

1        **A3: Call for Education and Research into Vitamin D Deficiency/Insufficiency**

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3        **Introduction**

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5        Vitamin D deficiency and insufficiency is recognized as a major public health concern  
6        for both children and adults in the United States.(Calvo, Whiting et al. 2005) It has been  
7        estimated that 30 – 40% of children and 40 – 50% of adults in the United States are at  
8        risk of vitamin D deficiency.(Holick 2006) It has also been estimated worldwide that one  
9        billion people are at risk of vitamin D deficiency.(Holick 2007) There are four  
10       populations in the USA that are at highest risk for vitamin D deficiency and they are: 1)  
11       women of childbearing age, 2) breast-fed infants not receiving vitamin D supplements, 3)  
12       persons with dark or brown skin type, including a large portion of African Americans  
13       and Mexican American adolescents and adults, and 4) elderly persons.(Looker, Dawson-  
14       Hughes et al. 2002; Weisberg, Scanlon et al. 2004; Aloia, Talwar et al. 2005; Holick,  
15       Siris et al. 2005; Moore, Murphy et al. 2005; Zadshir, Tareen et al. 2005) It is also  
16       recognized from studies in other countries that persons whose cultural or religious  
17       practices require extensive covering which can impede subcutaneous vitamin D  
18       synthesis,(Andersen, Molgaard et al. 2008) therefore these cultural groups may also be at  
19       risk, although this has not been adequately studied in North America. Factors such as low  
20       sunlight exposure, age-related decreases in vitamin D formation through the skin, and  
21       diets low in vitamin D all contribute to the high prevalence of vitamin D  
22       deficiency.(Holick 2004) Vitamin D is a fat-soluble vitamin which is provided either  
23       through the diet or by syntheses through exposure to sunlight. The vitamin has two major

1 forms: D2 (or ergocalciferol) and vitamin D3 (or cholecalciferol).(Holick 2003) The  
2 vitamin D produced from its precursor under the skin after exposure to sunlight and/or  
3 intake from vitamin D-rich or enriched foods such as wild salmon, milk and orange juice,  
4 are typically not enough to maintain adequate levels of vitamin D.(Heaney, Davies et al.  
5 2003) The interventions for this widespread public health problem are simple, safe,  
6 effective, accessible, and affordable supplemental doses of the vitamin and increased  
7 availability of vitamin D fortified food staples.(Heaney, Davies et al. 2003; Calvo and  
8 Whiting 2006)

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### 11 **Vitamin D Syntheses and Metabolism**

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13 Once vitamin D3 is made in the skin, or provided from the diet (as vitamin D2 or D3), it  
14 is converted in the liver to 25-hydroxyvitamin D [25(OH)D], which is the major  
15 circulating form of vitamin D.(Lips 2006) This 25(OH)D is the substrate for production  
16 of the active form, 1,25-dihydroxyvitamin D, via two pathways. In the endocrine  
17 pathway, 1,25-dihydroxyvitamin D is made in the kidney under tight regulatory  
18 control.(Holick 2007) In the paracrine/autocrine pathway, 1,25-dihydroxyvitamin D is  
19 made and used locally by a variety of cells in most tissues of the body, including those of  
20 the immune system.(Liu, Wu et al. 2006)

