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RESEARCH ARTICLE

Can Black Americans Reduce or Eliminate Racial Health Disparities by Getting More Sun Exposure?

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ABSTRACT

It is becoming well understood that insufficient sun exposure is a risk factor for many adverse health effects. Increased adverse health effects such as cancers and cardiovascular disease have been quantified with respect to measured levels of vitamin D, a marker for sun exposure. Most acquired vitamin D [measured as 25(OH)D level] is a result of ultraviolet radiation from sun exposure. Because of higher levels of melanin in the skin, individuals with darker skin obtain less vitamin D and other beneficial sun-produced biomolecules such as nitric oxide from a given amount of sun exposure. It is the purpose of this paper to report the published observed 25(OH)D levels in Black Americans and the correlated levels of adverse health effects. Our conclusion is that insufficient sun exposure is a major component of the observed health disparities between Black and White Americans, and that Black Americans can significantly attenuate these disparities by having enough additional sun exposure to raise their 25(OH)D levels to 30 ng/mL.

## Introduction

Racial disparities in health between Black and White Americans have existed in the United States for well over 100 years<sup>1</sup> and continue to exist, but the reasons for these disparities have never been satisfactorily explained. Recent efforts by the U.S. Centers for Disease Control and Prevention to eliminate these disparities are focused on the social determinants of health, which include inequities in access to a range of social and economic benefits such as housing, education, wealth and employment<sup>2</sup>, but there is little evidence that the social determinants of health are responsible for the observed disparities<sup>3,4</sup>.

Here, the author reviews the health risks that have been found to be associated with insufficient sun exposure, the emergence of insufficient sun exposure as a significant public health problem for the population as a whole, the reasons for the higher amounts of melanin in the skin of Black Americans, the role of melanin in causing the health risks associated with insufficient sun exposure to be greater for Black Americans than for White Americans, and the higher incidence of diseases associated with insufficient sun exposure in Black Americans compared to White Americans. The author suggests that racial disparities in health between Black Americans and White Americans may be attributable in large part to the movement of Black Americans' ancestors from equatorial Africa, where they evolved with high amounts of cutaneous melanin to protect them from the strong equatorial sun, to the higher latitudes of the United States where the weaker sun combined with higher amounts of cutaneous melanin exacerbates the health risks associated with insufficient sun exposure for Black Americans.

To the knowledge of the author, this is the first published article to examine this component of racial health disparities between Black and White Americans and to suggest that Black Americans can attenuate these disparities by getting more sun exposure. Other recent reviews examined this same component of racial health disparities, but suggested vitamin D supplementation to attenuate these disparities rather than sun exposure<sup>5,6</sup>. This is an important distinction since vitamin D supplementation has been found not to be an effective substitute for sun exposure<sup>7</sup>.

This review focuses on Black-White health disparities in the United States because other high-

latitude countries with significant Black populations, such as France, Germany, Spain, Italy, and most other European countries do not keep health data by race<sup>8</sup>, and the few that do such as Great Britain do not have sufficient data for detailed analysis<sup>9</sup>.

## Health Risks of Insufficient Sun Exposure

The first disease to be attributed to insufficient sun exposure was rickets in 1919<sup>10,11</sup>, a widespread bone deforming disease in children that arose during the industrial revolution with its close-packed cities, heavy air pollution from coal burning and use of child labor, all of which resulted in children having very little sun exposure<sup>12</sup>. This was followed in 1924 by the discovery that an inactive lipid in the diet and skin could be converted by ultraviolet radiation (UVR) from the sun into an antirachitic substance<sup>13</sup>. The identification of this antirachitic substance as vitamin D occurred in 1931<sup>14</sup>. Vitamin D is formed in human body by UVR photons from the sun converting 7-dehydrocholesterol located in the lower layers of the skin<sup>15</sup> into previtamin D<sub>3</sub>, which is rapidly converted to vitamin D<sub>3</sub>. Vitamin D<sub>3</sub> is then metabolized in the liver to form 25-hydroxyvitamin D (25(OH)D), which is used to determine a person's vitamin D status. 25(OH)D is then metabolized in the kidneys to its active form, 1-25-dihydroxyvitamin D<sup>16</sup>.

Discoveries like these would have normally led to a flurry of scientific inquiry into other health problems associated with insufficient sun exposure. However, the discovery in the 1920s that sun exposure could induce skin cancer<sup>17</sup>, together with the high prevalence of skin cancer in white persons who moved to Australia from England, changed the direction of scientific inquiry into health problems associated with excessive sun exposure, as Australia became the skin cancer center of the world<sup>18</sup>. The white skin of these persons contained enough melanin to protect them from skin cancer in the weak sun of England but not nearly enough to stand up to the strong tropical sun of Australia<sup>19</sup>. The first chemical sunscreen was developed in 1928<sup>20</sup>, creating a commercial interest in protection from the sun. The prevailing health advice regarding sun exposure became avoid sun exposure when possible, seek shaded areas, wear sun-protective clothing and hats and apply sunscreen daily<sup>21</sup>.

