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ROLE OF VITAMIN D IN LACTATION – A Review

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ABSTRACT

Vitamin D has traditionally been viewed as a fundamental hormone in the regulation of phosphorus and calcium and bone metabolism. In recent years, the discovery of a new world of extra skeletal and particularly immune modulator effects renewed the interest of research on vitamin D. In the present experiment we are studying the role of vitamin D in pregnancy.

***Keywords*:** Vitamin D , Calcium levels

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INTRODUCTION

Vitamin D, synthesized in the skin, enters the circulation bound to vitamin D-binding

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protein. Dietary vitamin D2 or D3enters the circulation through lymphatic system. Subsequently both vitamins D2 and are metabolized similarly In the liver, vitamin D is metabolised by vitamin D-25-hydroxylase to form 25-hydroxyvitamin D J25(OH)d3. The enzyme is located in the mitochondrial and microsomal fractions of the hepatic cells (Ponchon and De Luca, 1969; De Luca, 1984). Although there are a few reports of the presence of extrahepatic vitamin D-25-hydroxylase in the chick and the rat (Tucker et al. , 1973 ; Olson et al., 1976), the liver seems to be the only site of 25(OH)D synthesis in the humans. The reserve capacity of vitamin D-25-hydroxylase in the liver is substantial. Severe parenchymal damage is required to lower the level of plasma 25(OH)D (Long et al., 1976). The enzyme vitamin D-25-hydroxylase does not seem to be tightly regulated since the circulating levels of 25(OH)D very with the amount of dietary intake of vitamin D or the degree of solar exposure (Holick et al., 1986). Although bone and muscle accumulate about 60% of the injected dose of vitamin D (De Luca»1977) and gross skeletal abnormalities have been observed in vitamin D deficient animals, no direct effect of 1,25(OH)2D on the process of ossification has been observed. When serum calcium and phosphate levels were maintained in the normal range in vitamin D deficient rats by dietary manipulation, the skeletal histology was found to be normal (Holtrop et ,, 1986). In patients with postmenopausal osteoporosis, 1,25(0H)2D administration has been shown to increase

Circulating osteocalcin level (Zerwekh et al., 1985). In the past, human milk was thought to be an adequate source of antirachitic activity for neonates and growing infants. Even before the discovery of vitamin D, McCollum et al (7) and Park (8) stated that rickets was attributable to the lack of sunlight and a dietary factor X. They observed that factor X was found in "good breast milk" and cod liver oil and that, although rickets did develop among breast-fed children, it was rarely as severe as that among artificially fed infants. Those investigators did not know that the source of vitamin D in the mother's milk was the mother's exposure to the sun, which cutaneously generated large amounts of vitamin D. Early attempts to quantify the antirachitic potential in human milk were crude and yielded little information. Specker et al (13) determined that the antirachitic content of human milk was lower among African American than white mothers. If a lactating mother has limited sun exposure and/or limited vitamin D intake (such as occurs with the current 400 IU DRI), then the vitamin D content of her milk is poor, especially if she has darker pigmentation.

In the present paper we are discussing the role of Vitamin D in Lactating mothers.

MATERIALS AND METHODS

Lactation presents a calcium challenge to the mother similar to that experienced in pregnancy. In some species e.g. the rat, the stress on the calcium homeostatic mechanism

is a far greater in lactation than in pregnancy. In 21 days of lactation, the rat

