Published in final edited form as:

Public Health Nutr. 2011 April ; 14(4): 748–749. doi:10.1017/S1368980011000309.

The vitamin D requirement during human lactation: the facts and IOM's 'utter' failure

Bruce W. Hollis and **Carol L. Wagner**

Division of Neonatology, Department of Pediatrics, Medical University of South Carolina, 173 Ashley Avenue, MSC 513, Charleston, SC 29425, USA

Bruce W. Hollis: hollisb@musc.edu

Madam

The new Institute of Medicine (IOM) recommendation for vitamin D intake is stated to be 10 and 10–15 μg/d for the newborn infant and lactating mother, respectively[(1)], and represents only a marginal change from its previous recommendations[(2)]. We have no issue with respect to the infant recommendations; however, the lactating woman's recommendation is another matter. Our lab has been investigating this area for more than three decades and was the first to actually quantify the vitamin D compounds in human milk[(3)]. Surprisingly, most of our data have been ignored in favour of the original recommendation – or, more appropriately, 'the estimation' – by Blumberg, Forbes and Fraser in 1963[(4)].

As a graduate student in human nutrition in the 1970s (B.W.H.), the senior investigator in our lab Dr Hollis was struck by the teaching that human milk was the 'perfect' food for the human neonate with one exception: it was inadequate with respect to vitamin D content, and rickets could result in the nursing infant if not provided with exogenous vitamin D supplementation. How could this be? What did these infants do prior to the discovery of vitamin D and how could nature have allowed this to happen? Actually, the answer is quite simple: we in medicine believed our own dogma instead of actually following the science, and thus we tried to 'fit' our 10 μg/d recommendation to the physiology instead of applying the physiology to discover the true recommendation.

First, it was said that milk had plenty of vitamin D due to the presence of vitamin D-sulfate. In fact, research 'conveniently' demonstrated that vitamin D-sulfate provided activity of about 10 μg/d in human milk[(5)]. The problem was that this research was faulty: vitamin D-sulfate did not exist in milk at all[(6)], so we were back to the drawing board. Accurate assessment had shown the vitamin D content of human milk in 'normal' lactating women to be less than 2.5 μg/l[(3,7)]. We had shown that lactating women exposed to UV light or given high oral doses of vitamin D to control hypoparathyroidism could produce milk that contained extremely high levels of antirachitic activity of up to 200 μg/l[(8,9)]. This increase in activity was almost totally due to the parent compound, vitamin D, gaining access to the milk and not the major circulating form, 25-hydroxyvitamin D (25(OH)D)[(8,9)]. But, how could this knowledge be applied to 'normal' women since it was 'well known' that intakes of vitamin D in excess of 50 μg/d would result in toxicity?[(2)] Because of this belief, this area of research lay dormant for nearly two decades; our laboratory being as guilty as anyone

else's for believing it. Fortunately, our view on this matter changed when Vieth *et al.*[10] published a seminal paper in 2001 that demonstrated oral intakes of vitamin D_2 up to 100 μg/d were safe.

Let us piece together the physiology for vitamin D metabolism in the human female. The parent compound, vitamin D_3, is mostly derived from human skin following exposure to UV light, which can result in the release of several thousand IU/d into the circulation[11]. This vitamin D_3 is 'loosely' bound to the vitamin D-binding protein (DBP) with a circulating half-life of approximately 1 d[12]. A portion of this parent compound is metabolized to 25(OH)D, which is 'tightly' bound to the DBP with a circulating half-life of approximately 3 weeks[12]. Here is where one has to pay attention to the physiology. While 25(OH)D is the major circulating form of vitamin D, it is poorly transferred into human milk while the parent vitamin D is readily transferred[8,9,13]. The problem is that because the half-life of vitamin D is so fast, it has to be replenished daily to be effective and this replenishment has to be substantially greater than the 'artificial' requirement of 10 μg/d, which does nothing to raise the circulating parent vitamin D_3 levels in the mother. In fact, one can use all this data and simply calculate that for each 25 μg intake of vitamin D by the mother daily she will deposit approximately 2.5 μg of antirachitic activity into a litre of her milk. Thus, one can supplement the lactating women with vitamin D at 150 μg/d or let her obtain significant sun exposure and she will not only replete herself but also supply her nursing infant with vitamin D in her milk at 12.5 μg/l or so. The sun exposure part does not currently fit into our culture but it was how vitamin D was obtained for untold thousands of years before we became civilized and warned that sunlight was a carcinogen to be avoided.