It was not until the beginning of the 21st century that scientific interest and research turned toward the health problems associated with insufficient sun exposure<sup>7</sup>. People in the United States were

experiencing less and less sun exposure because of migration of the workforce from outdoor to indoor work and increasing attractions of being indoors such as radio, air conditioning, television, video games and the internet<sup>7</sup>. In addition, advice of dermatologists and governmental health authorities was generally sun avoidance for fear of skin cancer<sup>21,22</sup>. According to a report of the Environmental Protection Agency to Congress in 1989, people in the United States at that time were spending only 2% of their time outdoors<sup>23</sup>.

Advances in science in the 21st century have revealed the significant increased risks of a wide range of diseases and adverse health conditions associated with insufficient sun exposure, including decreased life expectancy, hypertension, cardiovascular disease, stroke, cancer, type 1 and type 2 diabetes, multiple sclerosis, Alzheimer's disease, metabolic syndrome, autism, schizophrenia, asthma, respiratory infection, rheumatoid arthritis, sudden infant death syndrome, preeclampsia, pre-term birth and COVID-19<sup>7,24</sup>. In 2020, fifteen of the leading scientists in the world on sun exposure and human health published a comprehensive review alerting that insufficient sun exposure had become a real public health problem in the United States, responsible for 340,000 premature deaths per year, second only to tobacco with 480,000 premature deaths per year<sup>7</sup>.

Sun-produced mediators other than vitamin D have been discovered, the most important of which is nitric oxide<sup>7</sup>. The 1998 Nobel Prize in Medicine was awarded to three scientists who discovered the importance of nitric oxide as a ubiquitous signaling molecule in the cardiovascular system that protects the heart, stimulates the brain and kills bacteria<sup>25,26</sup>, but where the nitric oxide came from remained a mystery until the discovery in 2009 that UVR converts cutaneous stores of photolabile nitric oxide derivatives in the lower levels of the skin to nitric oxide which causes a pronounced decrease in blood pressure<sup>7,27-30</sup>.

Other UVR-induced mediators include cytokines, corticotropin-releasing hormone, urocortins, proopiomelanocortin-peptides and enkephalins that are released into circulation to produce systemic effects independent of vitamin D synthesis<sup>31,32</sup>. UVR has been shown to have a large effect on the skin transcriptome and there is evidence that UVR may also affect the blood transcriptome including genes for immunity<sup>33,34</sup>.

## Solar Radiation

The sun emits ultraviolet radiation (UVR) across a broad spectrum from the highest-energy UVC band (wavelengths below 280 nanometers) to the UVB band (wavelengths between 280 and 315 nanometers) to the UVA band (wavelengths between 315 and 400 nanometers)<sup>35</sup>. UVR borders the visible band (wavelengths from 400 to 800 nanometers)<sup>35</sup>. Almost all of the UVC is absorbed by the atmosphere with negligible amounts reaching the surface of the Earth<sup>35</sup>. UVB, but not UVA, produces vitamin D in the human body<sup>12</sup>. Because the Earth is round, the sun strikes the surface at different angles, ranging from 0° (just above the horizon) to 90° (directly overhead). When the sun's rays are vertical, the Earth's surface gets all the energy possible. The more slanted the sun's rays are, the longer they travel through the atmosphere, becoming more scattered and diffuse<sup>36</sup>. As a result, the power of the sun is much higher at the equator than it is in the United States. The average UV Index at the equator is 18 year round, while the average UV Index in Miami, Florida is 10-11 in the summer and 4 in the winter, and the average UV Index in New York, New York is 6-7 in the summer and 1-2 in the winter<sup>37</sup>.

## Melanin

Melanin is a photoprotective pigment in human skin that protects the body from excessive UVR from the sun<sup>38</sup>. It is a redox UVR-absorbing agent that functions as a shield to reduce UVR action on DNA in epidermal cells while permitting enough UVR penetration to produce 25-hydroxyvitamin D, nitric oxide and other beneficial biomolecules<sup>38</sup>.

The amount of melanin in human skins evolved to have the proper balance between absorbing enough UVR to protect against skin cancer without absorbing so much UVR as to inhibit the UVR-produced biomolecules necessary for good health<sup>5,7</sup>, an evolutionary compromise between the risks and benefits of sun exposure<sup>6</sup>. Thus humans who evolved under the powerful equatorial sun developed larger amounts of melanin in their skins than humans who evolved under the weaker sun of higher latitudes<sup>5-7</sup>. Since melanin is the most important pigment determining skin color, skin with more melanin is darker in color than skin with less melanin<sup>38</sup>. As a result, Black Americans, whose ancestors evolved under the equatorial African sun, have more melanin in their skins than White Americans and therefore require more sun exposure

than White Americans to make the same amounts of the UVR-produced biomolecules necessary for good health<sup>5</sup>.

