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Review

The Vitamin D connection to pediatric infections and immune function

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Abstract

Over the past 20 years, there has been a resurgence of vitamin D deficiency and relapse in nutrition rockets around the world, including in the United States. Inadequate serum vitamin D intake is also associated with complications of other health problems, including tuberculosis, cancer (prostate, breast, and colon), multiple sclerosis, and diabetes. These results support the notion of vitamin D, which has important pelotropic functions outside of calcium homeostasis and bone metabolism. In children, an association of respiratory nutrient reserves has long been recognized. Recent epidemiological studies clearly show a link between vitamin D deficiency and an increased incidence of respiratory infections. Further research has also shown the contribution of vitamin D to the host immune response to infection. However, the mechanism by which vitamin D levels play an important role in pediatric infections and immune function remains to be determined. This knowledge is particularly relevant and timely, as newborns and infants are more likely to be viral than bacterial infections due to vitamin D deficiency. The relationship between vitamin D, infection and immune function in the pediatric population indicates possible interventions and the potential role of vitamin D supplementation in appropriate treatment.

Keywords: Immune system, Pediatric infection, Vitamin D.

INTRODUCTION

After the discovery that vitamin D deficiency was the cause of malnutrition, the emphasis on the status of vitamin D in children was drawn to a debate that focused primarily on disease prevention and treatment. However, some early physicians surprisingly recognized the increasing incidence of respiratory infections in newborns and infants (White, 2008). However, it is often thought that the increasing incidence of respiratory infections in these children reflects compliance with the lungs' involvement with severe rickets and rib defects associated with poor nutrition. Epidemiological studies have now identified a link between inadequate vitamin D concentration and infectious disease (Sechert-Hellert et al., 2006). In addition, the role of vitamin D in host defense against infection has been clarified. The aim of this study is to provide an overview of the current knowledge about the role of vitamin D in immunological function and the expression of infectious diseases in the child population (Lehman, 2000). According to the definition of vitamin D deficiency, vitamin D is not a real vitamin because proper exposure to sunlight negates or significantly reduces the need for extra food. Instead, it refers to a group of misinterpreted steroid molecules that contain vitamin D2 (derived from plants that use ergosterol instead of cholesterol) and vitamin D3 molecule (derived from cholesterol) (Lehman et al., 2000). In this way, the human body obtains vitamin D in two independent ways: through the photochemical action