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transfers to her litter over 2.5 g of calcium, equal to 60% of calcium content of her skeleton. The daily loss of calcium in the milk in lactating rat usually exceeds 100 mg which is 100 times more than the daily urinary calcium excretion. In human female, the calcium secretion in milk (350mg/day) is only marginally greater than the 24 hrs urinary calcium excretion. In the lactating rat, food intake is 3-4 times that of a non-lactating rat. The increase in appetite is attributed to suckling-induced stimuli as well as to the metabolic drain of milk production. The structural changes in the jejunum include increase in villus height, crypt depth and total tissue mass (Cripps and Williams, 1975; Burdett and Ruk.,1979). The mechanism of intestinal adaptive changes appear to include both the presence of an increased amount of food in the intestine and the action of some hormones. Prolactin has been suggested as one of such hormones (Mainoya, 1978).In vitro prepration (Kostial et al . , 1969a; Kostial et al.,1979b; Toverud et al.,1976) have confirmed the increased intestinal calcium absorption in lactating rats. In the studies of Fournier and Susbielle (1952) when the diet contained 100 mg calcium per day, calcium absorption in lactating rats was 50% of the dietary intake as compared to 10% in controls. When the dietary calcium was reduced to 27mg/day, the intestinal absorption of calcium was almost 100%. Increased intestinal calcium absorption may be attributed to the increased level of plasma l,25(OH)2D in view of the well known action of the hormone on-calcium binding

protein synthesis. However, the intestinal calcium absorption remains high even in vitamin D deprived lactating rat (Toverud et al.,1978). Halloran and De Luca (1980b) studied the intestinal calcium absorption by everted gut sac technique. Duodenum sac of the lactating rat which had been deprived of vitamin D for long time (and had undetectable circulating levels of 25(OH)D and l,25(OH)2D) showed significantly higher calcium absorption than the duodenum sac of a non-lactating rat. It may be added that the duodenum sac of a vitamin D replete lactating rat showed still higher calcium absorption. From these experiments it has been concluded that while vitamin D is important for the increased active transfer of calcium in the intestine during lactation, there is also a vitamin D independent component of active transfer associated with pregnancy and lactation.

DISCUSSION

While scanning the literature on intestinal calcium absorption in lactating women it may be pertinent to note the species difference in calcium metabolism. In the rat the calcium requirement of the fetus is almost negligible as compared to the calcium requirement during lactation, while the daily fetal calcium requirement in the last two months of human pregnancy usually exceeds the amount of calcium secreted in the milk (Spray, 1950). This fact may explain why firm evidence for enhanced calcium' absorption from the intestine in lactating women is not available. Some studies have revealed enhanced calcium absorption in the later months of

pregnancy but no further increase during lactation (Heaney and Skillman, 1971). Many studies have,, although revealed the improvement in intestinal calcium absorption in a lactating woman after vitamin D supplementation (Toverud and Toverud, 1931; Liu et al.,1937) . We have been able to identify only 3 prospective studies that examined vitamin D supplementation during lactation. Greer and Marshall (Greer and Marshall ., 1989) found that exclusively breastfed white infants nursed during the winter in a northern climate maintained a "minimally normal" vitamin D status for a period of 6 mo. It should be noted, however, that the circulating 25(OH)D concentrations among the breastfeeding infants declined as the study progressed, as noted in our own study. This decline occurred despite a maternal vitamin D intake of ~700 IU/d. A Finnish study showed that maternal supplementation with 1000 IU/d vitamin D resulted in a "minimal" increase in circulating 25(OH)D concentrations among nursing infants (Ala-Houhala ., 1985). The same investigators performed a similar study with 2000 IU/d maternal supplementation and found that the nursing infants' vitamin D status improved significantly (Alq Houhala et al.,1986). The increase in maternal circulating 25(OH)D concentrations during the 4-mo study period averaged 23 ng/mL. Supplementation with high-dose vitamin D for mothers resulted in increases in circulating 25(OH)D concentrations that were completely attributable to increased 25(OH)D_2 concentrations .This increase was more pronounced among mothers who received 3600 IU/d vitamin D_2. A similar profile was observed for circulating vitamin D_2 It is of interest that, in both groups, circulating 25(OH)D_3 concentrations decreased although the mothers were receiving 400 IU/d vitamin D_3. This observation reinforces the uselessness of a 400 IU DRI for adults. It is important to note that, while the mothers received 4000 IU/d vitamin D for a period of 3 mo, maternal 25(OH)D concentrations were elevated to and remained in a normal healthy range. Again, no adverse side effect was observed.

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