### **Serum 25(OH)D as a Marker for Sun Exposure**

The sufficiency of sun exposure received by a person can be determined by measuring the level of the biomolecule 25(OH)D in the person's blood, since 70-90% of this biomolecule is produced by sun exposure<sup>39-41</sup>. Levels of 25(OH)D below 30 ng/mL indicate insufficient sun exposure<sup>7,42</sup>, and 62.2% of White Americans and 94.5% of Black Americans have 25(OH)D levels below 30 ng/mL<sup>42</sup>. In the CDC's NHANES III study by the CDC, 13,331 adults had their 25(OH)D levels measured with the data divided into four quartiles (>32.1, 24.4-32.1, 17.8-24.4 and <17.8 of ng/ml 25(OH)D). Among those in the lowest vitamin D level quartile, 9.5% were non-Hispanic whites while 50.3% were non-Hispanic blacks. In the highest quartile, 43.5% were non-Hispanic whites and only 7.8% were non-Hispanic blacks<sup>43</sup>. In a study conducted by Nesby-O'Dell et al. of the CDC's NHANES III cohort, 1546 Black Americans had their 25(OH)D levels measured. The prevalence of hypovitaminosis D, defined as 25(OH)D < 15 ng/mL, was 42.4% among Black American women<sup>44</sup>. Maasai tribesmen in Africa today still living traditional pastoral lives outdoors have 25(OH)D levels of 46 ng/mL<sup>45</sup>, indicating that this may be the natural level of 25(OH)D for persons with black skin and possibly for all persons. In comparison, the average level of 25(OH)D in Black Americans is 16 ng/mL<sup>46</sup>.

### **Diseases and Adverse Health Conditions Associated With Insufficient Sun Exposure and Incidence of Such Diseases and Adverse Health Conditions in Black Americans Compared to White Americans**

The following are some of the major diseases and adverse health conditions associated with insufficient sun exposure, together in each case with a comparison of the incidence of such diseases and adverse health conditions in Black Americans and White Americans. In this discussion, low levels of serum 25(OH)D are considered as a marker or proxy for insufficient sun exposure.

#### *All-Cause Mortality*

In 2014, Chowdhury et al.<sup>47</sup> found that 25(OH)D levels of less than 17.5 ng/mL compared to more than 24.3 ng/mL were associated with shortened life expectancy and that 12.8% of U.S. deaths

(340,000 deaths) were attributable to 25(OH)D levels less than 30 ng/mL. Lindqvist et al.<sup>48</sup> found that avoidance of sun exposure is a risk factor for early death of the same order of magnitude as smoking. The life expectancy of Black Americans is four years lower than that of White Americans<sup>49</sup>.

#### *Colorectal Cancer*

In 2007, Gorham et al.<sup>50</sup> found that levels of 25(OH)D less than 12 ng/mL as compared to more than 33 ng/mL were associated with a 100% increased risk of colorectal cancer. The incidence of colon cancer is 19% higher in Black Americans than in White Americans, and Black Americans are 145% more likely to die from colon cancer than White Americans<sup>51</sup>.

#### *Stomach Cancer*

In 2016, Vyas et al.<sup>52</sup> found that persons with 25(OH)D less than 20 ng/mL compared to more than 30 ng/mL have a 170% increased risk of stomach cancer. Black American men are 1.7 times as likely to have stomach cancer as White American men and are 2.5 times more likely to die from stomach cancer, and Black American women are twice as likely to have stomach cancer as White American women and are 2.2 times as likely to die from stomach cancer<sup>53</sup>.

#### *Hypertension and Cardiovascular Disease*

In 2009, Mowbray et al.<sup>27</sup> and Oplander et al.<sup>28</sup> found that UVA radiation converts cutaneous stores of photolabile nitric oxide derivatives to nitric oxide with the result of significant enhanced concentration of plasma nitroso compounds and a pronounced decrease in blood pressure. Hypertension is the leading risk factor for cardiovascular and cerebrovascular disease and underlies 18% of all deaths worldwide<sup>7</sup>. A 2016 study found that people with habitual low sun exposure were at twice the risk of cardiovascular mortality than those with the greatest sun exposure<sup>54</sup>. Notably, no association of cardiovascular disease with vitamin D has been found, either in Mendelian randomization studies or in randomized controlled trials with vitamin D supplementation<sup>5</sup>. The incidence of hypertension is 37% higher in Black Americans than in White Americans<sup>55</sup>, and mortality due to hypertension and its consequences is 4 to 5 times more likely in Black Americans than in White Americans<sup>56</sup>. The incidence of hypertension in black-skinned persons is far lower in Africa and increases in a consistent gradient from Africa to the

Caribbean to the United States<sup>57</sup>. Black Americans are 30% more likely to die from heart disease than White Americans<sup>58</sup>. Black American women are 60% more likely to have high blood pressure than White American women<sup>58</sup>.

