Vitamin D Deficiency and Type 2 Diabetes in African Americans: The Common Denominators

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Abstract

The prevalence of vitamin D deficiency and type 2 diabetes is disproportionately elevated in African Americans compared to other ethnic groups in the United States. Despite recent advances in diabetes treatment and management, the most significant escalation in incidence of type 2 diabetes has been in this group. Some studies suggest a possible role for vitamin D deficiency in the development of type 2 diabetes and that insulin secretion and insulin sensitivity are reduced when vitamin D levels are deficient.

Obesity, hyperglycemia, cardiovascular disease, and minority race are common among people with type 2 diabetes and vitamin D deficiency. These phenomena are known precursors to the development of type 2 diabetes and exacerbate the risk for complications where diabetes exists. Poverty, urban living settings, and lactose intolerance are also common among African Americans. These conditions promote opportunities for vitamin D deficiency to manifest and attenuate opportunities for participation in health-promoting behaviors by those affected. The common traits between vitamin D deficiency and type 2 diabetes merit careful attention.

In the past decade, a compelling body of evidence has emerged identifying the role of vitamin D in metabolic function extending beyond calcium regulation,[1] bone mineralization, and cancer prevention[2] to include glucose metabolism and a relationship with obesity. Vitamin D deficiency in African Americans is widespread,[3] and deficiencies in vitamin D are indeed associated with obesity, impaired glucose metabolism, insulin resistance, and defective insulin secretion.[4] African-American ethnicity is a risk factor for type 2 diabetes development, and evidence suggests vitamin D may also be a risk factor for its development.[4,5]

African Americans are 77% more likely to incur a diagnosis of diabetes than non-Hispanic white Americans. New estimates from the Centers from Disease Control and Prevention indicate that 18% of African Americans have diabetes compared to 10.2% of non-Hispanic whites.[5]

The reasons behind the higher prevalence rates of type 2 diabetes in African Americans are multidimensional. African Americans experience poverty,[6] lactose intolerance,[7] and obesity[8] more often than other ethnic groups, and each of those may influence access to sources of vitamin D. Therefore, improving the understanding of comorbid conditions associated with vitamin D deficiency is an important health care priority.

To gain a better understanding of the health consequences that deficiency in vitamin D may trigger, common risk factors of vitamin D deficiency and type 2 diabetes in African Americans merit thorough consideration. The purpose of this article is to increase awareness of the health consequences associated with vitamin D deficiency among diabetes educators and health care providers that reach people with diabetes. This article will highlight the shared attributes of vitamin D deficiency, type 2 diabetes, and obesity in this group. It

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will also define vitamin D deficiency and discuss normal recommendations for intake. Relationships between vitamin D deficiency, insulin resistance, and impaired insulin secretion will be discussed, as well as the role of diabetes educators in strengthening advocacy programs to prevent vitamin D deficiency.

Vitamin D: Background

Vitamin D is a fat-soluble nutrient that is obtained from dietary sources (some fortified) as D_2 (ergosterol) or D_3 (cholecalciferol).[9] Cholecalciferol (D_3) may also be produced in the skin when exposed to sufficient ultraviolet rays.[10] Once obtained, vitamin D is hydroxylated in the liver to form 25(OH) vitamin D and used by the kidney to construct its biologically active form, 1-25(OH)$_2$ vitamin D (calcitriol). The 25(OH) vitamin D_3 has a half-life of several weeks, and hence, measurement of this level is appreciated as the gold standard for determining sufficiency in circulating vitamin D.[9]

Cholecalciferol (D_3) is largely found in fish, eggs, and meats, as well as in milk and foods that have been fortified. Ergosterol (D_2) is a plant-derived source of vitamin D.[11] Presently, only ergosterol (D_2) is regulated by the Food and Drug Administration and available by prescription. Most over-the-counter vitamin D supplement preparations are vitamin D_3 (cholecalciferol).