21

### 22 **Factors Affecting Vitamin D Deficiency/Insufficiency and Metabolism**

23

1 One's vitamin D status depends on several factors.(Holick 2007)The time and length of  
2 sun exposure, the season, geography, percentage of skin covered by clothing, and skin  
3 pigmentation all can affect how much vitamin D one's body will produce.(Holick 2007)  
4 Ultraviolet B (UVB) irradiation of the skin for photochemical production of Vitamin D is  
5 highest at noon time.(Holick 2007) Vitamin D levels, i.e. 25-hydroxyvitamin D  
6 [25(OH)D] the major circulating form markedly fluctuate with the change of seasons,  
7 because the angle of the sun's rays, -- which is critical to the amount of UVB radiation  
8 reaching the surface of the earth -- changes during the seasons, as well.(Holick 2007)  
9 Therefore, maximal Vitamin D production occurs in the summer months, and depending  
10 on the latitude, little or no vitamin D may be generated in winter months.(Norman 1998)  
11 In terms of geography, the farther from the equator that one migrates equates to residing  
12 at higher latitudes. The higher the latitude that which one lives—the less effective or  
13 sufficient the solar ultraviolet radiation from the sun will be for vitamin D  
14 production.(Holick 2007) Clothing can be a significant factor, as well.(Holick 2007)  
15 Vitamin D deficiency is rampant among women in Saudi Arabia, despite sunlight  
16 exposure, because the traditional clothing nearly completely covers their skin.(Holick  
17 2007; Andersen, Molgaard et al. 2008)Skin pigmentation or melanin is a dominant factor  
18 in regulating the production of Vitamin D, under conditions of low sunlight exposure.  
19 Melanin acts as a sunscreen, so a person with dark skin (type 5 or 6) requires 10-50 times  
20 the exposure to sunlight to produce the same amount of vitamin D<sub>3</sub> in their skin as does a  
21 lighter-skinned person with skin type 1 or 2.(Holick 2004) Subsequently, in the USA,  
22 African-Americans have low levels of 25(OH)D which typically is more severe the  
23 further north one resides.(Looker, Dawson-Hughes et al. 2002; Holick 2007)

1

## 2 **Functions of Vitamin D**

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4 The most well known biologic function of vitamin D is to maintain normal blood levels  
5 of calcium and phosphorus.(DeLuca 2004) The 1,25-dihydroxyvitamin D acts to promote  
6 active calcium and phosphorous absorption, and working with parathyroid hormone it  
7 helps regulate bone metabolism and kidney re-absorption of calcium. By promoting  
8 calcium absorption, vitamin D helps to form and maintain strong bones.(Heaney 2003;  
9 Holick, Siris et al. 2005; Holick 2006)Additionally, Vitamin D helps with maintaining a  
10 healthy immune system, regulation of cell growth and differentiation (thereby exhibiting  
11 antitumor activity), and stimulation of insulin production from the pancreas.(Holick  
12 2004)Evidence-based research also shows that vitamin D is increasingly recognized as  
13 having a role in the health of the cardiovascular system, neurodevelopment,  
14 immunomodulation, and regulation of cell growth.(Holick 2003; Holick 2004)

15

## 16 **Sources of Vitamin D**

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### 18 **Sun Exposure**

19 As noted above, vitamin D<sub>3</sub> is synthesized in the skin by exposure to direct sunlight  
20 (ultraviolet B radiation) and absorbed from foods. Once vitamin D is produced in the skin  
21 or absorbed from food, it requires chemical conversion in the liver to form 25-  
22 hydroxyvitamin D (25OHD), the main circulating form of vitamin D which is also  
23 referred to as the intermediate metabolite. This form of vitamin D is carried in the blood  
24 to the kidney where 1,25 dihydroxyvitamin D, the physiologically active form of vitamin

1 D is synthesized and may be released to the circulation. The intermediate form or  
2 25OHD can also be carried in the blood to other tissues that possess the ability to  
3 synthesize the active form of vitamin D which usually acts within the cells where it is  
4 synthesized in nearby tissues.(Lips 2006) Sunscreen use and dark skin pigmentation also  
5 reduce skin synthesis of vitamin D.

6

### 7 **Dietary Sources**

8 Only a few foods naturally contain vitamin D as ergocalciferol and cholecalciferol.

9 Human breast milk is typically low in vitamin D in temperate climates, containing an  
10 average of only 10% of the amount in fortified cow's milk.(Hollis and Wagner 2004)

11 Vitamin D is found in fortified foods such as milk, breakfast cereals, and juices.

12 [Response 8a] Vitamin D<sub>3</sub> is also obtained from foods like fish liver oils and cold-water  
13 fish.<sup>14</sup> In the wild, fish are part of a food chain that allows for concentration of vitamin D  
14 in the flesh of fatty fish (e.g., salmon, sardines, mackerel), while in lean fish, vitamin D is  
15 concentrated in liver (e.g., cod liver oil). Land animals that are exposed to sunlight or  
16 have vitamin D in their feed may be a source of vitamin D, but the amount of vitamin D  
17 provided in meat is not well documented except for liver.(Holden, Lemar et al. 2008)<sup>17b</sup>

