African American females: a potential link between vitamin D insufficiency and type-2 diabetes

Abstract

African American females are a high-risk population for both vitamin D insufficiency and type-2 diabetes. Numerous comparison studies have been conducted between African Americans and other racial groups, regarding vitamin D insufficiency and type 2-diabetes. African Americans, especially African American females, are at a higher risk for becoming insufficient in vitamin D when compared to other ethnic groups. The prevalence of hypovitaminosis D among African American women is ten times higher than white women. In addition, African Americans, especially African American females, have a higher prevalence of type 2-diabetes. African American females surpass white females with 100% prevalence. Although many factors may contribute to the onset of type 2-diabetes, serum 25-hydroxyvitamin D concentrations have been found to have a positive association with insulin sensitivity. In this article, we review the effects of diet and subcutaneous vitamin D synthesis on serum vitamin D status as well as type 2-diabetes prevalence in the most vulnerable population of both conditions, African American females. We further focus on the mechanistic studies in cell cultures and animal models, as well as cohort, intervention, and clinical studies in human subjects. In conclusion, development of vitamin D assessment in the prevention of type 2-diabetes for African American females may provide further insight for intervention and education. Adequate vitamin D intake should be promoted and recommended to decrease disparities within this population.

Keywords: African Americans, deficiency, females, insufficiency, sun exposure, type-2 diabetes, vitamin D

Introduction

Vitamin D insufficiency

Vitamin D is a fat-soluble vitamin found in a few food sources, predominately in foods of animal origin and fortified food products such as milk, margarine, orange juice, and fortified cereals.1-4 This vitamin is well known for its role in bone mineral metabolism, homeostasis of serum blood calcium, regulation of serum phosphorus concentrations, and the effects on cell growth, differentiation, and proliferation.1-3,5 To accurately measure circulating vitamin D to determine adequacy, intoxication, and insufficiency, serum 25-hydroxyvitamin D [25(OH) D] concentrations are commonly used,6 although it has been seen as controversial due to its hydrophobic behavior.6 25(OH) D is the measurement of the body’s storage of vitamin D and indicative of adequate concentrations, while 1, 25-hydroxyvitamin D [1, 25(OH) D] is the active metabolite correlated with kidney function rather than adequacy.7 Serum 25(OH) D concentrations are commonly assessed in nmol/L, with > 50nmol/L being considered desirable. Vitamin D insufficiency, therefore, is defined as having a serum 25(OH) D concentration between 30-50nmol/L, whereas concentrations <30nmol/L are considered deficient.8

Since the Recommended Dietary Allowance (RDA) has increased to 15 μg (600 IU) for vitamin D,5,8 the most recently published National Health and Nutrition Examination Survey (NHANES) for 2009-2010 indicates average amounts of vitamin D consumed per individual by age and gender, as well as ethnicity/race and gender. From these data, female’s ≥20years of age consumed an average of 4.5μg from food per day,9 while African Americans of the same age group consumed an average of 4.1μg from food per day.10 These data suggest that females and African Americans may be at an increased risk for vitamin D insufficiency.

Type 2 diabetes

It is well known that type-2 diabetes (T2D) is a non-insulin dependent form of diabetes, which can develop due to lifestyle factors, including poor diet and lack of physical activity. This chronic condition affects how the body metabolizes glucose due to the pancreas not being able to produce enough insulin. Consequently, when the insulin response becomes compromised, insulin resistance and chronic elevated blood glucose concentrations occur.11 T2D is now a worldwide epidemic with significant prevalence in the United States, affecting 8.3% as of 2011,12 and is projected to affect 36million Americans by 2010-2030.13 These statistics are expected to triple from 2010 to 2050,14 primarily due to the growth of high-risk ethnic minority populations in the United States.14,15

Although many factors may contribute to the onset of T2D, serum 25(OH) D concentrations have been found to have a positive association with insulin sensitivity.16-18 In this article, we review the effects of diet and subcutaneous vitamin D synthesis on vitamin D status as well as T2D prevalence in the most vulnerable population of both conditions, African American females. We further focus on the mechanistic studies in cell cultures and animal models, as well as cohort, intervention, and clinical studies in human subjects.

Current status of knowledge

Vitamin D insufficiency in African Americans and African American females

Numerous comparison studies have been conducted to determine the prevalence of vitamin D insufficiency and differences among various ethnic groups. African Americans, especially females, are at a higher risk of vitamin D insufficiency, when compared to other ethnic groups. One study examined the cross-sectional, epidemiologic data.
from NHANES III and found lower concentrations of vitamin D among women and non-Hispanic blacks, when compared to males and other ethnicities. Adjusting for confounding variables, non-Hispanic blacks (n=2,634) had serum 25(OH)D concentrations of 48.5±1.2nmol/L, compared to non-Hispanic whites (86.5±1.5nmol/L) and Mexican American/Hispanics (65.3±1.2nmol/L). Female subjects (n=4,318) were found to have 25(OH)D concentrations of 75.7±1.4nmol/L, compared to males (n=3,652) who had 80.5±1.3nmol/L. This study suggested the prevalence of vitamin D insufficiency among African Americans especially African American females.