The risk for deficiency in vitamin D is substantial among African Americans and other ethnic groups with dark complexions. The elderly, people with sedentary lifestyles, and those who regularly wear clothes that cover most of the body also have an elevated risk for vitamin D deficiency.[12] Sunlight stimulates vitamin D_3 (cholecalciferol) production in the skin; hence, limited sun exposure from seasonal changes, sunscreen, pollution, or other causes reduces its production. Melanin acts as a natural sunscreen and limits vitamin D production; thus, vitamin D deficiencies in people with darker skin is common.[10] Intake of vitamin D is also affected by the common occurrence of lactose intolerance in African Americans.[13] Jarvis and Miller[14] indicated that African Americans have lower intake of dairy food nutrients and that eating at least three foods in the dairy group daily may go far to minimize chronic disease risks. Milk is a vitamin D–fortified source of nutrition, and lactose intolerance can contribute to lower intake of vitamin D in people affected by it. Lactose intolerance is an exacerbating factor for vitamin D deficiency, but in sum, an aggregate of risks factors is most responsible for hypovitaminosis D collectively in African Americans.

Vitamin D in Glucose Metabolism

Impaired insulin secretion and insulin resistance are the hallmark defects that define type 2 diabetes.[15] Vitamin D deficiency is related to impairments in insulin secretion and impaired insulin synthesis. Calcium regulates insulin synthesis within pancreatic β-cells, as well as insulin secretion; and plasma calcium levels are mediated by vitamin D.[16,17] Because of this mediation, some studies suggest that the deficiency in vitamin D is causal and that it precipitates impairments in insulin secretion among people with type 2 diabetes.[4,16,17]

This relationship of vitamin D with glucose metabolism was identified nearly three decades ago in a study by Norman et al.[18] It compared the insulin and glucagon secretion of vitamin D–deficient and vitamin D–replenished rat specimens in vitro and determined that insulin secretion was 48% lower among the vitamin D–deficient rat specimens. These findings provided the basis for numerous small intervention studies examining the impact of vitamin D supplementation on glycemia. None to date have been sufficiently powered to trigger recommendations for supplementing vitamin D to improve glycemic control.[19]

Insulin resistance is inversely related to serum 25(OH) vitamin D and positively related to obesity.[4] Obesity often precedes type 2 diabetes and influences the bioavailability of vitamin D_3 (cholecalciferol). These relationships are discussed later.

Recommended Intake of Vitamin D

Some of the proponents for vitamin D supplementation assert that adding 700–800 IU daily of a vitamin D supplement is a cost-effective approach to lowering risks for adverse health occurrences.[1] At present, there is no consensus on an optimal level for 25(OH) vitamin D. Recommendations have differed depending on the source of the endorsement and the clinical endpoints proposed.

There is consensus, however, in the parameters that define serum vitamin D deficiency. Scientists attending the 14th Workshop on Vitamin D[20] endorsed that 25(OH) vitamin D levels < 20 ng/ml represent deficiency and result in negative health consequences regardless of geographical location, age, or skin pigment. This scientific congress hosted more than 400 opinion leaders on vitamin D and metabolism from 35 nations. Harvard medical professor and leading authority on vitamin D Michael Holick, MD, PhD, also discussed negative health consequences associated with vitamin D levels < 20 ng/ml and defined 25(OH) vitamin D levels of 21–29 ng/ml as insufficient.[9]

An important clinical outcome from this scientific congress was a consensus statement acknowledging potential benefits to targeting higher vitamin D levels in preventing chronic disease. This is noteworthy considering the prevalence rates of chronic disease and diabetes in African Americans, as well as in the general population. Research is ongoing to determine whether minimal standards for vitamin D intake should be increased to optimize general health and prevent disease.

Grant and Peiris[21] examined disparity and all-cause mortality by evaluating 25(OH) vitamin D levels and chronic disease prevalence in African Americans and Caucasian Americans. This comprehensive literature review noted that the average 25(OH) vitamin D levels of these groups were 16 and 26 ng/ml, respectively, and that there is a significant body of observational data that directly link diabetes and other chronic diseases to low vitamin D levels.[21–23]

African Americans as an ethnic group are more obese, and exces-

sive fat can sequester vitamin D and other fat-soluble vitamins. Wortsman et al.[24] compared vitamin D levels in healthy and obese people who were provided equal doses of a vitamin D supplement and exposure to ultraviolet B irradiation phototherapy. Vitamin D levels in the obese group were 57% lower than in the healthy group despite greater skin surface area in the obese group and diet limitations at baseline in both groups before the intervention.[24] Consequently, obesity may influence the bioavailability of vitamin D. These findings, however, were limited by small sample size and a lack of ethnic diversity among study participants.