18 Eggs are a natural source of vitamin D that can be increased when vitamin D is added to  
19 chicken feed, but the level is not usually significant. However, eggs processed to remove  
20 cholesterol and saturated fat in the U.S. have a restored vitamin D content of  
21 approximately 6% of the Daily Value. Presently, neither the U.S. nor Canada require the  
22 vitamin D content of foods to be listed on the required NUTRITION FACTS PANEL of  
23 food labels.(Calvo, Whiting et al. 2004) Fortification of milk and other foods with  
24 vitamin D, such as selected cereals, margarines, juices and a few selected brands of

1 cheese, provide the majority (66-84% of the food sources) the vitamin D dietary intake of  
2 Americans.(Moore, Murphy et al. 2005) Plant foods such as cultivated edible mushrooms  
3 which when briefly exposed to UVB produce significant amounts of vitamin D3(Calvo  
4 MS 2006) and some fortified foods may contain vitamin D2. The biological equivalency  
5 of the two forms of vitamin D, ergocalciferol and cholecalciferol in humans, has recently  
6 been challenged.(Houghton and Vieth 2006) The effective widespread use of vitamin D2  
7 supplements, supports the contention that ergocalciferol-based supplements or food  
8 sources are efficacious. Physiologic evidence to support the equivalency of vitamin D2  
9 and vitamin D3 was recently reported.(Houghton and Vieth 2006)

10

### 11 **Supplements**

12 Supplements provide another source of vitamin D intake. Vitamin D3 (or cholecalciferol)  
13 or vitamin D2 (or ergocalciferol) is usually (but not always) found in multivitamin  
14 preparations at 10 µg (400 IU) per tablet, with some having only 5 µg (200 IU) but new  
15 formulations are being introduced with 25 µg (1000 IU). In addition to multivitamins,  
16 single vitamin D supplements are largely available as cholecalciferol in 10 µg (400 IU),  
17 25 µg (1000 IU) and 50 µg (2000 IU) dosages. Further, some calcium supplements  
18 contain various amounts of cholecalciferol or ergocalciferol, which are in the range 10 to  
19 25 µg (400 -1000 IU) per tablet. These supplements are intended for maintenance of  
20 vitamin D status or for persons who have less than adequate vitamin D intakes. They are  
21 not intended for repletion of vitamin D deficiency. For that purpose, higher dosage forms  
22 are available through prescription.(Holick 2004)

23

1 **Symptoms and Consequences of Vitamin D Deficiency**

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3 It has long been known that vitamin D is crucial for healthy bones. The presence of active  
4 vitamin D in the small intestine aids in the absorption of dietary calcium. Individuals  
5 with vitamin D deficiency are able to absorb only a third to half as much calcium as those  
6 with sufficient levels. Calcium is vital to the mineralization of bone. The two diseases  
7 traditionally associated with severe vitamin D deficiency -- rickets in children  
8 characterized by deformation or softening of bone and osteomalacia in adults.(Heaney  
9 2003; Plotnikoff and Quigley 2003) Vitamin D deficiency will not only cause rickets in  
10 children but also prevent growing children from reaching their maximal genetically  
11 determined bone mineral density.(Holick 2006) However, vitamin D deficiency is not the  
12 only cause of rickets in children. Rickets can be an outcome of in-born errors of  
13 phosphorus metabolism such as phosphate wasting,(Heaney 2003) dietary deficiencies of  
14 calcium and/or phosphorus, vitamin C deficiency and other dietary deficiencies, all of  
15 which may result in rickets despite normal circulating levels of 25(OH)D in  
16 children.(DeLucia, Mitnick et al. 2003; Pettifor 2004)

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18 **Prevention of Rickets and Evaluation of Patients with Rickets**

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20 Although Vitamin D deficiency is a major cause of rickets in children, it is not the only  
21 cause. Rickets can result due to dietary deficiencies of calcium and/or phosphorus,  
22 vitamin C deficiency and other dietary deficiencies. Also, rarer causes like an in-born  
23 error of phosphorus metabolism leads to phosphate wasting,(Calvo 2003) which results in  
24 rickets, despite normal circulating levels of 25(OH)D in children.(DeLucia, Mitnick et al.

