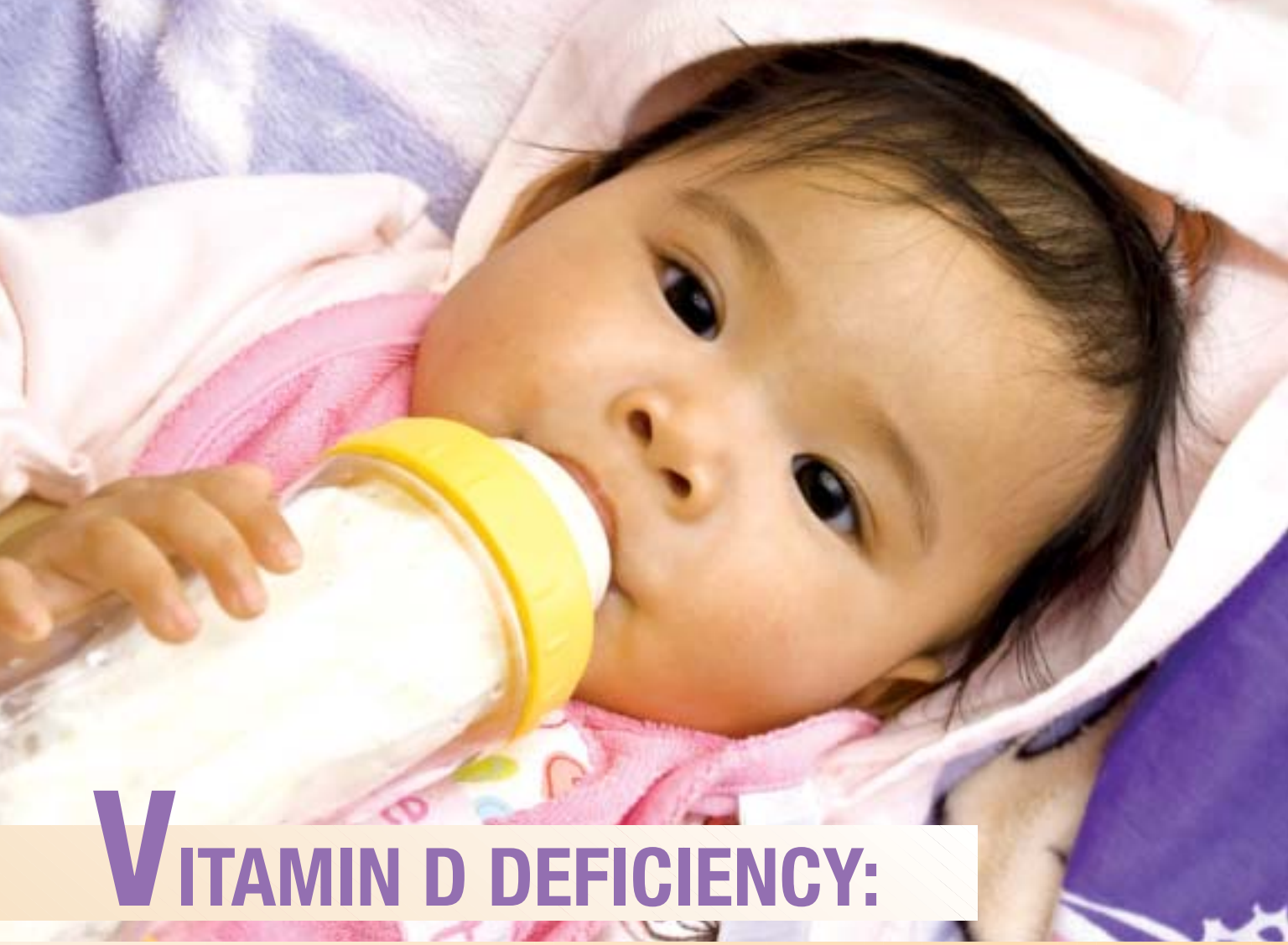

VITAMIN D DEFICIENCY:

IMPLICATIONS ACROSS THE LIFESPAN

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ABSTRACT

Vitamin D deficiency can occur in infancy, childhood, midlife, and aging. Research is emerging regarding vitamin D deficiency and its effect on neuromuscular function, cancer, cardiovascular disease, inflammatory illnesses, and bone mineralization. Recent evidence has shown that there is a need for increased vitamin D supplementation across the lifespan. Nurse practitioners must understand the effect of vitamin D deficiency on multiple body systems, assessment for deficiency, treatment, and prevention.