In a study by Ashraf et al.,[25] 78% of the obese African-American female adolescents were vitamin D deficient, and participants with vitamin D levels < 16 ng/ml had greater insulin resistance. Insulin resistance is a known and significant clinical feature that often results in abnormal glucose metabolism and diabetes. In general, however, African Americans are underrepresented in clinical research, and the outcomes of the aforementioned studies suggest a need for more attention toward vitamin D deficiency in African Americans.

In light of the common features of vitamin D deficiency, type 2 diabetes, and other chronic diseases, vitamin D assessment is now performed more routinely in clinical settings and often under the care of general practitioners. The recommendation to replace or supplement vitamin D has been suggested as an economical approach to consider for lowering chronic disease rates in this group.[21]

In contrast, a 2010 report from the Institute of Medicine (IOM) advanced the position that most North Americans actually receive adequate amounts of calcium and vitamin D and that health benefits purported to be associated with higher levels are not well supported by sufficient evidence on which to base sound recommendations. The exception to this position would be bone health.[19] In this report, the IOM reported the updated standards for Dietary Reference Intake (DRI) values for these nutrients by age-group and cautioned that calcium and vitamin D consumed in amounts exceeding the upper level intake standards can become unsafe. Further, the IOM reported that evidence to support benefits from nutritional supplementation beyond the current DRI values is sparse.[19] Because these options were broadly leveraged on available random clinical trials, they have been severely criticized by the vitamin D research community that advocates inclusion of outcomes from cohort, case-control, and ecological studies as future opinions are generated.[26–28]

The IOM report recommended that the nutritional standards contained therein are best used to assess the adequacy of calcium and vitamin D intake in healthy individuals. The adequate intake level of vitamin D for individuals aged 1–70 years is 600 IU daily and 800 IU daily for people > 70 years of age. Safe upper limits of vitamin D intake are 1,000–1,500 IU daily for infants and 4,000 IU daily for people ≥ 9 years of age.[29] Daily intake exceeding these recommendations increases the risks for toxicity.

Supra-physiological levels of vitamin D are difficult to attain by way of food consumption. This is the case because only a limited amount of vitamin D is present in foods naturally, and production of supra-normal amounts of vitamin D from sun exposure alone is not physically possible.[30] Therefore, toxic levels of vitamin D can be achieved only through over-supplementation.

Vitamin D toxicity may result in nausea, vomiting, poor appetite, constipation, elevated serum calcium levels, and muscle weakness.[31] Experts agree that current research has provided insufficient evidence on which to base recommendations for higher vitamin supplementation, despite compelling observational data correlating lower vitamin D levels with chronic diseases such as diabetes, obesity, and cardiovascular disease (CVD).[19,20]

Diabetes and Vitamin D in African Americans

Diabetes in the United States has increased tremendously in the past decade despite new discoveries about its origins, treatment, and management. More than 90% of people with diabetes have type 2 diabetes, the type that is defined by insulin resistance and impaired insulin secretion.[15] National estimates indicate that nearly 26 million Americans are affected by diabetes, and 18.7% of this total are African Americans.[5] African Americans are more than three times as likely to incur a diagnosis of diabetes as non-Hispanic whites.[5]

The risk factors for type 2 diabetes parallel the aggregate of conditions and behaviors that influence prevalence rates for vitamin D deficiency. An abundance of literature exists indicating that the risk for type 2 diabetes increases with African-American or minority race, obesity, hypertension, and insulin resistance.[5,15] The risk for developing diabetes is amplified by sedentary lifestyle, intake of calorie-dense and nutrient-deficient foods, and ethnic predisposition.[5]

Vitamin D deficiency is associated with diabetes, obesity, cancer, hypertension, and African-American race.[4] Associations of chronic disease with low vitamin D have spurred investigation into its role. Reis et al.[17] discussed the role of vitamin D in protein synthesis and production of insulin by pancreatic β cells. This research found that when vitamin D receptor expression was reduced, conditions of obesity, impaired glucose homeostasis, and vascular disease were more prevalent.