Several studies have been conducted with a focus on females. Nesby O’Dell et al. narrowed the NHANES III cohort study sample to African American women (n=1,546) and non-Hispanic white women (n=1,426), seeking to determine the prevalence of inadequate serum 25(OH)D concentrations among the two groups. Results indicated a 42.4±3.1% prevalence of hypovitaminosis D among African American women, whereas white women had a prevalence of 4.2±0.7%. This was a 10-fold difference. In a nested-case control study, 3,055 postmenopausal women from the Women’s Health Initiative Calcium plus Vitamin D Clinical Trial were assessed to determine variation of serum 25(OH)D concentrations from contributing factors such as oral consumption and latitude of residence. Results indicated that > 50% of subjects were vitamin D insufficient or deficient, with seasonal variation and race being contributing factors. This study also found that white women had higher serum 25(OH)D concentrations compared to other racial groups, as well as a more pronounced variation for those individuals residing in northern and middle latitudes.

In studies specifically focusing on African American females, the majority of subjects were deemed vitamin D insufficient or deficient regardless of age or location. One study was conducted in Waco, Texas at a latitude of 31°N, assessing a small sample size (n=38) of African American elderly women (≥70 years old) to determine serum 25(OH)D insufficiency prevalence. This prospective cohort study found that 86.4% of participants had inadequate serum 25(OH)D concentrations, a quarter of which was deemed deficient. Similarly, research has been conducted through the analysis of electronic medical records at the Jefferson Family Medicine Center, an urban minority primary care practice located in Buffalo, New York. With a sample size of 570 patients, 369 patients (65%) were classified as vitamin D deficient. The majority of the total sample size consisted of women (73%) and African Americans (88%). Although the study did not indicate the number of African American female subjects, 75% of females and 91% of African American subjects were reported to have low serum 25(OH)D concentrations, providing supporting evidence of the prevalence of vitamin D deficiency/insufficiency among females and African Americans.

### Effect of diet on Vitamin D insufficiency in African American and African American females

One of the factors that have been analyzed to determine an association between vitamin D status and African Americans is overall dairy consumption and the prevalence of lactose intolerance within this population. Individuals who do not consume an adequate amount of dairy products are less likely to meet the Dietary Reference Intake (DRI) for certain crucial vitamins and minerals, including vitamin D. Table 1 summarizes studies in vitamin D insufficiency and related dietary factors in African Americans, females, and African American females. African Americans, of different ages and genders in a random sample, did not meet dairy recommendations in a retrospective study comparing NHANES data from 1999-2000, as well as Continuing Survey of Food Intakes by Individuals (CSFII) 1994-1996. In particular, African American females, regardless of age, consumed less dairy food than males. According to Moore et al., within the population of African Americans, females were ½ as likely to consume enough vitamin D to reach recommended intake compared to their male counterparts.

| Subjects | Findings | Reference |
|----------|----------|-----------|
| 7,970 subjects, approximately 67% non-Hispanic black and Mexican American/Hispanic Male & Female | Higher prevalence of vitamin D insufficiency was observed in women and non-Hispanic blacks | 19 |
| African American (1,546 subjects) and non-Hispanic white (1,426 subjects) Female | Hypovitaminosis D was 10 times higher among African American females, compared to whites | 20 |
| 3,055 subjects, including African American, Asian/Pacific Islander, Hispanic, white, and American Indian Female | More than half of the women were vitamin D insufficient or deficient; white women had higher serum 25(OH)D concentrations compared to other racial groups | 21 |
| African American (38 subjects) Female | 86.4% of subjects had inadequate serum 25(OH)D levels | 22 |
| 570 subjects, 73% women and 88% African American Male & Female | 91% of African American subjects were vitamin D insufficient, and 75% of females were insufficient | 23 |
| African American (2,414 subjects) and non-African American (16,428 subjects) Male & Female | African Americans did not meet dairy recommendations, and African American females consumed less dairy food than males | 24 |
| 45,976 subjects, race not specified Male & Female | Female teenagers and adults reported lowest intakes of vitamin D from food | 25 |
| 409 subjects, race not specified Male & Female | The majority of subjects consumed less than the AI for vitamin D, and the number of insufficient females was greater | 26 |
| 4,727 subjects, African American and white Male & Female | African Americans consumed less vitamin D from both the diet and supplementation than were whites | 27 |
| 2,379 subjects, African American and white Female | African American girls had lower serum 25(OH)D levels compared to their counterparts, and an avoidance of dairy products among African Americans was observed | 28 |

Citation: Soriano KM, Li Y, Zhang T. African American females: a potential link between vitamin D insufficiency and type-2 diabetes. J Nutr Health Food Eng. 2014;1(3):96–103. DOI: 10.15406/jnhfe.2014.01.00015
Subcutaneous vitamin D Synthesis in African Americans and African American females

Aside from dietary consumption and supplementation, there are a variety of factors that affect the subcutaneous synthesis of vitamin D, with some specifically influencing the rate and amount of synthesis in African Americans. Production of cholecalciferol/vitamin D₃ is influenced by a number of factors including melanin (darker pigmentation), aging, clothing, use of sunscreen, and altitude.³,4,9 Since most humans depend on the sun to meet daily vitamin D requirements, lower production of cholecalciferol due to these contributing factors could cause a significant issue.⁵ For example, seasonal changes, especially during the winter and spring, tend to lead to lower concentrations of serum 25(OH)D in people of all ethnicities.³,4,9 An observational study estimating cholecalciferol production during each season for approximately 2,000 young Americans in the North (45°N) and the South (35°N) indicated an association between the amount of sun exposure and seasonal variation on vitamin D status.⁶

