section from TgAPT₁₂₁ mice (100X). The image shows two side-by-side glands with predominantly normal prostate epithelium and an area of hyperplasia in the top gland (focal crowding of epithelial cells with normal nuclear morphology). Normally, these lesions progress to high grade PIN and areas of focal adenocarcinoma over 6 months. The study shows that VDR deletion, low vitamin D diets, and high calcium diets that suppress 1,25-dihydroxyvitamin D production all increase the progression to high grade PIN and adenocarcinoma. Thus, the study indicates that the benefits of vitamin D may be present well before the expression of clinical symptoms and pathology.



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