Impairments in insulin-secreting capacity and insulin resistance have long been identified as core defects in the development of type 2 diabetes.[15] Pittas et al.[4] emphasized the influence of low vitamin D in the development of both insulin resistance and insulin secretion impairment. Vitamin D may enhance insulin sensitivity by lowering free fatty acids. Elevated levels of free fatty acids are associated with insulin resistance, and some studies have demonstrated bet-

ter insulin sensitivity with vitamin D supplementation.[32]

Vitamin D supplementation has been considered as an inexpensive means of indirectly affecting diabetes risk among those with deficiencies, although randomized, controlled clinical studies to support such recommendations are lacking. Researchers in the Women's Health Study administered vitamin D supplements and found that supplementation failed to influence the overall diabetes risk among the middle-aged and older women studied after controlling for body weight, food, and fiber intake.[19,33] Additionally, results from a 6-month experiment by Jorde and Figenscha[34] indicated that administration of 40,000 IU of vitamin D_3 weekly to people with diabetes also did not result in better glycemic control. These outcomes are limited by small sample sizes and a non-deficient vitamin D status of the participants at baseline. Interestingly, a secondary data analysis of 445 older adults provided with calcium and vitamin D supplements by Pittas et al.[4] showed benefit only among the participants with vitamin D deficiency. Given this, it is unreasonable to ignore the escalating number of observational studies identifying associations between vitamin D deficiency, glycemic control, and chronic disease.

Harris[35] identified higher rates of CVD and type 2 diabetes among African Americans in some of these studies and discussed the potential utility of vitamin D supplementation in reducing race-based health disparities should deficiencies in vitamin D be deemed causal for these conditions. More clinical studies are needed to support and validate recommendations to supplement vitamin D for the purpose of diabetes and chronic disease risk prevention, favoring the sentiments of the IOM.[19] Further research to examine the clinical and economic benefits of vitamin D supplementation on chronic disease should include well-designed and appropriately powered studies that demonstrate benefit from supplementation interventions.

Obesity in African Americans

More than 60% of African Americans are considered overweight or obese, and the disparate prevalence of diabetes in this group is more evident among African-American women.[5] Four out of five African-American women are overweight or obese,[8] and obesity is one of many modifiable risk factors that may precede type 2 diabetes.

Obesity and serum 25(OH) vitamin D_3 are inversely related.[4] Arunabh[36] examined 25(OH) vitamin D levels in healthy women with BMI ranges from 17 to 30 kg/m^2 and also found that 25(OH) vitamin D levels were negatively correlated with BMI after adjusting for influences including ethnicity, age, diet composition, and season. Blum et al.[37] discussed body fat as a storage depot for vitamin D, which may explain lower 25(OH) vitamin D_3 levels in obese people.

The inverse relationship between 25(OH) vitamin D and parathyroid hormone (PTH) levels is well established, and PTH in part regulates production of 1-25$(OH)_2$ vitamin D. It has been reported that **elevated** 1-25$(OH)_2$ **vitamin** D stimulates lipogenesis and triglyceride accumulation and inhibits lipolysis.[24,36] However, the outcomes of a study by Parikh et al.[38] refute these findings and note that the studies substantiating the relationship of obesity and 1-25$(OH)_2$ vitamin D were limited by small subject samples and lacked ethnic diversity. Parikh et al.[38] studied these relationships in a cross-sectional, observational study and found that 1-25$(OH)_2$ vitamin D levels are elevated in people with a BMI > 30 kg/m^2, but these levels fall as obesity increases, making it unlikely that elevated 1-25(OH) vitamin D levels are lipogenic. Despite these associations, vitamin D deficiency has not been clearly identified as the cause or the outcome of obesity.

The principles underlying obesity's pervasiveness in African-American communities are complex and significantly influenced by many biological and psychosocial variables. People with obesity may be less inclined to participate in outdoor activity, may consume low-nutrient calorie-dense diets, and may be generally less physically active, all of which are contributing factors to weight gain. These behaviors reduce opportunities for vitamin D intake and synthesis and are possibly compounded by diminished access and potential intolerance to food sources rich in vitamin D. Opportunities to attain sun exposure may also be limited by the physical restrictions of extreme obesity.

Large segments of the African-American population are also concentrated in urban areas, where access to the resources needed to engage in healthy lifestyle practices may be limited and safety concerns may minimize outdoor activity and sun exposure. Zenk et al.[39] studied factors influencing the relationship between produce consumption and income among urban African-American women in a neighborhood without ready access to supermarkets and determined that produce consumption was greater where the selection and quality of available produce was perceived as positive.