1 2003; Pettifor 2004) Therefore, during the initial evaluation of patients with rickets, it is  
2 important to assess for deficiencies in calcium and phosphorus, in addition to 25(OH)D  
3 levels. The differential diagnosis of rickets includes conditions that lead to hypocalcemia  
4 and/or hypophosphatemia through decreased intake, malabsorption, and/or increased  
5 excretion of calcium, phosphate, or Vitamin D.(Rauch 2007) Chronic vitamin D  
6 deficiency is strongly linked to osteoporosis and fractures.(Holick, Siris et al. 2005;  
7 Holick 2006) In adults, vitamin D deficiency precipitates and exacerbates osteoporosis  
8 and increases the risk of bone fracture.(Heaney 2003) Vitamin D deficiency in adults also  
9 causes osteomalacia, which is a painful bone disease that is often misdiagnosed as  
10 fibromyalgia or chronic fatigue syndrome, all of which may explain the underlying  
11 mechanism for the role of vitamin D in the prevention of falls.(Plotnikoff and Quigley  
12 2003; Broe, Chen et al. 2007) In addition to the adverse bone health consequences of  
13 vitamin D deficiency, it is now recognized that vitamin D deficiency may increase the  
14 risk of chronic diseases other than osteomalacia and osteoporosis.(Lind 1995; Hypponen,  
15 Laara et al. 2001; Fahrleitner, Dobnig et al. 2002; Li 2003; Chiu, Chu et al. 2004;  
16 Merlino, Curtis et al. 2004; Munger, Zhang et al. 2004; Mathieu, Gysemans et al. 2005;  
17 Garland, Garland et al. 2006; Giovannucci, Liu et al. 2006; Vieth and Kimball 2006;  
18 Freedman, Looker et al. 2007; Garland, Gorham et al. 2007; Lappe, Travers-Gustafson et  
19 al. 2007; Pittas, Lau et al. 2007) Vitamin D deficiency has also been associated with  
20 increased risk of type I(Hypponen, Laara et al. 2001) and type II diabetes,(Isaia, Giorgino  
21 et al. 2001; Chiu, Chu et al. 2004) multiple sclerosis,(Munger, Zhang et al. 2004;  
22 Munger, Levin et al. 2006) and rheumatoid arthritis.(Merlino, Curtis et al. 2004)

1 Hypertension and heart disease may also be associated with vitamin D deficiency.(Lind  
2 1995; Fahrleitner, Dobnig et al. 2002; Li 2003; Vieth and Kimball 2006)

3  
4 **Vitamin D Inadequacy Detection**  
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6 To identify vitamin D inadequacy, a simple blood test is required for detection of the  
7 level of 25(OH)D. Blood testing for vitamin D has only become available in the last 20  
8 years, and has been undergoing refinement since that time.(Heaney, Davies et al. 2003)  
9 Recent studies have shown that adequate levels of 25(OH)D are in the range of 30 – 100  
10 ng/ml, as opposed to the older standard of below 20 ng/ml defining deficiency.(Holick  
11 2007) To reflect this adjustment the newer term “insufficiency” is being used to reflect  
12 levels lower than 30 ng/ml. Specifically, insufficiency is defined as vitamin D levels that  
13 are equal to or greater than 20ng/ml but lower than 30 ng/ml.(Gonzalez, Sachdeva et al.  
14 2004; Bischoff-Ferrari, Giovannucci et al. 2006)

15

16 **Effects of Vitamin D Deficiency on Special Populations**

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18 **Women of child-bearing age**

19 Vitamin D deficiency in U.S. women of childbearing age has been described as a  
20 serious public health matter (Bodnar, Catov et al. 2007) and has been associated with low  
21 birth weight babies,(Mannion, Gray-Donald et al. 2006) and other health problems for  
22 mother and child. Vitamin D deficiency is disproportionately seen in infants of mothers  
23 with dark skin (Nesby-O'Dell, Scanlon et al. 2002) or wearing concealing clothes (i.e.  
24 veils).(Dijkstra, van Beek et al. 2007) A recent study found that 29% of Black and 5% of

1 White pregnant women residing in the northeastern United States had vitamin D  
2 deficiency, despite taking prenatal vitamins which contain approximately 400 IU of  
3 Vitamin D.(Bodnar, Simhan et al. 2007) Furthermore, in utero or early-life vitamin D  
4 deficiency has been associated with skeletal problems, type 1 diabetes, and schizophrenia.  
5 (Bodnar, Simhan et al. 2007) yet the prevalence of vitamin D deficiency in U.S. pregnant  
6 women have largely been unexplored.(Bodnar, Simhan et al. 2007) A 2004 review of  
7 studies that investigated maternal and neonatal outcomes of vitamin D deficiency or  
8 supplementation during pregnancy concluded that there wasn't evidence of a benefit of  
9 supplementation during pregnancy above amounts routinely required to prevent vitamin  
10 D deficiency.(Specker 2004) However, a recent study that looked at the disparity in  
11 Vitamin D deficiency of newborns infants of mothers at high risk of being deficient,  
12 concluded that the clinical implications are unknown and further research is necessary to  
13 determine the long-term consequences of maternal and neonatal vitamin D deficiency so  
14 that guidelines on vitamin D supplementation during pregnancy can be issued (Dijkstra,  
15 van Beek et al. 2007)