of solar UVB light (295 to 320 nm) in the skin and through some limited food sources. Noting that vitamin D2 is produced by plants, dietary sources (found naturally and / or obtained through oral supplementation) are the only source of it. Vitamin D3, on the other hand, is obtained primarily through the catnip reaction of sunlight as previously described or from dietary sources (Maureen et al., 1991). For adults, fatty fish and / or oral supplements provide an abundant supply of vitamin D3 (Xander et al., 2001). In contrast, major sources of vitamin D in the pediatric population provide fortified foods such as cereals, cheese, and milk, any of which are consumed in equal amounts by all age groups. Adult diets typically provide 10–20% of an individual's vitamin D stores, with a child's diet likely to provide as little vitamin D as possible. In the body, 25-hydroxy vitamin D (25D) is the main circulating vitamin D metabolite (Lin and White, 2004). It is produced primarily by the liver 25-hydroxylation by many potential catalysts, including CYP2R1 and CYP27A1. C CYP27B1 enzyme is required for hormonal 1,25-dihydroxyvitamin D (1,25D) conversion. The kidneys have long been considered an important site for 1D hydro acceleration from 25D to 1,25D (Vitha, 1999). Unlike the loose easily regulated hepatic hydroxylation of 25D, renal 1-hydroxylation comes under strict control of parathyroid hormone (PTH) and is primarily involved in calcium regulation and signaling. In sites other than the renal tubules, such as keratinocytes, the tropholastic layer of the umbilical cord, IFN-induced macrophages, and granulomatosis, this type of rapid regulation is either absent or does much work (Hulk, 2007). Compared with adults, strict control of renal 1-hydroxylation and normal repression by 1,25D is also less precise in newborns (Mesra et al., 2008). Once converted, 1,25D acts as an active form of vitamin D and binds to the vitamin D receptor (VDR), the nuclear receptor and ligand activated transcription factor. VDR is expressed in most tissues and works in cellular differentiation and many cell types (Lee et al., 2007). For example, VDR expression is found in monocytes as well as in stimulating macrophages, dendritic cells, natural killer cells, T cells, and B cells of the immune system (Ziegler et al., 2006). Activation of the VDR produces a flow gene product. In immune cells, VDR activation has elicited potent antipolytic, proliferative, and immunomodulatory effects. Initially, the research focused on the role of vitamin D in bone metabolism and calcium homeostasis (Raja et al., 2005). The regulation of intestinal calcium transport is still the most important effect of 1,25D which works while binding to its VDR (Sullivan et al., 2005). Recently, however, it has become clear that vitamin D has pleotropic effects. These include some VDR transcription-free processes and vitamin D, which play a key role in regulating the immune system (Gordon et al., 2004; Dawson et al., 2002). VDR activity via 1,25D alters cytokine secretion patterns, suppresses infective T cell activation, and stimulates regulatory T cells (Lawson and Thomas, 1999). In dry cells, it has also been shown to affect maturation, differentiation and migration (Nicolaido et al., 2006). 1.25D can increase the phagocytic activity of macrophages and increase the activity of natural killer cells (Felihan et al., 2001). Therefore, specific differences between tissue and cells in the 25D rules are highly related to the role of 25D and 1,25D as immunomodulators. The effects of vitamin D depend on the availability of the substrate. For designated, toxic, inadequate, and deficient states, the designation is defined by the serum concentration of 25D (Greenfield et al., 2001). The 25D concentration is usually 30 to 32 ng / mL (75–80 nm). Hypervitaminosis D is arbitrarily described as a 25D concentration of 100 ng / mL (250 nmol / L).However, those living or working in a sunrich environment, such as rescuers and sunbursts, do so without evidence of harmful effects beyond good-quality solar damage from ultraviolet radiation (UVR) (Leighton et al., 1999). Crossing reached 25D concentration. Symptoms of vitamin D addiction usually do not appear until the 25D concentration exceeds 150 ng / mL (375 nM). The most common adverse effect is hypercalcemia, which can lead to the formation of kidney or bladder stones and kidney failure. Vitamin D deficiency is generally defined as 25D concentration circulating 20 ng / mL (50 nM) (Ward et al., 2007). In this case, the resulting low ionized calcium (ICA) concentration stimulates the secretion of PTH, which eventually leads to an increase in 1,25D synthesis. Elevated PTH concentrations also lead to a decrease in bone minerals and osteoarthritis. In the immature bones of children, the term rectus refers to the abnormal organization of the cartilage growth plate with osteomalacia and cartilage growth instability (Huh and Gordon, 2008). By the time the 25D concentration reaches 30-40 ng / mL (75–100 nm), the PTH and the 25D concentration are intertwined, after which the PTH concentration falls sharply. With more information emphasizing important roles outside of vitamin D-calcium homeostasis and bone metabolism, the 25D concentration that extends the range between vitamin D deficiency (Zypitis et al., 2006) and loosely defined parameters is now associated with disease manifestations. In both the adult and pediatric populations, the term vitamin D deficiency is increasingly recommended for ranges that range from 20 ng / mL (50 nm) and 30 to 32 ng / mL. L (75–80 nm) to calculate these observations (Banner et al., 2008). During the winter solstice period (out of latitude lines, Tropic of K head and Tropic of Capricorn), surface solar UVB radiation is insufficient to stimulate adequate production of vitamin D3 (Zeterman et al., 2004) Climate change in the marine climate, altitude above the equator, and a decrease in sun exposure increase the risk of vitamin D deficiency (Davis et al., 1985). The content of melanin in an individual's epidermis also affects the absorption of UVB when darker lubricants cause less UVB absorption due to melanin acting as a natural sunscreen. Any