Fruits and vegetables are lower in calories and are rich in fiber, vitamins, and minerals. Risk factors for diabetes, heart disease, and obesity are lower when the diet contains sufficient quantities of these nutrients.[40] African Americans are disproportionately affected by obesity and vitamin D deficiency and are collectively more socioeconomically disadvantaged.[6] Gaining information from within African-American communities regarding cultural norms, food preparation, and time orientation for use in constructing culturally sound nutritional plans and activity programs should be afforded a higher priority to optimize program receptivity and program potential.

Although milk is fortified with vitamin D, African Americans have lower rates of milk consumption because of a higher general prevalence of lactose intolerance.[7] Milk is also rich in calcium. Diets that are higher in calcium are linked to lower body weight.[29] Adequate concentrations of serum calcium suppress PTH, a sequence that is dependent on the presence of 25(OH) vitamin D and interaction with 1-25$(OH)_2$

vitamin D.[41] As noted previously, 1-25$(OH)_2$ vitamin D is positively related to obesity according to Wortsman et al.[24] and Arunabh et al.[36] Saleh et al.[42] propose that hyperparathyroidism is a consequence of vitamin D deficiency, and these trends are common among obese people.

Lactose intolerance in African Americans further increases the likelihood for vitamin D deficiency. In the Coronary Artery Risk Development in Young Adults study, Pereira et al.[43] noted that insulin resistance was inversely associated with dairy product consumption and suggested that vitamin D deficiency may be a mediating factor. Interventions designed to mitigate nutrient deficiencies in African-American communities, particularly among youth, could prove to be important in reducing future diabetes and chronic disease prevalence in this population.

Implications for Risk Reductions

There are opportunities to increase awareness of vitamin D deficiency and encourage greater health-promoting behaviors in African-American communities. The associations noted among diabetes, obesity, and low vitamin D are helpful in motivating communities to adopt straightforward lifestyle changes that can reduce the risks for such occurrences. Health-promoting behaviors that include daily exercise and the consumption of foods rich in fiber and whole grains have been indicated to reduce the risk for diabetes and improve glycemic control.[44] Patient educators, nurses, and certified diabetes educators can design, teach, and facilitate community-sustaining interventions that directly target risk factors for vitamin and nutrient deficiencies, which in turn target risk factors for obesity and diabetes. Interventions at this level do much to convey a sense of interest among care recipients and can catalyze an atmosphere of positive change toward nutrition as well as improve attitudes towards activity in communities where such needs are profound.

The continued vigilance of patient educators is necessary to identify and implement strategies likely to result in more opportunities for obesity prevention and improved vitamin D intake from nutritious foods and sunlight. Health providers should also enhance screening for vitamin D deficiency, obesity, lactose intolerance, and diabetes to proactively address conditions augmenting the chronic disease risk in African Americans. Early intervention and research designed to combat these risks carry great potential for reducing the cost burden of obesity, diabetes, and possibly other chronic diseases that are disproportionately experienced by African Americans.

Interventions to address vitamin D deficiency should play a key role in future program development. The present climate for efficient resource utilization in health care necessitates cost-effective interventions for diabetes and chronic disease with great potential to affect long-term costs for disease management by way of prevention. Diabetes educators are well-positioned as nurses, physicians, pharmacists, dietitians, and other health professionals to design and implement strategies that promote healthy behavioral changes in the people and communities most affected.

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References

[1]Bischoff-Ferrari HA, Willett WC, Wong JB, Giovannucci E, Dietrich T, Dawson-Hughes B: Fracture prevention with vitamin D supplementation: a meta-analysis of randomized controlled trials. *JAMA* 293:2257–2264, 2004

[2]Bertone-Johnson ER, Chen WY, Holick MF, Hollis BW, Colditz GA, Willett WC, Hankinson SE: Plasma 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D and risk of breast cancer. *Cancer Epidemiol Biomarkers Prev* 14:1991–1997, 2005

[3]Yanoff LB, Parikh SJ, Spitalnik A, Denkinger B, Sebring NG, Slaughter P, McHugh T, Remaley AT, Yanovski JA: The prevalence of hypovitaminosis D and secondary hyperparathyroidism in obese Black Americans. *Clin Endocrinol* 64:523–529, 2006