16

### 17 **Breastfed infants**

18 Infants are capable of producing all of the vitamin D they need in their skin  
19 during casual exposure to sunlight, provided the sunlight is of sufficient  
20 intensity.(Ziegler, Hollis et al. 2006) However, heavy skin pigmentation reduces the  
21 amount of vitamin D formed in the skin, which is why infants with dark skin  
22 pigmentation tend to be in less satisfactory vitamin D status than infants with light skin  
23 pigmentation and why they are at increased risk of vitamin D deficiency rickets(Ziegler,

1 Hollis et al. 2006). A lack of supplemental vitamin D or inadequate sunlight exposure in  
2 breastfed infants results in increased risk of developing vitamin D deficiency or  
3 rickets.(Gartner and Greer 2003) The recommended intake of vitamin D cannot be met  
4 with human milk as the sole source of vitamin D for the breastfeeding infant, because it  
5 contains about 25 IU/L or less of vitamin D.(Gartner and Greer 2003) In light of growing  
6 concerns about sunlight and skin cancer and the various factors that negatively affect  
7 sunlight exposure, the American Academy of Pediatric recommends that all breastfed  
8 infants be given supplemental vitamin D.(Gartner and Greer 2003)

9       Supplementation should begin within the first 2 months of life.(Gartner and Greer  
10 2003) A daily intake of 400 IU of vitamin D is believed to prevent rickets reliably and is  
11 considered free of adverse effects.(Ziegler, Hollis et al. 2006) The Institute of Medicine  
12 considers 200 IU per day to be an adequate intake for infants.(Ziegler, Hollis et al. 2006)  
13 The following countries recommend higher levels of supplementation for infants:  
14 Bulgaria recommends 20 µg (800 IU) of vitamin D per day, Romania 10 µg (400 IU) and  
15 Canada 10 µg (400) [and 20 µg (800 IU) per day in the winter].( 2001) Canada has had  
16 this recommendation for over 10 years.( 2001)

17       Although prevention of rickets is the primary reason vitamin D supplements  
18 should be provided to breastfed infants, there is literature to suggest that supplementation  
19 during infancy has other health benefits such as protection against type I diabetes mellitus  
20 and promotion of higher bone mineral mass in prepubertal girls.(Ziegler, Hollis et al.  
21 2006)

22

1 **African Americans**

2 African Americans and other persons of color are at the highest risk of vitamin D  
3 deficiency. A 2004 study revealed that 52% of African American and Hispanic  
4 adolescent boys and girls were vitamin D deficient.(Gordon, DePeter et al. 2004) Even  
5 when adults and children of color reside in sunnier locations such as Florida, Arizona and  
6 Georgia, there is a greater prevalence of vitamin D insufficiency.(Levis, Gomez et al.  
7 2005) Higher prevalence of vitamin D insufficiency and deficiency among African  
8 Americans and Mexican Americans has been observed in small regional surveys as well  
9 as the nationally representative NHANES III survey.(Looker, Dawson-Hughes et al.  
10 2002) Analyses of the NHANES III survey results revealed that 42% of African  
11 American women ages 15 – 49 years were found to be vitamin D deficient throughout the  
12 United States at the end of the winter.(Gordon, DePeter et al. 2004) When specific  
13 geographic regions are surveyed, the prevalence of vitamin D insufficiency is often  
14 greater. Another study in 2004 reported that 76% of African American women at the time  
15 they gave birth were vitamin D deficient and 81% of their infants were also deficient in  
16 vitamin D. Healthy men and women over the age of 65 in Boston were surveyed for  
17 vitamin D status which revealed that a surprising 34% of white, 42% of Hispanic and  
18 84% of African American men and women were vitamin D deficient.(Holick 2004)  
19 These statistics point to the importance of both latitude and skin pigmentation (melanin)  
20 in persons of color in hindering vitamin D synthesis. From another perspective, melanin  
21 can be a potent sun screen that provides protection from the damaging effects of sun light  
22 and decreases the amount of ultraviolet B radiation (UVB) that penetrates the  
23 skin.(Clemens, Adams et al. 1982) The ultraviolet B radiation is what the skin uses to