procedure that inhibits UV absorption (clothing, increased contamination, prolonged indoor use) works in a similar way to prevent skin production of vitamin D3 (Gilani 2000). Using the above definitions, estimates suggest that 1 billion people worldwide may be due to vitamin D deficiency. Recognizing the global health status of children, especially in the last 20 years, there has been a significant increase in the recognition of vitamin D deficiency (Bryce et al., 2005). In the United States, healthy infants , and adolescents are more likely to have a racket than vitamin D deficiency. Infants, children and adolescents in other countries, including the United Kingdom , Greece , Lebanon , China and Finland , have also been found to be severely deficient in vitamin D (Williams et al., 2008). In addition to rectal rehabilitation, vitamin D deficiency in children is implicated as a risk factor for the development of chronic diseases in later life, including asthma, diabetes, heart disease and cancer .In a separate UK study, it also found that the cost of preventing vitamin D deficiency in a high-risk population of Asian children was theoretically higher than the financial burden of treating common health problems associated with chronic vitamin D deficiency (Moh et al., 1997). But this view is supported. Despite the small group, the study provides widespread motivation to consider more aggressive prevention and treatment of vitamin D deficiency. Vitamin D deficiency and infectious diseases are associated with different forms of the disease. In adults, "normal" vitamin D concentration is strongly associated with tuberculosis, influenza, autoimmune diseases, cancer (prostate, colon and breast), and myocardial infarction (Najada et al., 2004).

Studies in newborns and infants are also looking at the effects of vitamin D deficiency and type 1 diabetes mellitus, as well as the risk of developing allergic and atopic diseases (Yousafzai et al., 2004). Nevertheless, there is a significant difference in our understanding of the consequences of vitamin D deficiency in the child population (Karatekin et al., 2007). An example of the link between vitamin D deficiency and infectious disease susceptibility is tuberculosis (TB). Studies published over the past 20 years have shown a decrease in serum 25D and an increase in the severity and / or susceptibility to TB infection (Green et al., 2008). Davis et al showed significantly lower performance at D3 concentration compared with controls in culture-positive TB patients. More recently, a study of the Gujarati-Indian population in London found that patients with active TB (67%) were more likely to have a D3 deficiency, compared to those from insecure households. who served as the control group (Dhawan et al., 2007). In children, infections are a major cause of illness and death worldwide (Wolverine, 2006). Recent epidemiological studies have shown an association between high concentrations of vitamin D and hospitalization and / or respiratory infections in children. Williams, etc. Determined the vitamin D status of 64 children infected with TB. Eighty-six percent of their patients had insufficient vitamin D stores. Although TB is the prototypical association of vitamin D deficiency and infectious disease, other infectious diseases have also been linked to inadequate vitamin D stores in children (Johnson et al., 2007). In recent study examined the risk for developing pneumonia among Ethiopian children with nutritional rickets (Ruth et al., 2007). This case-control study found a strong positive correlation between vitamin D deficiency and respiratory compromise (Ruth et al., 2008). More recently, Najada et al. studied a cohort of hospitalized infants with respiratory diseases and found a higher incidence of nutritional rickets. Wayse et al. also investigated acute lower respiratory tract infections (ALRIs) in nonrachitic children admitted to a private hospital in India. Their study recognized a link between subclinical vitamin D deficiency, independent breastfeeding and increased risk for severe ALRIs. Karatekan ET presented a report from Turkey on ALRIs and nonrachitic vitamin D deficiency in newborns. They found that the serum 25D concentration in newborns with ARRI was lower than in the healthy control group (Kennel et al., 2006). The risk of developing LDR is also significantly increased with a 25D concentration of 10 ng / mL (25 nm). In their study population, infants with ALRIs spent an average of 8 hours in NICU, which again had the financial and social effects of vitamin D deficiency. The incidence of infections, especially in the child population, usually peaks during the winter months when the vitamin D synthesis in the peaks is poor. In contrast to data available from adult subjects, infections with insufficient stores of vitamin D in children are most often reported to be viral. Numerous previous studies have pointed to the high concentrations of vitamin D, which may play a role in protecting against upper and lower respiratory tract infections (Peltier et al., 2006). One of the key points in these studies is that the possibility of infection arises before many nutritional rickets are clearly visible. In particular, the risk of hospitalization for infection also reflects a deficient state of vitamin D, rather than the more severe vitamin D that is commonly documented in nutritional risk cases. Secondary deficiency is revealed. Inadequate concentration of vitamin D, in most populations of the population affects children's health medically more severe and often before the onset of rickets and osteomalacia (Diurex et al., 2007). Cystic fibrosis (CF) is another disease in the pediatric population that is characterized by recurrent infections and insufficient serum levels of vitamin D . Patients with CF usually have difficulty with malabsorption and are usually placed on oral supplements of fat-soluble vitamins, including vitamin D (Camarago et al., 2007). Treatment of these infections has resulted in the growing concern of antibiotic resistance by causative pathogens. One area of research to improve the efficacy of antibiotic therapy is the use of anti-microbial peptides (AMP) as a treatment aid.