[4]Pittas AG, Lau J, Hu FB, Dawson-Hughes B: The role of vitamin D and calcium in type 2 diabetes: a systematic review and meta-analysis. *J Clin Endocrinol Metab* 92:2017– 2029, 2007

[5]Centers for Disease Control and Prevention: National diabetes fact sheet, 2011 [article online]. Available from http://www.cdc.gov/diabetes/pubs/pdf/ndfs. Accessed 16 May 2010

[6]U.S. Census Bureau: Income, poverty, and health insurance coverage in the United States, 2009 [article online]. Available from http://www.census.gov/prod/2010pubs/p60-238.pdf. Accessed 20 November 2010

[7]National Institute of Diabetes and Digestive and Kidney Diseases: Lactose intolerance, 2010 [article online]. Available from http://digestive.niddk.nih.gov/ddiseases/pubs/lactoseintolerance. Accessed 20 November 2010

[8]Office of Minority Health: Obesity and African Americans [article online]. Available from http://minorityhealth.hhs.gov/templates/content.aspx?ID=6456. Accessed 20 November 2010

[9]Holick MF: Vitamin D status: measurement, interpretation, and clinical application. *Ann Epidemiol* 19:73–78, 2009

[10]Rajakumar K, Greenspan SL, Thomas SB, Holick MF: SOLAR ultraviolet radiation and vitamin D: a historical perspective. *Am J Public Health* 97:1746–1754, 2007

[11]Holick MF: Vitamin D deficiency. *N Engl J Med* 357:266–281, 2007

[12]Chen TC, Chimeh F, Lu Z, Mathieu J, Person KS, Zhang A, Kohn N, Martinello S, Berkowitz R, Holick MF: Factors that influence the cutaneous synthesis and dietary sources of vitamin D. *Arch Biochem Biophys* 460:213–217, 2007

[13]Simoons FJ: The geographic hypothesis and lactose malabsorption: a weighing of the evidence. *Am J Digest Dis* 23:963–980, 1978

[14]Jarvis JK, Miller GD: Overcoming the barrier of lactose intolerance to reduce health disparities. *J Natl Med Assoc* 94:55–66, 2002

[15]DeFronzo RA, Tripathy D: Skeletal muscle insulin resistance is the primary defect in type 2 diabetes. *Diabetes Care* 32:157–163, 2009

[16]Palomer X, González-Clemente JM, Blanco-Vaca F, Mauricio D: Role of vitamin D in the pathogenesis of type 2 diabetes mellitus. *Diabetes Obes Metab* 10:185–197, 2008

[17]Reis AF, Hauache OM, Velho G: Vitamin D endocrine system and the genetic suscep-

tibility to diabetes, obesity and vascular disease: a review of evidence. *Diabetes Metab* 31:318–325, 2005

[18]Norman A, Frankel J, Heldt A, Grodsky G: Vitamin D deficiency inhibits pancreatic secretion of insulin. *Science* 209:823–825, 1980

[19]Institute of Medicine: *Dietary Reference Intakes for Calcium and Vitamin D*. Washington, D.C., National Academies Press, 2010

[20]Henry HL, Bouillon R, Norman AW, Gallagher JC, Lips P, Heaney RP, Vieth R, Pettifor JM, Dawson-Hughes B, Lamberg-Allardt CJ: 14th Vitamin D workshop consensus on vitamin D nutritional guidelines. *J Steroid Biochem Mol Biol* 121:4–6, 2010

[21]Grant WB, Peiris AN: Possible role of serum 25-hydroxyvitamin D in black-white health disparities in the United States. *J Am Med Dir Assoc* 11:617–628, 2010

[22]Ginde AA, Liu MC, Camargo CA Jr: Demographic differences and trends of vitamin D insufficiency in the US population, 1988–2004. *Arch Intern Med* 169:626–632, 2009

[23]Scragg R, Sowers M, Bell C: Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the Third National Health and Nutrition Examination Survey. *Diabetes Care* 27:2813–2818, 2004

[24]Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF: Decreased bioavailability of vitamin D in obesity. *Am J Clin Nutr* 72:690–693, 2000