1 make vitamin D. Therefore, persons of color living in higher latitudes around the world  
2 are at particular risk, as are people living in Canada, Scandinavia, Russia, and all those  
3 whose lifestyles keep them from adequate access to sunlight when and where the sun is  
4 high in the sky.

5  
6 A typical African American with a skin type 6 (refers to more melanin pigmentation and  
7 lowest risk of skin cancer)(Heaney, Davies et al. 2003) has a 90% reduced capacity to  
8 produce vitamin D in their skin compared with the average "white" person.(Clemens,  
9 Adams et al. 1982) In addition, African Americans often have a lactase deficiency and  
10 avoid drinking milk which is one of the few foods that are fortified with vitamin  
11 D.(Adolfsson, Meydani et al. 2004; Harris 2006) It has also been suggested that the  
12 increased risks of diabetes, hypertension and heart disease in African Americans may  
13 also be associated with their higher prevalence of vitamin D deficiency.(Holick 2004;  
14 Holick 2004; Calvo and Whiting 2006) While vitamin D insufficiency is more prevalent  
15 among African Americans, the majority of which do not achieve optimal 25OHD  
16 concentrations at any time of year, they experience a much lower incidence of  
17 osteoporotic bone fracture than their Caucasian counterparts.(Harris 2006) A number of  
18 adaptive mechanisms through which African Americans maintain higher bone mineral  
19 density in the face of low vitamin D status have been proposed.(Harris 2006) However,  
20 most agree that skeletal resistance to parathyroid hormone action on bone and intestinal  
21 resistance to the action of 1,25 (OH)<sub>2</sub>D are key factors in the adaptation. Recent studies  
22 fail to show inverse correlations between bone mineral density and 25OHD levels in  
23 African American men compared to white men(Bohannon, Hanlon et al. 1999) or  
24 significantly higher bone turnover markers in African American men compared to white

1 and Hispanic men.(Hannan, Litman et al. 2008)Such adaptive mechanisms may not fully  
2 correct skeletal effects of low vitamin D status especially among elderly African  
3 Americans. It is important to encourage clinicians and health educators to promote  
4 improved vitamin D status among African Americans of all ages and possibly other  
5 racial/ethnic groups. Dietary intervention with vitamin D is low cost and low risk with  
6 potentially broad health effects.(Heaney, Davies et al. 2003; Harris 2006)

### 7 8 **The Elderly**

9 Older adults (age > 50 years) are at greater risk of vitamin D deficiency than younger  
10 adults for several reasons. Physiologically, there are two concerns. The enzyme  
11 responsible for synthesis of 1,25(OH)<sub>2</sub>D in the kidney (the endocrine pathway) is  
12 resistant to parathyroid hormone (PTH).(Holick 2006) This means that the 1-alpha-  
13 hydroxylase is not increased by PTH when there is need for calcium, so there is  
14 prolonged secondary hyperparathyroidism leading to increased bone loss. A low level of  
15 25(OH)D exacerbates this hyperparathyroidism. Additionally, skin cells are less able to  
16 make cholecalciferol as there are fewer molecules of 7-dehydrocholesterol (provitamin  
17 D<sub>3</sub>) in the epidermis. Skin production of vitamin D following a standard exposure to  
18 UVB light decreases with age due to decreased levels of 7-dehydrocholesterol. W when  
19 young and old subjects are exposed to the same amount of UVB, elderly subjects produce  
20 only one-third the amount of cholecalciferol.(Holick 2006)

21  
22 Upwards of 80% of the elderly is are thought to be vitamin D deficient.(Holick, Siris et  
23 al. 2005; Holick 2007) It causes muscle weakness and osteoporosis that leads to falls

1 with devastating vertebral, hip, and other bone fractures. Clinical studies have  
2 demonstrated that vitamin D with and without calcium supplementation reduces fracture  
3 risk and falls in the institutionalized elderly and in individuals over the age of 65 living at  
4 home.(Mosekilde 2005; Broe, Chen et al. 2007)