Because melanin in the darker skin acts as a natural sunscreen,⁷ African Americans are at a higher risk of vitamin D insufficiency, especially during spring and winter months. One intervention study focused on African American women (n=117) located in Nashville, Tennessee and Caucasian women (n=102) who resided in Hershey, Pennsylvania. Results were indicative of racial differences, with African American females having significantly lower serum 25(OH)D concentrations (27.3nmol/L) compared to Caucasian women (52.4nmol/L).⁸ This study provided strong evidence for the disparity between these racial groups, considering the location of African Americans was more southern with more sun exposure. A Southern Community Cohort Study was conducted to assess the serum 25(OH)D concentrations for 395 subjects in the southern United States.⁹ Enrollees in this study were African American and Caucasian decent. Vitamin D deficiency was prevalent among 45% of African Americans, compared to only 11% of Caucasians. These two studies suggested that African Americans and African American females have a higher risk and prevalence for vitamin D deficiency in relation to seasonal variation and the amount of sun exposure, regardless of geographical location.³,9 Therefore, increased sun exposure time, without the use of sunscreen, has been recommended for this population.³,¹⁴,¹⁵ Otherwise African Americans may not achieve optimal cholecalciferol production during these months.⁶

Type 2 diabetes in African Americans and African American females

Among the millions of people with T2D, prevalence remains the highest for minority groups, including African Americans, Asian Americans, and Hispanic Americans.¹⁰ Within these groups, African Americans are twice as likely to be diagnosed with T2D when compared to non-Hispanic white Americans,²⁹ with the number of undiagnosed cases being higher as well.³⁰ The African American race has been independently associated with the incidence of T2D.³⁰ African American females are at a higher risk for becoming diagnosed with T2D, when compared to African American males. While African American men have a 60% higher prevalence compared to non-Hispanic white men, African American women surpass non-Hispanic white women with 100% prevalence.⁴¹

A variety of factors contribute to the development of T2D, including insulin insensitivity, weight status, family history, or poor glycemic control. African American women showed a decrease in insulin sensitivity independent of fat distribution and other potential contributing factors such as weight status and inflammation.²⁶,⁴¹
may contribute to the higher prevalence and risk in this population compared to other racial groups. In addition, they have been found to have poorer glycemic control, as well as the highest odds of having increased glycosylated hemoglobin (HbA1c) level. These are indicative of an increase in plasma glucose level over a prolonged amount of time, which may lead to T2D and potentially more severe consequences of the eyes, kidneys, and nerves. In Table 2, we summarized studies in T2D prevalence in African Americans and African American females.

| Subjects | Findings | Reference |
|----------|----------|-----------|
| 14,611 subjects, including Non-Hispanic black, non-Hispanic white, and Mexican American Male & Female | Non-Hispanic blacks were twice as likely to be diagnose with T2D when compared to non-Hispanic whites, with the number of undiagnosed cases being higher as well | 39 |
| African American (2,322 subjects) and white (8,840 subjects) Male & Female | African American race was determined to be independently associated with the incidence of T2D | 40 |
| African American and non-Hispanic white (number not specified) Male & Female | African American men had a 60% higher prevalence compared to non-Hispanic white men, while African American women surpassed non-Hispanic white women with 100% prevalence | 41 |
| African American (108 subjects) and white (105 subjects) Female | African American women had decreased insulin sensitivity independent of obesity, inflammation, and fat distribution | 42 |
| African American and white, number not specified Male & Female | African Americans had a high risk and prevalence for T2D, insulin resistance, and vitamin D deficiency | 43 |
| 1,480 subjects, including non-Hispanic white, non-Hispanic black, and Mexican American Male & Female | More non-Hispanic blacks were treated with insulin compared to non-Hispanic whites and Mexican Americans; poor glycemic control was more common among non-Hispanic black women | 44 |

**Table 2** Type 2 diabetes in African Americans and African American females

A link between Vitamin D insufficiency and type 2 diabetes: cell and animal studies

A number of cell and animal studies have been conducted to understand the mechanistic relationship between T2D and vitamin D. Vitamin D is suggested to play a functional role in the regulation of glucose tolerance, insulin secretion and sensitivity. An early study observed that 1,25-hydroxyvitamin D₃ injection increased insulin secretion in vitamin D-deficient rats, suggesting a possible feedback loop between insulin synthesis and 1,25-hydroxyvitamin D₃. In a more recent study which used T2D models established from Wistar and spontaneously hypertensive rats, cholecalciferol supplementation elicited a significant reduction of 40% in blood glucose concentrations in all Wistar rats and a reduction by 60% in 40% of the spontaneously hypertensive rats. In relation to insulin response, vitamin D-deficient Wistar rats were followed over a 4week time span and treated with 1,25-hydroxyvitamin D₃. Results indicated an improved insulin release from islets upon glucose stimulation and an increased rate of conversion of proinsulin to insulin. These studies presented evidence of potential activation of insulin biosynthesis in islets upon 1,25-hydroxyvitamin D₃ treatment or cholecalciferol supplementation.

