Review Article

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 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

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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,[3] 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 innmol/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.[5]

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. Consequentially, 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.[19] Adjusting for confounding variables, non-Hispanic blacks (*n*=2634) 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*=4318) were found to have 25(OH) D concentrations of 75.7±1.4nmol/L, compared to males (*n*=3652) who had 80.5±1.3nmol/L.[19] 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.[20] narrowed the NHANES III cohort study sample to African American women (*n*=1546) and non-Hispanic white women (*n*=1426), 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%.[20] This was a 10-fold difference. In a nested-case control study, 3055 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.[21] 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.[21]

In studies specifically focusing on African American females, the majority of subjects were deemed vitamin D insufficient or deficient regardless of age or location.[22,23] 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 (≥70years 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.[22] 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 Ddeficiency/insufficiency among females and African Americans.[23]

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.[24] According to Moore et al.,[25] 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.[26]

Table 1 Vitamin D insufficiency and related dietary factors in African Americans, females, and African American females

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 (1546 subjects) and non-Hispanic white (1426 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]

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Table continued..

Subjects	Findings	Reference
African American (2,016 subjects) and general population (1,084 subjects) Male & Female	African Americans were more likely to experience discomfort after eating dairy and identified themselves lactose intolerant	29
African American (94 subjects) and Caucasian (46 subjects) Male & Female	African American children had lower vitamin D concentrations compared to Caucasians	33
African American (117 subjects) and white (102 subjects) Female	African American women (located in Nashville, Tennessee, more southern with more sun exposure) had lower serum 25 (OH) D levels compared to white women (located in Hershey, Pennsylvania)	36
395 subjects, African Americans and Caucasian Male & Female	Hypovitaminosis D was more prevalent among African Americans (45%) compared to Caucasians (11%), and insufficient levels were more prevalent among females compared to males	37

One study aimed to compare black and white subjects from the longitudinal Coronary Artery Risk and Development in Young Adults study and their vitamin D intake from dietary and supplement sources. Vitamin D intake was quantified based on self-reported consumption through diet histories and 24hour recalls. When comparing the two ethnic groups, blacks were found to have consumed less vitamin D from both the diet and supplementation than were whites.[27] The National Heart, Lung and Blood Institute of Growth and Health Survey was a 10year longitudinal study focusing on vitamin D intake among African American and Caucasian girls ages 9 and 10.[28] This biracial cohort study found that African American girls had significantly lower serum 25(OH) D concentrations compared to their counterparts. The most common vitamin D food sources consumed by Caucasian girls were fortified milk, margarine and eggs, while African American girls consumed vitamin D from fish, fats/oils, and meat,[28] suggesting that the preferences differ among racial groups. This study indicated an avoidance of dairy products among African Americans. In relation to lactose intolerance and avoidance of dairy products, the National Dairy Council sought to determine the prevalence of lactose intolerance between African Americans and the general population, as well as its influence on dairy choices. Of the 2016 African American subjects participating in an online survey, 49% of them indicated physical discomfort when consuming dairy products, were more likely to identify themselves lactose intolerant, and ate fewer dairy products.[29] This population has a high prevalence of lactose intolerance and experiences more discomfort when consuming dairy products,[29,30] which limits their selection of vitamin D-rich foods. The prevalence of lactose intolerance among the African American population may be due to genetic transmission.[31]

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 D_3 is influenced by a number of factors including melanin (darker pigmentation), aging, clothing, use of sunscreen, and altitude.[3,14,32] 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.[3] 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.[3,33,34] An observational study estimating cholecalciferol production during each season for approximately 2,000 young Americans in the North (45 ⁰N) and the South (35 0N) indicated an association between the amount of sun exposure and seasonal variation on vitamin D status.[34]

Because melanin in the darker skin acts as a natural sunscreen,[35] 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).[36] 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.[37] 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.[36,37] Therefore, increased sun exposure time, without the use of sunscreen, has been recommended for this population.[3,14,34] Otherwise African Americans may not achieve optimal cholecalciferol production during these months.[34]

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.[38] Within these groups, African Americans are twice as likely to be diagnosed with T2D when compared to non-Hispanic white Americans[38,39] with the number of undiagnosed cases being higher as well.[39] The African American race has been independently associated with the incidence of T2D.[40] 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.[42,43] This

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.[44] 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.[45] In Table 2, we summarized studies in T2D prevalence in African Americans and African American females.