5

## 6 **Problems Associated with Excess Vitamin D**

7

8 Intoxication of Vitamin D is very rare but can be caused by ingestion of excessively high  
9 doses, whether intentional or not.(Heaney 2003) Doses of more than 50,000 IU per day  
10 raise levels of 25-hydroxyvitamin D to more than 150 ng per milliliter (374 nmol per  
11 liter) and are associated with hypercalcemia and hyperphosphatemia.(Heaney 2003)  
12 Doses of 10,000 IU of vitamin D3 per day for up to 5 months, however, do not cause any  
13 signs of toxicity.(Heaney 2003) In 2006, a major review of the literature on vitamin D  
14 and health reported that vitamin D intake above current dietary reference intakes was not  
15 reported to be associated with an increased risk of adverse events.(Cranney, Horsley et al.  
16 2007) However, this report did raise the notion that more methodologically sound  
17 research is needed regarding assessment of higher doses of Vitamin D supplementation  
18 for long-term effects. A Tolerable-upper Intake Level (UL) for vitamin D was set in  
19 1997 as 1000 IU for infants 0 to 1y and 2000 IU for all others. ULs were established to  
20 discourage potentially dangerous self-medication. The UL represents a safe intake (i.e.,  
21 zero risk of adverse effects in an otherwise healthy person), and when a patient is  
22 undergoing therapeutic treatment under a health professional's care, this amount can be  
23 exceeded. The primary adverse effect that is expected at very high levels of vitamin D is

1 hypercalcemia, which can lead, over time, to calcification of soft tissues such as arteries  
2 (arteriosclerosis) and kidney (nephrocalcinosis). A less specific indicator is  
3 hypercalciuria, which may lead to increased risk of kidney stones. The value for children  
4 and adults has been criticized as being based on a single study of dubious quality.(Vieth  
5 2006) A recent risk assessment for vitamin D used new data (post-1997) to derive a more  
6 realistic estimate of a UL.(Hathcock, Shao et al. 2007) A thorough examination of studies  
7 indicated there was absence of any signs of toxicity when healthy adults were given over  
8 250 µg (10,000 IU) daily. Thus a reasonable estimate of a UL for adults is 10,000IU.  
9 The result of vitamin D intoxication is primarily elevation of the calcium levels in the  
10 blood, which could cause stone formation in the body tissues (over time), including  
11 urinary stones. With very high calcium levels one develops a type of diabetes, or frequent  
12 urination, in which calcium causes the same effect on the urinary system as does sugar in  
13 diabetes mellitus. The hypercalcemia is corrected by stopping vitamin D  
14 supplementation.(Sigmundsdottir, Pan et al. 2007)

15

## 16 **Vitamin D Dietary Reference Intakes**

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18 Current dietary guidelines established by the National Academy of Sciences recommend  
19 daily values of 200 IU (International Units) for children and adults up to age 50, 400 IU  
20 for adults ages 51-70, and 600 IU for adults over 70y.(Medicine 1997) However, many  
21 experts believe that a daily dosage of 1000 IU or greater may be more beneficial.(Holick  
22 2006) The growing awareness of the need for greater consumption of vitamin D beyond  
23 those recommended by the National Academy of Science with the 1997 Dietary

1 Reference Intakes,(Medicine 1997) particularly by African Americans and the elderly,  
2 was conveyed to the public as a key recommendation in the 2005 Dietary Guidelines for  
3 Americans.(Medicine 1997) The 2005 recommendation advised older adults, people with  
4 dark skin and people exposed to insufficient ultraviolet band radiation (i.e. sunlight) to  
5 consume extra vitamin D from fortified foods and or supplements, and stated a daily goal  
6 of 1000 IU vitamin D intake for those at risk. A 2000 study examined the estimated  
7 amount of oral vitamin D intake necessary to maintain adequate vitamin D status in  
8 sunlight exposure in sunlight deprived individuals living in Denmark, and suggested that  
9 the daily oral intake of vitamin D should probably be 1000 IU per day.(Glerup,  
10 Mikkelsen et al. 2000 The National Academy of Sciences has set 2,000 IU daily as the  
11 "tolerable upper limit" for vitamin D.{Medicine, 1997 #110)