One of the mechanisms behind the effect of vitamin D on insulin relates to the presence of vitamin D receptors (VDR) and vitamin D-binding proteins (VDBP) on pancreatic β-cells. Furthermore, the active metabolite of vitamin D has been demonstrated to act as a stimulator of insulin receptor expression and insulin response for glucose transport. In a recent study conducted by Cheng et al., mice with diet-induced hypovitaminosis D were found to develop impaired glucose tolerance and islet function gene transcription. While vitamin D is necessary to suppress renin production, pharmacological renin inhibitor was able to reduce insulin dysfunction and insulin resistance and improve glucose tolerance during continuing vitamin D deficiency. Comparatively, the altered gene expression of insulin receptors and VDR in the cerebellum of streptozotocin-induced diabetic rats was recovered to near control level when the rats were treated with vitamin D₃ supplementation and insulin injection. Another study observed that nutritional therapy using vitamin D₃, as well as curcumin, improved glucose homeostasis and reversed an array of molecular events in the skeletal muscle of streptozotocin-induced diabetic rats to near normal concentrations, including β₂-adrenoceptor function and insulin receptor expression. For instance, reversal of the insulin receptor down-regulation was found in vitamin D₃-, insulin-, and curcumin-treated rats. In addition, 1,25-hydroxyvitamin D₃ has been found to have protective effects against diabetic retinopathy in male diabetic Sprague-Dawley rats, which might be explained by the inhibition of VEGF and TGF-β₁ expression in the retinal tissue. All of these studies provided evidence to support that vitamin D supplementation can reduce blood glucose levels by regulating pancreatic insulin synthesis/secretion, insulin receptor expression, and VDR expression, which may result in the reduced risk of T2D.

A link between Vitamin D insufficiency and type 2 diabetes: human studies

Among human studies, individuals who are at risk for vitamin D insufficiency are also at risk for developing T2D, as these two conditions share similar risk factors. A number of studies have suggested a relationship between vitamin D status and insulin sensitivity/fasting blood glucose as well as T2D risk (Table 3). Insufficient serum 25(OH)D concentrations have been inversely related to increased adiposity, elevated blood glucose concentrations and insulin resistance. For example, the intake of vitamin D was inversely associated with high blood glucose, low HDL, and the risk of developing metabolic syndrome in African Americans and whites. A study focusing on women at late reproductive age found that vitamin D deficiency was correlated with increased body fat and glucose levels, and decreased insulin sensitivity. In addition, vitamin D deficiency has been suggested to contribute to the impairment of insulin secretion and insulin action in T2D patients.

**Citation:** Soriano KM, Li Y, Zhang T. African American females: a potential link between vitamin D insufficiency and type-2 diabetes. J Nutr Health Food Eng. 2014;1(3):96–103. DOI: 10.15406/jnhfe.2014.01.00015
Newton et al. found an inverse association between fasting glucose and increased vitamin D consumption within a group of African American girls in a clinical study. The authors also observed a positive relationship between increased vitamin D and fasting insulin and insulin sensitivity. Similarly, an analysis of the cross-sectional NHANES III to determine an association between serum 25(OH)D, ethnicity and diabetes risk, was conducted. Insulin resistance was found to be higher among non-Hispanic blacks compared to whites, and the diabetes risk varied inversely across quartiles of 25(OH)D in a dose-dependent manner. However, there was no inverse association observed in non-Hispanic blacks, which was explained by the threshold effect experienced by them. Overall, this study showed that vitamin D status was associated with diabetes risk. Furthermore, an intervention study found that insulin resistance and fasting blood glucose were decreased in T2D patients who received vitamin D supplementation.

Table 3 Relationship between vitamin D status and type 2 diabetes in human subjects

| Subjects | Findings | Reference |
|----------|----------|-----------|
| 4,727 subjects, African American and white Male & Female | The intake of vitamin D was inversely associated with high blood glucose, low HDL, and the risk of developing metabolic syndrome | 37 |
| Obese African American (28 subjects) Female | An inverse association was found between fasting glucose and increased vitamin D consumption within this group, as well as a positive relationship between increased vitamin D and fasting insulin and insulin sensitivity | 58 |
| Non-Hispanic white (2,766 subjects), non-Hispanic black (1,736 subjects), and Mexican American (1,726 subjects) Male & Female | Insulin resistance was found to be higher among non-Hispanic blacks compared to whites, and the diabetes risk varied inversely across quartiles of 25(OH)D in a dose-dependent manner | 62 |
| 320 subjects, race not specified Female | Vitamin D deficiency was correlated with increased body fat and glucose levels, and decreased insulin sensitivity | 64 |
| 10 T2D patients, race not specified Female | Vitamin D deficiency was suggested to contribute to the impairment of insulin secretion and insulin action in T2D patients | 65 |
| 100 T2D patients, race not specified Male & Female | Insulin resistance and fasting blood glucose were decreased in T2D patients who received vitamin D supplementation | 66 |
| 147 pregnant women, race not specified Female | Serum 25(OH)D and blood glucose levels have been found to be independent predictors of HbA1c levels (which is the strongest predictor for T2D incidence) | 68 |
| 120 T2D patients, race not specified Male & Female | The 25(OH)D concentrations were lower in these patients, and these concentrations were inversely associated with HbA1c levels; vitamin D supplementation was suggested to improve glucose control in these patients | 69 |
| 309 T2D patients and 143 controls, French Male & Female | Polymorphisms of the VDR gene were associated with susceptibility to obesity and early-onset of T2D | 71 |
| African American (379 subjects) and Caucasian (379 subjects) Male & Female | Of the five single nucleotide polymorphisms (SNPs) being investigated, three SNPs accounted for a 4.6% variation in serum vitamin D among African Americans. African Americans indicated a six-fold risk in of vitamin D insufficiency compared to Caucasians. | 75 |