Catalysedin is an AMP with multifunctional roles in host defense, expressed through 1,25D. Recently, the production of catalysedin in primary cultures of normal and CF bronchial epithelial cells was investigated. They were able to demonstrate 1.25 rapid concentrations of catalysedin in this cell type. They also provided evidence for 1,25 D-treated bronchial epithelial cells exhibiting increased antibacterial activity against common CF airway pathogens such as Pseudomonas aeruginosa and Bortetelbronchoseptica.Based on these results, they speculate on the target use of inhaled 1,25D to increase the expression of catalysedin on the mucosal surface of the bronchial epithelium. An example of an HIV infection is where clinical and genetic evidence suggest that vitamin D may play a role in susceptibility and control of infection. To our knowledge, no study to date has found a link between the status of vitamin D and the risk of death from HIV. However, in one study, HIV-positive patients showed additional positive effects with vitamin D (Arcola et al., 2008).

T cell count Further research into the relationship between vitamin D and HIV is ongoing, including studies on the role of vitamin D signaling in HIV infection and VDR. Research on the VDR gene polymorphisms also supports the association between vitamin D and other infectious diseases. Johnson et al. Reported a significant association between genetic susceptibility to physiological immune function between respiratory syncytial virus (RSV) bronchiolitis and genetic multiple nucleotide polymorphisms (SNPs), including VDR. Is. Ruth et al. (39) An earlier report did not indicate a link between ALRI and vitamin D concentration in Canadian hospitalized children.