Table 2 Type 2 diabetes 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

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.[46,47] An early study observed that 1,25-hydroxyvitamin D_3 injection increased insulin secretion in vitamin D-deficient rats, suggesting a possible feedback loop between insulin synthesis and 1,25-hydroxyvitamin D_3.[48] 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.[49] In relation to insulin response, vitamin D-deficient Wistar rats were followed over a 4week time span and treated with 1,25-hydroxyvitamin D_3. Results indicated an improved insulin release from islets upon glucose stimulation and an increased rate of conversion of proinsulin to insulin.[50] These studies presented evidence of potential activation of insulin biosynthesis in islets upon 1,25-hydroxyvitamin D_3 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.[46,47] 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.[51,52] In a recent study conducted by Cheng et al.,[53] 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 islet dysfunction and insulin resistance and improve glucose tolerance during continuing vitamin D deficiency.[53] 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 D_3 supplementation and insulin injection.[54] Another study observed that nutritional therapy using vitamin D_3, 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 β_2-adrenoceptor function and insulin receptor expression. For instance, reversal of the insulin receptor down-regulation was found in vitamin D_3-, insulin- and curcumin-treated rats.[55] In addition, 1,25-hydroxyvitamin D_3 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.[56] 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.[57] 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,[27,58–61] elevated blood glucose concentrations[27,58] and insulin resistance.[12,13,43,58,62,63] 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.[27] 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.[64] In addition, vitamin D_3 deficiency has been suggested to contribute to the impairment of insulin secretion and insulin action in T2D patients.[65]

Newton et al.,[58] 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.[58] 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.[62] 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.[62] Furthermore, an intervention study found that insulin resistance and fasting blood glucose were decreased in T2D patients who received vitamin D supplementation.[66]

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	27
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	73
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.	78

Research has been conducted to determine how HbA1c level, the strongest predictor for T2D incidence,[67] 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.[68] 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.[69] Interestingly, a cohort study, which focused on insulin resistance and hyperinsulinemia in obese patients, 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 $HOMA_{IR}$ (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.[70]

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

Gene polymorphisms have been another focus of current vitamin D and diabetes research. Variances 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.[71] 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.[72] This receptor has also been found to be associated with an increased risk of

becoming obese, among subjects who had early-onset diabetes.[73] A study analyzing VDR single nucleotide polymorphisms (SNPs) (*Fok*I, *Apa*I, *Bsm*I, *Taq*I) found that certain polymorphisms had a greater impact on diabetes risk than others.[74] Wang et al.,[74] aimed to determine associations among these polymorphisms and diabetes risk for types 1 and 2. Results indicated the *Fok*I polymorphism to be significantly associated with T2D risk but not type 1, whereas the *Bsm*I polymorphism was associated with type 1 diabetes risk and no associations found with *Apa*I and *Taq*I in either type of diabetes.[74] Alternatively, a cross-sectional study aiming to determine associations among VDR polymorphisms and T2D among individuals of Indian and African decent found the prevalence of vitamin D deficiency to be significantly lower in the presence of FokI and *Apa*I polymorphisms.[75] This study did not observe an association between vitamin D status and *Bsm*I or *Taq*I polymorphisms. Additional research focusing on VDR polymorphisms in related 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 *Gc*. *CYP27B1* is a gene which promotes the hydroxylation of vitamin D to its active form vitamin D_3, which then binds to the VDR, regulating calcium metabolism and homeostasis.[76] *Gc*, 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.[77] 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.[78] identified significant associations with two SNPs in the *Gc* 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.[76] 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 particularly in need of vitamin D intervention.[78] Therefore, there are a variety of risk factors that may lead to insufficient serum 25(OH) D concentrations and the development of T2D, including genetics. Although African Americans are at greater risk for T2D or insufficient vitamin D status based on genetic predisposition, there is not enough supportive evidence yet to promote race or genetics as the sole factor for vitamin D insufficiency and/or T2D prevalence among African Americans.[79] More research involving genetic factors will contribute to understanding the health disparities of insufficient vitamin D and diabetes risk/prevalence among African Americans.

Conclusion

Vitamin D insufficiency and T2D are two major health concerns for the general population, however, due to the increased amount of melanin in the skin, a higher prevalence of insulin insensitivity, gene polymorphisms, and decreased consumption of vitamin D-rich foods, African American females are the most vulnerable population to become affected by one or both of these conditions. Females and African Americans have been the subjects of many comparison studies, with the majority of the research indicating females, African Americans, or African American females to be at higher risk for the development of vitamin D insufficiency and T2D. Importantly, a number of studies support the potential connection between vitamin D insufficiency/deficiency and development of T2D, and a few of them specifically investigated the African American population. Studies using cell culture and animal models provide 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. Furthermore, several human studies have shown the effects of serum 25(OH) D on blood glucose, insulin sensitivity/resistance, HbA1c, and T2D risk.

All of these studies support the need of increasing vitamin D recommendation for the population. As the prevalence of T2D and vitamin D insufficiency continue to rise in number, increasing the AI of vitamin D may help to decrease the incidence of insufficiency and indirectly aid in the prevention of T2D. Increasing vitamin D needs will benefit the general population, but more specifically, African Americans and African American females. Nutrition education programs may strongly help this population, with a focus on self-monitoring of dietary consumption and promotion of vitamin D-rich foods. Because African Americans have a higher prevalence of lactose intolerance and consume less dairy products compared to other races.[24,28,29] alternative vitamin-D rich food sources should be promoted, such as fatty fish, orange juice, and fortified cereals. Vitamin D supplementation may be recommended as another source for individuals who are vitamin D deficient. As with food consumption, sun exposure recommendation should be increased for African Americans. Because the higher melanin levels in the darker skin of African Americans inhibit adequate synthesis of cholecalciferol,[35,80] 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.

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Conflict of interest

Author declares that there is no conflict of interest.

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