Research has been conducted to determine how HbA1c level, the strongest predictor for T2D incidence, is influenced by 25(OH)D. In a sample of 147 pregnant women at Westmead Hospital, serum 25(OH)D and blood glucose levels were found to be independent predictors of HbA1c levels, with lower 25(OH)D concentrations being independently associated with poorer glycemic control. The 25(OH)D concentrations were lower in T2D patients, and these concentrations were inversely associated with HbA1c levels in the diabetic patients. This study suggested that vitamin D supplementation may improve glucose control in T2D patients. Interestingly, a cohort study, which focused on insulin resistance and hyperinsulinemia in obese persons, found both variables to drive a negative association among vitamin D and inflammation parameters, suggesting higher insulin levels and/or insulin resistance are the main factors responsible for decreased vitamin D concentrations. Results indicated that insulin or HOMA\textsubscript{IR} (homeostasis model assessment, used to assess insulin resistance) maintained a significant independent association with 25(OH)D levels in all multiple regressions presented, but not BMI or triglycerides.

**Gene polymorphisms, vitamin D insufficiency, and type-2 diabetes**

Gene polymorphisms have been another focus of current vitamin D and diabetes research. Variance among genes involved in vitamin D functions and processes, such as its transport and synthesis, can influence vitamin D status. Although there have been numerous studies conducted involving different genes, The VDR gene is the most common one. There was evidence that VDR is an underlying factor in the development and continuance of several metabolic conditions, although the pathophysiological reasoning remains unclear. For example, VDR gene polymorphisms have been noted in relation to diabetes and obesity status. VDR gene may influence the development of type-1 and type-2 diabetes. This receptor has also been found to be associated with an increased risk of...
African American females: a potential link between vitamin D insufficiency and type-2 diabetes

74

A study analyzing VDR single nucleotide polymorphisms (SNPs) (FokI, Apal, BsmI, TaqI) found that certain polymorphisms had a greater impact on diabetes risk than others. Wang et al., aimed to determine associations among these polymorphisms and diabetes risk for types 1 and 2. Results indicated the FokI polymorphism to be significantly associated with T2D risk but not type 1, whereas the BsmI polymorphism was associated with type 1 diabetes risk and no associations found with Apal and TaqI in either type of diabetes. Alternatively, a cross-sectional study aiming to determine associations among VDR polymorphisms and T2D among individuals of Indian and African descent found the prevalence of vitamin D deficiency to be significantly lower in the presence of FokI and Apal polymorphisms.

This study did not observe an association between vitamin D status and BsmI or TaqI polymorphisms. Additional research focusing on VDR polymorphisms in relation to diabetes and vitamin D deficiency is warranted in the future.

Amidst the research on VDR, several SNPs of vitamin D pathway genes have been investigated to determine an association with serum 25(OH)D, including CYP27B1 and GGC. CYP27B1 is a gene which promotes the hydroxylation of vitamin D to its active form vitamin D₃, which then binds to the VDR, regulating calcium metabolism and homeostasis. GGC, the group-specific complement gene, encodes the protein that binds to vitamin D and its metabolites, which are then transported to target tissues for use. As the participants in the Southern Community Cohort Study, African Americans were compared to Caucasians for specific genetic predictors relating to vitamin D status. Signorello et al. identified significant associations with two SNPs in the Ggc gene (rs2298849 and rs2282679) and one SNP in the CYP27B1 gene (rs10877012), but only for African Americans. These results suggested that African Americans might be genetically at greater risk for insufficient serum 25(OH)D concentrations, compared to Caucasians. This study, among the first few studies to investigate common genetic variation in relation to vitamin D levels in African Americans, not only provided insights regarding racial disparities, but also contributed to the identification of subpopulation who may reduce the risk factors associated with the development of T2D, and a balanced diet rich in vitamin D and African Americans inhibit adequate synthesis of cholecalciferol, increasing sun exposure time is needed in order to penetrate the skin and produce vitamin D subcutaneously. Thus serum 25(OH)D concentrations will increase and may prevent the onset of T2D.

In conclusion, low serum 25(OH)D concentrations may lead to the development of T2D, and a balanced diet rich in vitamin D may reduce the risk factors associated with the development of T2D. Due to African American females being at a higher risk for the development of T2D and vitamin D insufficiency, adequate vitamin D intake should be promoted and recommended to decrease disparities within this population. 