In several pediatric studies, an association with VDRK's FF genotype increased the risk of developing severe lower respiratory tract infections (primarily viral bronchiolitis). The FF genotype appears to encode a less active VDR and reduces the ability of immune cells to use vitamin D for its immunomodulatory effects or to produce anti-microbial activity. With an estimated cost of over 500 42,500 million per year, bronchiolitis places a significant financial burden on the healthcare system in the United States.. Relatively simple interventions such as vitamin D supplementation can reduce the incidence of the disease, which is a major benefit provided by healthcare providers and requested by parents and public health officials. Will be. Effects of Vitamin D on Immune Function Effects of Vitamin D on Immune Function Diseases characteristic of autoimmune diseases, such as asthma, type 1 diabetes mellitus, and multiple sclerosis-related features can be considered. Inadequate intake of vitamin D during pregnancy and low serum concentrations of 25D are associated with an increased risk of coronary heart disease in children (Huh and Gordon, 2008). A Boston, MA study found that an increase in vitamin D intake during pregnancy may reduce the risk of recurrent flies by following the mother's recommendations in early childhood. - In type 1 diabetes, studies of vitamin D supplementation during pregnancy and early childhood have shown potential to reduce the risk of the disease. A study of congenital malformations based on a population of 10,366 Finnish children for three decades found that those who regularly consumed 2,000 IU of vitamin D during the first year of life were more likely to have type 1 diabetes mellitus (Zeterman et al., 2004). The probability is 80% lower. Contradictory results have been reported in some other studies. A Swedish congenital harmonization study of 11,081 babies found that supplementing with 200 IU of vitamin D during pregnancy reduced maternal imbalance at 1 year of age, but the effect did not persist. When children aged 21-22 were examined. The increase with a daily dose of 400 IU during childhood was not related to islet autoimmunity . Differences between study designs may explain the results in part. As a child, vitamin D deficiency has been implicated as a risk factor for the development of multiple sclerosis. Semen used as a substitute marker of vitamin D concentration during pregnancy was associated with more than one family case of multiple sclerosis in a population-based study in several different countries . Using samples of serum stored in the U.S. Department of Defense serum repository, another group of investigators conducted a study on possible, domestic case control and found it interesting among the ethnically diverse populations represented in the repository. Find differences in white adults, those with the highest quintile of vitamin 25D measured before diagnosis have a lower risk of developing multiple sclerosis than those with more than one quintile (difficulty ratio, 0.59) (50). The inverse relationship between vitamin D and the risk of multiple sclerosis diagnosis was strongest when 25D concentration was measured in subjects before they reached the age of 20. However, these reports were not replicated in samples of African-American and Latino Sierra in the collection. The effects of vitamin D on the immune system extend beyond the expression of disease spontaneity, with numerous studies examining and summarizing the role of 1,25D on surgery (51–59) and adaptive (59–62) immune response (Raja et al., 2005). Is done in general, 1.25D not only works to promote the natural immune response of microbial pathogens but also prevents the pathogen from becoming a highly contagious infection immune response that effectively handles macrophages. Proves difficult. Although 1,25D has a direct effect on the adaptive immune system, it also affects the innate immune system's ability to direct the input immune response.. In this example, the 1,25D IL-12 is a strong suppressor of production (63) and dendritic cell (DC) difference (64). It is important to note that all in vitro studies that showed an effect on natural immunity up to 1,25D included 1,25D in cell cultures in addition to the 1,25D present in body serum levels or in excess serum, Done media. Furthermore, as mentioned, serum 1.25D levels are strongly controlled by parathyroid feedback,

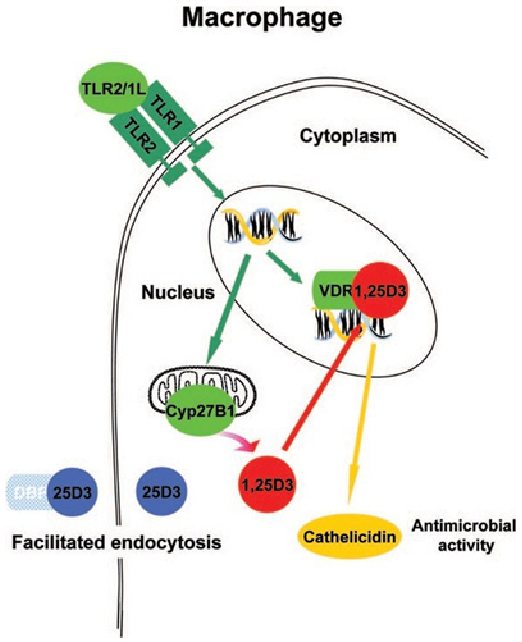

Figure 1. Effect of vitamin D on immune system.