[25]Ashraf A, Alvarez J, Saenz K, Gower B, McCormick K, Franklin F: Threshold for effects of vitamin D deficiency on glucose metabolism in obese female African-American adolescents. *J Clin Endocrinol Metab* 94:3200–3206, 2009

[26]Bischoff-Ferrari H, Willett W: Comment on the IOM vitamin D and calcium recommendations: For adult bone health, too low on vitamin D—and too generous on calcium [article online]. Available from http://www.hsph.harvard.edu/nutritionsource/what-should-you-eat/vitamin-d/#iom-vitamin-d. Accessed 25 May 2011

[27]Heaney RP, Holick MF: Why the IOM recommendations for vitamin D are deficient. *J Bone Min Res* 26:455–457, 2011

[28]Gorham ED, Garland CF: Vitamin D and the limits of randomized controlled trials. *Public Health Nutr* 14:741–743, 2011

[29]National Institutes of Health Office of Dietary Supplements: Dietary supplement fact sheet: Vitamin D [article online]. Available from http://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional. Accessed 10 November 2010

[30]Holick MF: Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. *Am J Clin Nutr* 80 (Suppl. 6):1678S–1688S, 2004

[31]Overcash JA: Vitamin D in older patients with cancer. *Clin J Oncol Nurs* 12:655–662, 2008

[32]Rammos G, Tseke P, Ziakka S: Vitamin D, the renin-angiotensin system, and insulin resistance. *Int Urol Nephrol* 40:419–426, 2008

[33]Robinson JG, Manson JE, Larson J, Liu S, Song Y, Howard BV, Phillips L, Shikany JM, Allison M, Curb JD, Johnson KC, Watts N: Lack of association between 25(OH)D levels and incident type 2 diabetes in older women. *Diabetes Care* 34:628–634, 2011

[34]Jorde R, Figenscha Y: Supplementation with cholecalciferol does not improve glycaemic control in diabetic subjects with normal serum 25-hydroxyvitamin D levels. *Eur J Nutr* 48:349–354, 2009

[35]Harris SS: Does vitamin D deficiency contribute to increased rates of cardiovascular disease and type 2 diabetes in African Americans? *Am J Clin Nutr* 93:1175S–1178S, 2011

[36]Arunabh S, Pollack S, Yeh J, Aloia JF: Body fat content and 25-hydroxyvitamin D levels in healthy women. *J Clin Endocrinol Metab* 88:157–161, 2003

[37]Blum M, Dolnikowski G, Seyoum E, Harris S, Booth S, Peterson J, Saltzman E, Dawson-Hughes B: Vitamin D_3 in fat tissue. *Endocrine* 33:90–94, 2008

[38]Parikh SJ, Edelman M, Uwaifo GI, Freedman RJ, Semega-Janneh M, Reynolds J, Yanovski JA: The relationship between obesity and serum 1,25-dihydroxy vitamin D concentrations in healthy adults. *J Clin Endocrinol Metab* 89:1196–1199, 2004

[39]Zenk SN, Schulz AJ, Lachance LL, Mentz G, Kannan S, Ridella W, Galea S: Multilevel correlates of satisfaction with neighborhood availability of fresh fruits and vegetables. *Ann Behav Med* 38:48–59, 2009

[40]Linda N, Amy LY, Jennifer DS, Joel K: Still not enough: can we achieve our goals for Americans to eat more fruits and vegetables in the future? *Am J Prev Med* 32:354–355, 2007

[41]Lappe JM, Davies KM, Travers-Gustafson D, Heaney RP: Vitamin D status in a rural postmenopausal female population. *J Am Coll Nutr* 25:395–402, 2006

[42]Saleh F, Jorde R, Sundsfjord J, Haug E, Figenschau Y: Causes of secondary hyperparathyroidism in a healthy population: the Tromsø study. *J Bone Min Metab* 24:58–64, 2006

[43]Pereira MA, Jacobs DR Jr, Van Horn L, Slattery ML, Kartashov AI, Ludwig DS: Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. *JAMA* 287:2081–2089, 2002

[44]Bantle JP, Wylie-Rosett J, Albright AL, Apovian CM, Clark NG, Franz MJ, Hoogwerf BJ, Lichtenstein AH, Mayer-Davis E, Mooradian AD, Wheeler ML: Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care* 31:61–78, 2008

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