Acknowledgments

None.

Conflict of interest

Author declares that there is no conflict of interest.

References

1. Gropper SA, Smith JL, Groff JL. Advanced nutrition and human metabolism. 5th ed. Australia: Wadsworth/Cengage Learning; 2009. 623p.

2. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington DC: National Academy Press. 1997. p. 250–287.

3. Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr. 2004;80(6 Suppl):1678S–1686S.

4. McGuire M, Beerman KA. Nutritional sciences: from fundamentals to food. 2nd ed. California: Thomson/Wadsworth. 2006. 704p.

5. http://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/

6. Hollis BW, Horst RL. The assessment of circulating 25(OH)D and 1,25(OH)2D: where we are and where we are going. J Steroid Biochem Mol Biol. 2007;103(3–5):473–476.
African American females: a potential link between vitamin D insufficiency and type-2 diabetes

7. Hollis BW. Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency; implications for establishing a new effective dietary intake recommendation for vitamin D. J Nutr. 2005;135(2):317–322.

8. Ross AC. Dietary reference intakes: calcium, vitamin D. Washington, DC: National Academies Press. 2011.

9. What We Eat in America, NHANES 2009-2010 (p. 4). Nutrient Intakes from Food: Mean Amounts Consumed per Individual, by Gender and Age. Washington, DC: U.S. Department of Agriculture, Agricultural Research Service. 2012.

10. http://www.mayoclinic.com/health/type-2-diabetes/DS00585

11. http://diabetes.niddk.nih.gov/dm/pubs/statistics/

12. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract. 2010;87(1):4–14.

13. Boyle JP, Thompson TJ, Gregg EW, et al. Projection of the year 2050 burden of diabetes in the US adult population: dynamic modeling of incidence, mortality, and prediabetes prevalence. Popul Health Metr. 2010;8(29):1–12.

14. Holland AT, Zhao B, Wong EC, et al. Racial/ethnic differences in control of cardiovascular risk factors among type 2 diabetes patients in an insured, ambulatory care population. J Diabetes Complications. 2013;27(1):34–40.

15. Alvarez JA, Ashraf AF, Hunter GR, et al. Serum 25-hydroxyvitamin D and parathyroid hormone are independent determinants of whole-body insulin sensitivity in women and may contribute to lower insulin sensitivity in African Americans. Am J Clin Nutr. 2010;92(6):1344–1349.

16. Chiu KC, Chu A, Go VL, et al. Hypovitaminosis D is associated with insulin resistance and beta cell dysfunction. Am J Med. 2004;79(5):820–825.

17. Shaban LH, Zarini GG, Esehio JC, et al. Serum vitamin D insufficiency and diabetes status in three ethnic minority groups. J Immigr Minor Health. 2012;14(6):926–932.

18. Ganji V, Milone C, Cody MM, et al. Serum vitamin D concentrations are related to depression in young adult US population: the third national health and nutrition examination survey. Int Arch Med. 2010;3(29):1–8.

19. Nesby-O’Dell S, Scanlon KS, Cogswell ME, et al. Hypovitaminosis D prevalence and determinants among African American and white women of reproductive age: third national health and nutrition examination survey, 1988-1994. Am J Clin Nutr. 2002;76(1):187–192.

20. Millen AE, Wactawski-Wende J, Pettinger M, et al. Predictors of serum 25-hydroxyvitamin D concentrations among postmenopausal women: the women’s health initiative calcium plus vitamin D clinical trial. Am J Clin Nutr. 2010;91(5):1324–1335.

21. Weaver SP, Passmore C, Collins B, et al. Vitamin D, sunlight exposure, and bone density in elderly African American females of low socioeconomic status. Fam Med. 2010;42(1):47–51.

22. Kahn LS, Satishchandran N, Kopparapu A, et al. High prevalence of undetected vitamin D deficiency in an urban minority primary care practice. J Natl Med Assoc. 2011;103(5):407–411.

23. Fulgoni V 3rd, Nichols J, Reed A, et al. Dairy consumption and related nutrient intake in African-American adults and children in the United States: continuing survey of food intakes by individuals 1994-1996, 1998, and the national health and nutrition examination survey 1999-2000. J Am Diet Assoc. 2007;107(2):256–264.

24. Moore C, Murphy MM, Keast DR, et al. Vitamin D intake in the United States. J Am Diet Assoc. 2004;104(6):980–983.

25. Seabolt L, Spence TB, Silver HJ. Consistent prevalence of inadequate micronutrient intakes across six years of second-year medical school students. Health. 2012;4(7):357–365.

26. Fung TJ, Steffen LM, Zhou X, et al. Vitamin D intake is inversely related to risk of developing metabolic syndrome in African American and white men and women over 20 y: the Coronary Artery Risk Development in Young Adults study. Am J Clin Nutr. 2012;96(1):24–29.

27. Van Horn LV, Bausermann R, Affenito S, et al. Ethnic differences in food sources of vitamin D in adolescent American girls: the national heart, lung, and blood institute growth and health study (NGHS). Nutr Rev. 2011;31(8):579–585.

28. Keith JN, Nicholls J, Reed A, et al. The prevalence of self-reported lactose intolerance and the consumption of dairy foods among African American adults are less than expected. J Natl Med Assoc. 2011;103(1):36–45.