even due to 25D deficiency. These results raised questions about whether vitamin D played a role in the body's concentration in the host's immune response. The association of 25D insufficiency with various disease states, in conjunction with in vitro studies, prompted researchers to investigate the direct effects of 25D on natural immunity. Husain et al. (58) found that at the physiological level, 25D (100 nm) achieved CD40L-induced IL-12 production in day 7-GM-CSF / IL-4 - source DCs. Similarly, little is known about the effects of changing the status of 25D in Vivo on the host's immune status. Yang At () 65) showed that a significant decrease in serum 25D in mice was the result of a significant disruption of the cell-mediated immune response to the DNFB challenge. In humans with head and neck squamous cell carcinoma, administration of 25D increased plasma IL-12 and IFN levels, and improved T cell blastogenesis (66). One possible solution to this contradictory data is that the GM-CSF / IL-4-derived DC cell surface makers do not represent the physiological process DC that can be detected in human tissue (67) (Zypitis et al., 2006). So it remains an area of new investigation. Significant studies from Rock et al., 1980s. (68) And Crowell, etc. (69) Mycobacterium tuberculosis demonstrated the ability of 1,25D to produce anti-microbial activity in infected microfibers. These studies were performed before natural changes in extracellular tissue from 25D to 1,25D, which were widely recognized and appreciated. Similarly, investigators had to be careful in properly raising concerns about their method, which they used at a level far higher than normal serum levels of 1.25 d. He questioned whether his findings were replicable in the presence of a physiological serum of 1,25D available to macrophages. Recent studies in our laboratory (70) have addressed these questions and concerns, giving new insights into the involvement of vitamin D in activating receptors as a tool for antimicrobial response to infection (Figure 1) using microarray studies. In doing so, we found that signaling by human macrophages TLR2 / 1 heterodyne pneumococcal revealed both bacterial lipopoiesis CYP27B1 and VDR. An interesting aspect of the results was that TRL 2/1 activated human macrophages in the presence of human serum, driven by flow VDR, relying entirely on serum 25D concentration. VDR driven responses from people with vitamin D deficiency were either very low or absent in serum. This reaction can be "recovery", with physically equivalent serum concentrations of 25D extra in vitro, thus providing a belief in vitamin D supplementation as a preventive and / or therapeutic aid. Vitamin D Supply As mentioned earlier, the natural sources of vitamin D are limited. Foods rich in vitamin D include fatty fish (such as squid salmon, raw Atlantic herring, pickled herring, and canned pink salmon with bone in oil), fish oil (such as cod liver), and sun-dried shatik mushrooms (9), 10) are included. None of this includes the diet of a normal teen, baby, or newborn. Strong foods such as baby formulas, cow's milk, orange juice, breakfast pulses, cheese and butter are used more, but they are significantly lower and more abundant in vitamin D. In addition, the strength of dairy products in the United States is not necessarily the same as in Canada. Food fortification is fraught with problems with compliance with anti-depressant requirements, as