Clinically, this fact has been clearly demonstrated in a recent publication from our group that effectively raised the antirachitic activity of human milk to a level that sustains the nursing infant with no harm to the mother[14]. Subsequently we received a large grant from the National Institutes of Health to study this approach further, in which we give mothers 50 or 150 μg vitamin D_3/d compared with controls receiving 10 μg vitamin D_3/d (and concomitant vitamin D_3 drops of 0 IU to the infants of mothers in the high-dose groups and 10 μg/d to the infants whose mothers are receiving 10 μg/d) to sustain not only maternal circulating levels of vitamin D and 25(OH)D, but also her nursing infant's. The 5-year project is nearing completion and we have not encountered a single adverse event related to high-dose maternal vitamin D supplementation. It should be noted, however, that we had to terminate the 50 μg/d arm of the trial because through our DSMC it was determined that this dose was 'inadequate' at supplying the nursing infant with sufficient amounts of vitamin D to maintain normal infant total circulating 25(OH)D level. Why, because a 5 μg/d intake even for a neonate is not an adequate amount. Just think, only a few years ago, that 50 μg/d dose was thought to cause vitamin D toxicity. Isn't science a wonderful force if one actually pays attention and follows the data?

References

1. Institute of Medicine. Dietary Reference Intakes for Vitamin D and Calcium. Washington, DC: National Academies Press; 2011.

2. Food and Nutrition Board, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Washington, DC: National Academies Press; 1997.

3. Hollis B, Roos B, Lambert P. Vitamin D and its metabolites in human and bovine milk. J Nutr. 1981; 111:1240–1248. [PubMed: 6788913]

4. Blumberg R, Forbes G, Fraser D. The prophylactic requirement and the toxicity of vitamin D. Pediatrics. 1963; 31:512–525.

5. Lakdawala DR, Widdowson EM. Vitamin D in human milk. Lancet. 1977; 1:167–168. [PubMed: 64698]

6. Hollis B, Roos B, Drapper H, et al. Occurrence of vitamin D sulfate in human milk whey. J Nutr. 1981; 111:384–390. [PubMed: 6257870]

7. Hollis BW. Individual quantitation of vitamin D_2, vitamin D_3, 25(OH)D_2 and 25(OH)D_3 in human milk. Anal Biochem. 1983; 131:211–219. [PubMed: 6311049]

8. Greer FR, Hollis BW, Cripps DJ, et al. Effects of maternal ultraviolet B irradiation on vitamin D content of human milk. J Pediatr. 1984; 105:431–433. [PubMed: 6088746]

9. Greer FR, Hollis BW, Napoli JL. High concentrations of vitamin D_2 in human milk associated with pharmacologic doses of vitamin D_2. J Pediatr. 1984; 105:61–64. [PubMed: 6610738]

10. Vieth R, Chan PC, MacFarlane GD. Efficacy and safety of vitamin D_3 intake exceeding the lowest observed adverse effect level. Am J Clin Nutr. 2001; 73:288–294. [PubMed: 11157326]

11. Matsuoka LY, Wortsman J, Haddad JG, et al. *In vivo* threshold for cutaneous synthesis of vitamin D_3. J Lab Clin Med. 1989; 114:301–305. [PubMed: 2549141]

12. Haddad JG, Matsuoka LY, Hollis BW, et al. Human plasma transport of vitamin D after its endogenous synthesis. J Clin Invest. 1993; 91:2552–2555. [PubMed: 8390483]

13. Hollis BW, Pittard WB, Reinhardt TA. Relationships among vitamin D, 25(OH)D, and vitamin D-binding protein concentrations in the plasma and milk of human subjects. J Clin Endocrinol Metab. 1986; 62:41–44. [PubMed: 2999182]

14. Wagner C, Hulsey T, Fanning D, et al. High dose vitamin D_3 supplementation in a cohort of breastfeeding mothers and their infants: a six-month follow-up pilot study. Breastfeed Med. 2006; 2:59–70. [PubMed: 17661565]