29. http://digestive.niddk.nih.gov/ddis/pubs/lactoseintolerance/

30. Jarvis JK, Miller GD. Overcoming the barrier of lactose intolerance to reduce health disparities. J Natl Med Assoc. 2002;94(2):55–66.

31. Holick MF, Chen TC, Lu Z, et al. Vitamin D and skin physiology: a D-lightful story. J Bone Miner Res. 2007;22(Suppl 2):V28–V33.

32. Rajakumar K, Holick MF, Jeong K, et al. Impact of season and diet on vitamin D status of African American and Caucasian children. Clin Pediatr (Phila). 2011;50(6):493–502.

33. Godar DE, Pope SJ, Grant WB, et al. Solar UV doses of young Americans and vitamin D production. Environ Health Perspect. 2012;120(1):139–143.

34. Clemens TL, Adams JS, Henderson SL, et al. Increased skin pigment reduces the capacity of skin to synthesise vitamin D. Lancet. 1982;1(8263):74–76.

35. Conzy P, Demers LM, Dudoon WC, et al. Determination of vitamin D in relation to body mass index and race in a defined population of black and white women. Int J Gynaecol Obstet. 2012;119(1):21–25.

36. Egan KM, Signorello LB, Munro HM, et al. Vitamin D insufficiency among African-Americans in the southeastern United States: implications for cancer disparities (United States). Cancer Causes Control. 2008;19(5):527–535.

37. http://minorityhealth.hhs.gov/templates/content.aspx?ID=3017

38. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and high risk for diabetes using A1C criteria in the U.S. population in 1988-2006. Diabetes Care. 2010;33(3):562–568.

39. Chatterjee R, Brancati FL, Shafi T, et al. Non-traditional risk factors are important contributors to the racial disparity in diabetes risk: the Atherosclerosis Risk in Communities Study. J Gen Intern Med. 2013;28(2):290–297.

40. Harris MI. Noninsulin-dependent diabetes mellitus in black and white Americans. Diabetes Metab Rev. 1990;6(2):71–90.

41. Hyatt TC, Phadke RP, Hunter GR, et al. Insulin sensitivity in African-American and white women. Obesity (Silver Spring). 2009;17(2):276–282.

42. Tsi A, Giovannucci EL, Hyperinsulinemia, insulin resistance, vitamin D, and colorectal cancer among whites and African Americans. Dig Dis Sci. 2012;57(10):2497–2503.

43. Harris MI, Eastman RC, Cowie CC, et al. Racial and ethnic differences in glycemic control of adults with type 2 diabetes. Diabetes Care. 1999;22(3):403–408.

44. http://www.nlm.nih.gov/medlineplus/ency/article/003640.htm

45. Palomer X, Gonzalez-Clemente JM, Blanco-Vaca F, et al. Role of vitamin D in the pathogenesis of type 2 diabetes mellitus. Diabetes Obes Metab. 2008;10(3):185–197.