well as soft regulations for reducing the consumption of vitamin-D fortified foods and beverages. Sun exposure due to skin cancer risks Many people rely on inadequate foods for vitamin D intake. In addition to the difficulty in obtaining abundant amounts of vitamin D through dietary sources, the availability of vitamin D in food and drink is often due to the methods used in preparation and cooking to meet the deficiency of D3 synthesis. Is reduced. In general, oral supplementation with multivitamins has become a widely accepted means of overcoming nutritional deficiencies and deficiencies. With oral supplements, most commercially available multivitamins contain vitamin D2. In terms of potency, plant-derived vitamin D2 is considered less potent than vitamin D3.There is a lack of uniformity or standardization of the amount of vitamin D available through oral supplements. These contradictions in the contradictory recommendations for the strength of food and the use of oral supplements are in direct conflict with the study of the use of vitamin D supplements in the treatment of diseases other than malnutrition. In 1827, the introduction of the usual oral cod liver oil administration successfully removed the expression of nutritional rockets. However, the last 20 years have seen a resurgence of vitamin D deficiency and relapse. Further studies have linked vitamin D deficiency to a number of other health problems, including asthma, cancer, diabetes, respiratory infections and multiple sclerosis. There is a long history of documentation of the use of vitamin D to treat mycobacterial infections with apparent success. In 1946 reported treating patients with lupus vulvaris (a form of catenary TB) with oral vitamin D2.Apparently, 18 of the 32 patients have recovered and nine have improved. In another study new diagnostic cases of TB were treated in children with and without standard chemotherapy without vitamin D. Total 67 patients with TB were treated with vitamin D or placebo after the sixth week of standard TB. Of the 60 patients treated in the group with vitamin D, there were more salivary changes and radiological improvement (100%) than in the placebo group (76.7%) (Franzcock et al., 2003). This difference was statistically significant (p. 0.002).The current recommendations outline a number of major issues related to vitamin D deficiency in the United States. The Consensus Committee emphasized that despite the fact that rickets are a completely contagious disease, exclusively breastfed and / or dark-skinned newborns are at greater risk of vitamin D deficiency and rickets. Furthermore, vitamin D deficiency in pregnant women puts newborns at higher risk of vitamin D deficiency because vitamin D depends on the fetus and the newborn concentration and is associated with maternal serum 25D concentration. In modern pairs, the blood concentration in the bone of 25D (reflex of the newborn) is significantly lower than the 25D concentration normally measured in the mother. Newborns born to moderately enough women are still at risk of vitamin D deficiency, and those born to women with insufficient vitamin D are almost certainly at risk (Hypponen et al., 2001). To increase anxiety for newborns, vitamin D content in human milk is also found in the mother's serum 25D concentration. Due to a number of factors affecting a person's vitamin D status, supplementing a daily dose of 400 IU does not guarantee that the mother's 25D serum concentration is sufficient for a particular breastfed infant. Will increase significantly (Brekke and Ludvigsson, 2007). In the United States, infant formulas require a minimum (40 IU / 100 kcal) and a maximum (100 IU / 100 kcal) vitamin D concentration. Based on the typical 20 kcal / oz formula, this equates to 258 IU / L to 666 IU / L. Fortunately, all American formulas contain at least 400 IU / L of vitamin D3.In the case of formula-fed infants, who are expected to consume at least one liter of formula each day, commercially available formula products will provide at least 400 IU / d of vitamin D3. However, with increasing recognition of vitamin D deficiency in women of childbearing age and an increase in exclusive and partial breastfeeding, many infants may be prevented from receiving adequate amounts of vitamin D from their diet. There seems to be an increase in rickets, and hospital admissions in children who are deficient in vitamin D (Willer et al., 2005). Vitamin D Deficiency Survivors Despite malnutrition in infants and children, adolescents also have malnutrition. As mentioned earlier, common foods lack an abundant supply of vitamin D fortified foods and beverages. Based on a number of anxiety concerns, the AAP now recommends 400 IU of vitamin D daily, starting in the first few days after birth and continuing through childhood and adolescence (Munger et al., 2006). The Consensus Committee recommends further studies for adults with additional doses with higher doses (1000 - 4000 IU) as well as some recommendations for pregnant women in Canada. The exact amount of vitamin D needed to prevent the deteriorating health associated with vitamin D deficiency is not clear. Differences in the cut-off values for vitamin D deficiency in the child population also complicate the interpretation of recommendations. Furthermore, recommendations for promoting adequate amounts of vitamin D3 synthesis to increase exposure to sunlight must balance the risks associated with increasing exposure to the sun in dealing with UV radiation (Adams et al., 1986). Based on the research currently available, however, there is far more support for completeness that prevents even more negative consequences (i.e. severe rickets).Conclusion Over the past 20 years, physicians around the world have informed us about the restoration of vitamin D-associated rickets. Even in the United States, a number of vulnerable populations have been identified, including premature infants, critically ill infants, and especially black infants (Adams et al., 1985). A large amount of historical evidence and epidemiological data support the association between adequate concentrations of vitamin D and infection. Researchers have shown how adequate

serum concentration of vitamin D facilitates the ability of immune cells to defend against bacterial and viral infections. Research in this area has provided new ways to understand the immune system and how the pyotropic actions of vitamin D play an important immunoregulatory role in proper immune function (Adams and Gacad, 1985). With growing evidence of the harmful effects of vitamin D deficiency, the whole story behind the role of vitamin D deficiency / deficiency in pediatric infections and immune function, beyond the classically determined cause of rickets, awaits a complete cut (Barnes et al., 1989).

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