12

13 **Why are current efforts to address Vitamin D deficiency not sufficient?**

14

15 In addition to sun exposure, the body gets vitamin D in two other ways – from foods and  
16 from supplements. The reality is that very few foods are rich in vitamin D except fatty  
17 fish such as salmon and mackerel, and fortified foods such as milk and some brands of  
18 orange juice.(Calvo, Whiting et al. 2004) The few food staples such as milk which are  
19 fortified are not uniformly consumed by all racial/ethnic groups.(Calvo, Whiting et al.  
20 2004) Also, studies show that the elderly may particularly benefit from vitamin D  
21 supplementation to maintain strong bones and to prevent falls.(Bischoff-Ferrari,  
22 Giovannucci et al. 2006) However, those populations at greatest risk of vitamin D  
23 insufficiency, African American adults and children, have significantly lower use of

1 supplements in the U.S and may require higher doses. Daily supplementation of African  
2 American children with 400 IU of vitamin D was inadequate to raise circulating levels of  
3 25OHD to a level of sufficiency.(Bischoff-Ferrari, Giovannucci et al. 2006) A 3-y  
4 randomized, double-blind, placebo-controlled study found that daily vitamin D  
5 supplementation of 800 IU for the first 2 years and 2000 IU for the third and final year in  
6 postmenopausal African American women failed to increase serum 25OHD levels to the  
7 optimal range in 40% of the study subjects.(Aloia, Talwar et al. 2005) In both of these  
8 recent studies the dose of vitamin D was well above the National Academy of Sciences  
9 recommendations of 200 and 600 IU of vitamin D, respectively.

#### 10 **Supplementation as an intervention**

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12 Besides adequate exposure to sunlight, and eating food rich in Vitamin D, dietary  
13 supplements provide another source of intake as noted above under Dietary Supplements  
14 for populations at highest risk for vitamin D deficiency and insufficiency. For neonates,  
15 there is liquid Vitamin D, and for children and adults there is a tablet form that is easily  
16 tolerated with no significant side effects. In 2006, it was noted that the cost of 1000 IU of  
17 vitamin D3 is less than five cents, balanced against the huge human and economic costs  
18 of treating cancer attributable to vitamin D insufficiency.(Garland, Garland et al. 2006)  
19 Many other examples can be cited to underscore the benefits of adequate levels of  
20 vitamin D.(Garland, Garland et al. 2006)

21

#### 22 **Previous APHA Statements**

23

1 Previously, APHA had a statement that briefly mentioned vitamin D in relation to infant  
2 feeding (1980, policy n. 8022) and ensuring urging mothers and caretakers of infants to  
3 provide vitamin D and fluoride as the only vitamin-mineral supplements. Yet, too few  
4 health care practitioners, public health professionals and the public-at-large have  
5 adequate information/knowledge about the benefits of vitamin D in protecting against  
6 many chronic diseases and its promise in decreasing health disparities.

7

### 8 **Action Statements**

9 Therefore the American Public Health Association:

- 10 1. Urges the Centers of Disease Control and Prevention to advocate and provide  
11 funding for a coordinated and integrated approach to **educating health care**  
12 **providers/practitioners** about the science and benefits of adequate levels of  
13 vitamin D.  
14
- 15 2. Urges the United States Department of Agriculture (USDA) and other Federal  
16 agencies to promote the 2005 *Dietary Guidelines for Americans* as the scientific  
17 basis for **increased public awareness** using current nutrition guidance for healthy  
18 eating, notably for populations at highest risk for vitamin D  
19 deficiency/insufficiency.  
20
- 21 3. Recommends to the Department of Health and Human Services and USDA that  
22 the APHA have organizational representation on the panel of the *2010 Dietary*  
23 *Guidelines for Americans* for strategies to improve vitamin D intake.

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4. Requests that it become a participating organization in planning Healthy People 2020 to **promote national awareness** of the magnitude of the problem of poor vitamin D status; and the associated increased risk for chronic disease development by introducing specific objectives in planning Healthy People 2020 goals.
  
5. Urges Congress to appropriate funds to conduct **research** in diverse populations to determine population specific vitamin D intakes needed to produce and maintain optimal vitamin D status associated with reduced risk of chronic diseases.
  
6. Recommends to the Food and Drug Administration to add vitamin D to the list of required nutrients appearing on the **Nutrition Facts Panel** that is required on all foods in the U.S. market place.

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