46. Sung CC, Liao MT, Lu KC, et al. Role of vitamin D in insulin resistance. J Biomed Biotechnol. 2012;2012:634195.
47. Clark SA, Stumpf WE, Sar M. Effect of 1,25 dihydroxyvitamin D₃ on insulin secretion. *Diabetes*. 1981;30(5):382–386.
48. de Souza Santos R, Vianna LM. Effect of cholecalciferol supplementation on blood glucose in an experimental model of type 2 diabetes mellitus in spontaneously hypertensive rats and Wistar rats. *Clin Chim Acta*. 2005;358(1–2):146–150.
49. Bourlon PM, Billaudel B, Faure-Dussert A. Influence of vitamin D₃ deficiency and 1,25 dihydroxyvitamin D₃ on de novo insulin biosynthesis in the islets of the rat endocrine pancreas. *J Endocrinol*. 1999;160(1):87–95.
50. Maestro B, Campion J, Davila N, et al. Stimulation by 1,25-dihydroxyvitamin D₃ of insulin receptor expression and insulin responsiveness for glucose transport in U-937 human promonocytic cells. *Endocr J*. 2000;47(4):383–391.
51. Maestro B, Molero S, Bajo S, et al. Transcriptional activation of the human insulin receptor gene by 1,25-dihydroxyvitamin D₃. *Cell Biochem Funct.* 2002;20(3):227–232.
52. Cheng Q, Boucher BJ, Leung PS. Modulation of hypovitaminosis D-induced islet dysfunction and insulin resistance through direct suppression of the pancreatic islet renin-angiotensin system in mice. *Diabetologia*. 2013;56(3):553–562.
53. Peeyush KT, Savitha B, Sherin A, et al. Cholinergic, dopaminergic and insulin receptors gene expression in the cerebellum of streptozotocin-induced diabetic rats: functional regulation with vitamin D₃ supplementation. *Pharmacol Biochem Behav*. 2010;95(2):216–222.
54. Xavier S, Sadanandan J, George N, et al. β₂-Adrenoceptor and insulin receptor expression in the skeletal muscle of streptozotocin-induced diabetic rats: antagonism by vitamin D₃ and curcumin. *Eur J Pharmacol*. 2012;687(1–3):14–20.
55. Ren Z, Li W, Zhao Q, et al. The impact of 1,25-dihydroxy vitamin D₃ on the expressions of vascular endothelial growth factor and transforming growth factor-β1 in the retinas of rats with diabetes. *Diabetes Res Clin Pract*. 2012;98(3):474–480.
56. Alvarez JA, Ashraf A. Role of vitamin D in insulin secretion and insulin sensitivity for glucose homeostasis. *Int J Endocrinol*. 2010;2010:351385.
57. Newton AL, Hanks LJ, Ashraf AP, et al. Macronutrient intake influences the effect of 25-hydroxyvitamin D status on metabolic syndrome outcomes in African American girls. *Cholesterol*. 2012;2012:581432.
58. Rajakumar K, de las Heras J, Chen TC, et al. Vitamin D status, adiposity, and lipids in black American and Caucasian children. *J Clin Endocrinol Metab*. 2011;96(5):1560–1567.
59. Tidwell DK, Valiant MJ. Higher amounts of body fat are associated with inadequate intakes of calcium and vitamin D in African American women. *Nutr Rev*. 2011;69(7):527–536.
60. Via M. The malnutrition of obesity: micronutrient deficiencies that promote diabetes. *JSRN Endocrinol*. 2012;2012:103472.
61. Scarfeg R, Sowers M, Bell C, et al. Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the Third National Health and Nutrition Examination Survey. *Diabetes Care*. 2004;27(12):2813–2818.
62. Anderson JI, May HT, Horne BD, et al. Relation of vitamin D deficiency to cardiovascular risk factors, disease status, and incident events in general healthcare population. *Am J Cardiol*. 2010;106(7):963–968.
63. Grineva EN, Karonova T, Micheeva E, et al. Vitamin D deficiency is a risk factor for obesity and diabetes type 2 in women at late reproductive age. *Aging (Albany NY)*. 2013;5(7):575–581.
64. Borissova AM, Tanevka T, Kirilov G, et al. The effect of vitamin D₃ on insulin secretion and peripheral insulin sensitivity in type 2 diabetic patients. *Int J Clin Pract*. 2003;57(4):258–261.
65. Talaci A, Mohamadi M, Adgi Z. The effect of vitamin D on insulin resistance in patients with type 2 diabetes. *Diabetol Metab Syndr*. 2013;5(1):8.
66. Lim S, Kim MJ, Choi SH, et al. Association of vitamin D deficiency with incidence of type 2 diabetes in high-risk Asian subjects. *Am J Clin Nutr*. 2013;97(3):524–530.
67. Lau SL, Gunton J, Athayde N, et al. Serum 25-hydroxyvitamin D and glycated haemoglobin levels in women with gestational diabetes mellitus. *Med J Aust*. 2011;194(7):334–337.
68. Kostoglou-Athanssou I, Athanasissou P, Giountouvas A, et al. Vitamin D and glycemic control in diabetes mellitus type 2. *Ther Adv Endocrinol Metab*. 2013;4(4):122–128.
69. DePergola G, Nitti A, Bartolomeo N, et al. Possible role of hyperinsulinemia and insulin resistance in lower vitamin D levels in overweight and obese patients. *Biomed Res Int*. 2013;2013:921348.
70. http://www.ncbi.nlm.nih.gov/gene/7421
71. De Azevedo Silva J, Guimaraes RL, Brandao LA, et al. Vitamin D receptor (VDR) gene polymorphisms and age onset in type 1 diabetes mellitus. *Autoimmun*. 2013;46(6):382–387.
72. Ye WZ, Reis AF, Dubois-Laforge D, et al. Vitamin D receptor gene polymorphisms are associated with obesity in type 2 diabetic subjects with early age of onset. *Eur J Endocrinol*. 2001;145(2):181–186.
73. Wang Q, Xi B, Reilly KH, et al. Quantitative assessment of the associations between four polymorphisms (FokI, Apal, BsmI, TaqI) of vitamin D receptor gene and risk of diabetes mellitus. *Mol Biol Rep*. 2012;39(10):9405–9414.
74. Velayoudom-Cephise FL, Larilfa L, Donnet JP, et al. Vitamin D deficiency, vitamin D receptor gene polymorphisms and cardiovascular risk factors in Caribbean patients with type 2 diabetes. *Diabetes Metab*. 2011;37(6):540–545.
75. http://www.ncbi.nlm.nih.gov/gene/1594
76. http://www.ncbi.nlm.nih.gov/gene/2638
77. Signorelli LB, Shi J, Cai Q, et al. Common variation in vitamin D pathway genes predicts circulating 25-hydroxyvitamin D levels among African Americans. *PLoS ONE*. 2011;6(12):e28623.
78. McCormack S, Grant SF. Genetics of obesity and type 2 diabetes in African Americans. *J Obes*. 2013;2013:396416.
79. Harris SS, Vitamin D and African Americans. *J Nutr*. 2006;136(4):1126–1129.

Citation: Soriano KM, Li Y, Zhang T. African American females: a potential link between vitamin D insufficiency and type 2 diabetes. *J Nutr Health Food Eng*. 2014;1(3):96–103. DOI: 10.15406/jnhe.2014.01.00015