AT10 (vitamin D2); in which AT10 was considered as equivalent to vitamin D2. AT10 (dihydrotachysterol) is quite different from vitamin D2 which is ergocalciferol. Although both compounds, after their hydroxylation in the liver and kidney would be effective on serum calcium levels, they differ in that AT10 may be rachitogenic in long usage but vitamin D is the compound recommended for the prevention and treatment of rickets.

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Drs Thong and Robertson comment:

We thank Professor Ozsoylu for his correction of our error. AT10 (dihydrotachysterol) is a reduction product of vitamin D and differs from vitamin D in the geometrical configuration of the A ring. This product has been used in the treatment of hypoparathyroidism for many years, and is preferred to calciferol by some because of its more rapid onset of biological activity and its more rapid excretion (Root and Harrison, 1976). We have seen no evidence of a rachitogenic effects in patients with hypoparathyroidism treated with either dihydrotachysterol or calciferol, nor have we seen such a report in the literature.

Perhaps Professor Ozsoylu's concern is based on the *in vitro* work of Trummel *et al.* (1971) and Reynolds *et al.* (1973) who showed a direct effect of both dihydrotachysterol and its 25-hydroxylated metabolite, 25-hydroxy-dihydrotachysterol on bone resorption in culture, which was greater than the effect of 25 hydroxycholecalciferol.

Both calciferol and dihydrotachysterol have been used effectively in the long-term management of hypoparathyroidism. Since, in the absence of parathyroid hormone, the production of the most active form of vitamin D, 1, 25-hyhidroxycholecalciferol is impaired, a more rational form of therapy would be this product or its synthetic analogue, 1α-hydroxycholecalciferol which have been shown to be effective. These products are not yet generally available (Russell *et al.*, 1974).

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