Keywords: Deficiency, lifespan, osteoporosis, vitamin D

The role of vitamin D and calcium absorption in the eldery with osteoporosis has been well documented. However, as more evidence is developed, it is becoming clear that vitamin D deficiency is not only affecting the elderly population, it has negative effects across the lifespan. It is estimated that 1 billion people worldwide have vitamin D deficiency or insufficiency.[1] Review of research in medical, nursing, and nutritional literature reveals the need for vitamin D education, supplementation, and follow-up in all ages and treatment settings.

VITAMIN D: AN OVERVIEW

Vitamin D is a fat-soluble vitamin that is metabolized by the body by either sun (ultraviolet light) exposure or dietary intake. There are 2 molecules that make up vitamin D: ergocalciferol (D_2) and cholecalciferol (D_3).[2] Ultraviolet B (UVB) radiation is absorbed by the skin and converts 7-dehydtocholesterol to vitamin D_3.[3] Vitamin D_3 then goes into the capillary bed, where it binds to vitamin D binding protein (DBP). Vitamin D_2 and vitamin D_3 are also ingested through the diet from fortified milk, fatty fish (salmon), and fortified cereals. Once ingested, they are incorporated into chylomicrons. The chylomicrons are transported through the lymphatic system and then are released into venous circulation, where vitamin D is bound to lipoproteins and DBP[3]. Both vitamin D from the sun and diet then enter the liver and are converted to 25-hydroxyvitamin D [25(OH)D_3] (calcidiol)[3]; 25(OH)D_3 (calcidiol) is the major circulating form of vitamin D and is used to determine vitamin D status. To become biologically active, it requires additional hydroxylation in the kidneys to form active 1,25- hydroxyvitamin D [1,25$(OH)_2$D] (calcitriol); however, 1,25$(OH)_2$D is not used to determine vitamin D status because it circulates at 1000 times less concentration than 25(OH)D_3 and it has a half life of 6 hours in comparison to 2 weeks for 25(OH)D_3.[3]

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Once reaching the form of calcitriol, vitamin D acts as a hormone. Calcitriol is important in the function of intestine, bone, and kidney that result in the maintenance of plasma calcium, phosphorus, and magnesium. It has been recently determined that there are vitamin D receptors in a variety of cells and therefore, vitamin D has a biological effect on more than mineral metabolism.[4,5]

Vitamin D has its greatest effect on the maintenance of adequate levels of serum calcium. Calcitriol works with parathyroid hormone to maintain adequate calcium and phosphorus levels in the blood. When serum calcium is too low, parathyroid hormone stimulates calcitriol to act to increase the intestinal absorption of calcium, increase the resorption of calciu m by the kidneys, and stimulate the release of calcium from the bone. In response to elevated serum calcium, calcitriol decreases intestinal absorption and stimulates bone to take up calcium, decreasing serum calcium.[5]

Deficiency

Deficiency occurs when people do not have adequate exposure to UVB rays or adequate dietary intake. According to the National Institutes of Health (NIH), 10 to 15 minutes of direct sunlight at least twice a week to the face, arms, hands, or back is sufficient to maintain optimum serum Vitamin D levels.[6] The common application of sunscreen and decreased outdoor sun exposure can result in inadequate production of vitamin D in the skin. Sunscreen application reduces vitamin D synthesis—SPF 8 by 92.5% and SPF 15 by 99%.[1,7] While there are varying opinions on whether or not sunscreen can be a primary cause of deficiency, it has been shown that sunscreen will reduce the synthesis of the vitamin.[7-9]

People with darker skin pigmentation have a reduction of synthesis by 99% because the UVB rays are being absorbed by melanin. There is a reduction of 7-dehydrocholesterol in the skin as aging occurs—only 25% of vitamin D_3 is synthesized in a 70-year-old.[10] Therefore, people with darker skin, and elders, need to ingest more vitamin D in their diets.

Because the sun does not rise high enough in the sky in the winter months, people who live in high latitudes require more dietary intake of vitamin D_3 and D_2. It has been shown that above 35° north latitude (Atlanta), little or no vitamin D_3 can be produced from November to February.[10]

The use of tanning beds has been discussed as an option for increasing UV exposure and, in turn, increasing serum vitamin D. While tanning beds can result in vitamin D photosynthesis, they also increase the incidence of skin cancer and photo aging. Due to the damaging effects of UVB radiation from artificial sources, it is not recommended that people use these devices as a main source of vitamin D.[11]