Growth Response of Hodgkin's Lymphoma to Vitamin D3 and Its Chemical Analogs (EB1089 and Calcipotriol) In Vitro

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ABSTRACT

Vitamin D3 receptor (VDR) signaling has attracted much attention in cancer therapies, including therapies for B-cell lymphomas. Deficiency in vitamin D3 (1,25-dihydroxyvitamin D3 [VD3]), the main ligand for VDR, is a negative prognostic factor in some lymphomas. The objective of this study was to investigate the effects of VD3 and its vitamin D3 analogs (VDAs) on the growth of Hodgkin's lymphoma (HL). Immunofluorescence and Western blot were used to study expression of VDR in primary tumor cells and HL cell lines, respectively. Cells were treated with 10 μM of VD3 or VDAs (EB1089 and calcipotriol) for 72 hours, and changes in cell growth were measured by water-soluble tetrazolium 1 cell proliferation assay. One-way analysis of variance and F statistics were used to test for differences between the means as defined by $P < 0.05$. The results showed that VDR is highly expressed by the Hodgkin's and Reed-Sternberg cells, the primary tumor cells in HL. However, low but varying levels of VDR expression were detected in the HL cell lines HDLM2, Hs445, KMH2, and L428. VD3 itself produced only a modest decrease in cell growth, whereas a significantly lower decrease in cell growth was the result of treatment with calcipotriol and EB1089. Although ligand-stimulated VDR causes nuclear accumulation of VDR (nuVDR), we did not detect any difference between nuVDR for dimethyl sulfoxide control and VD3 treatment. However, calcipotriol and EB1089 resulted in higher levels of nuVDR. These data suggest that VDAs may be important in HL therapy.

ABBREVIATIONS: HL - Hodgkin's lymphoma, nuVDR - nuclear accumulation of VDR, VD3 - 1,25-dihydroxyvitamin D3, VDA - vitamin D3 analog, VDR - vitamin D3 receptor.

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