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JOURNAL ARTICLE

Vitamin D and prostate cancer

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The original hypothesis of Schwartz and Hulka (1990) proposing that vitamin D deficiency may be a risk factor for prostate cancer has triggered many studies. Epidemiological studies have supported this hypothesis with findings that sunlight exposure is inversely proportional to prostate cancer mortality and that prostate cancer risk is greater in men with lower levels of vitamin D (Hanchette and Schwartz, 1992; Corder et al, 1993; Ahonen et al, 2000). Prostate cancer cells express receptors for 1,25(OH)2D3 and some cell lines are growth inhibited when treated with 1,25(OH)2D3 (reviewed in Blutt and Weigel, 1999). The mechanism of action of these growth inhibitory effects of 1,25(OH)2D3 in LNCaP cells involves G1 accumulation, induction of quiescence, and an increase in apoptosis of the cancer cells (Blutt et al, 1997, 2000a; Zhuang and Burnstein, 1998). In vivo, 1,25(OH)2D3 and its analogs slow tumor growth and hinder metastasis of prostate tumors in rodent models (Schwartz et al, 1995; Getzenberg et al, 1997; Lokeshwar et al, 1999; Blutt et al, 2000b), and 1,25(OH)2D3 may have clinically relevant effects (Gross et al, 1998). More work is required to elucidate the mechanism of 1,25(OH)2D3 action in prostate cancer cells and to identify optimal 1,25(OH)2D3 analogs in a search for compounds with a better separation of growth inhibitory effects from hypercalcemic effects.

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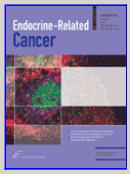

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