#### *Lower Respiratory Tract Infection (LRTI)*

In 2011, Belderbos et al.<sup>59</sup> found that neonates born with 25(OH)D concentrations less than 20 ng/mL had a 500% increased risk of viral LRTI in the first year of life compared with those with 25(OH)D concentrations greater than 30 ng/mL, indicating that insufficient sun exposure of pregnant women increases the risk of LRTI in offspring. The LRTI infant mortality rate for Black American infants is 44.1 per 100,000 live births compared to 18.7 per 100,000 for White American infants<sup>60</sup>.

#### *Type 2 Diabetes*

In 2013, Afzal et al.<sup>61</sup> found that 25(OH)D levels of less than 5 ng/mL compared to more than 20 ng/mL were associated with a 35% increased risk of type 2 diabetes. The incidence of type 2 diabetes is 100% higher in Black Americans than in White Americans<sup>62</sup>, and Black Americans are twice as likely to die from type 2 diabetes as White Americans<sup>63</sup>.

#### *Alzheimer's Disease*

In 2014, Littlejohns et al.<sup>64</sup> found that persons with serum 25(OH)D less than 10 ng/mL compared to more than 20 ng/mL have a 122% increased risk of Alzheimer's disease. The incidence of Alzheimer's disease in Black Americans is 100% higher than in White Americans<sup>65</sup>.

#### *Stroke*

In 2013, Brondum-Jacobsen et al.<sup>66</sup> found that 25(OH)D levels of less than 5 ng/mL compared to more than 20 ng/mL were associated with an 82% increased risk of ischemic stroke, and that 25(OH)D levels of less than 10 ng/mL compared to more than 30 ng/mL were associated with a 36% increased risk of ischemic stroke. Black Americans are 50% more likely to have a stroke than White Americans, Black American women are twice as likely to have a stroke as White American women and Black American men are 70% more likely to die from a stroke as White American men<sup>67</sup>.

#### *Rheumatoid Arthritis*

In 2013, Arkema et al.<sup>68</sup> found that persons with the lowest category of UVB exposure compared to the

highest category of UVB exposure had 37% increased risk of rheumatoid arthritis. The incidence of rheumatoid arthritis in Black American men is 50% higher than in White American men, and is 160% higher in Black American women than in White American women<sup>69</sup>.

#### *Sudden Infant Death Syndrome (SIDS)*

In 2013, Cohen et al.<sup>70</sup> found that in a cohort of 25 cases of SIDS, 75.5% had serum 25(OH)D less than 30 ng/mL and 63.5% had 25(OH)D less than 20 ng/mL. SIDS rates of Black American children are more than twice those of White American children<sup>71</sup>.

#### *Asthma*

In 2014, Zosky et al.<sup>72</sup> found that maternal 25(OH)D levels at 16 to 20 weeks' gestation of less than 20 ng/mL compared to more than 20 ng/mL were associated with a 203% increased risk of their male offspring having asthma at age 6. The incidence of asthma in the United States is 40% higher in Black Americans than in White Americans, the mortality rate of asthma is 200% higher for Black Americans than for White Americans, and Black American children are 5 times more likely to be admitted to the hospital for asthma than White American children<sup>73</sup>.

#### *Preeclampsia*

In 2014, Bodnar et al.<sup>74</sup> found that women who have 25(OH)D of less than 20 ng/mL compared with more than 20 ng/mL in the first 26 weeks of pregnancy have a 54% increased risk of preeclampsia. The preeclampsia rate is 60% higher in Black American women than in White American women, and Black American women are more likely to develop severe preeclampsia<sup>75</sup>.

#### *Obesity*

In 2014, Geldenhuys et al.<sup>76</sup> found that skin induction of the sunlight-induced mediator nitric oxide, but not vitamin D supplementation, suppresses obesity. The incidence of obesity in Black Americans is 30% higher than in White Americans<sup>77</sup>.

#### *Preterm Birth*

In 2015, Bodnar et al.<sup>78</sup> found that maternal 25(OH)D levels at or before 20 weeks' gestation of less than 20 ng/mL compared to more than 30 ng/mL were associated with a 1.8-fold risk of preterm birth. The preterm birth rate is 64% higher for Black American women than for White American



women<sup>79</sup>. Black American infants have 2.3 times the mortality rate of White American infants, and Black American infants are 3.8 times more likely to die from complications related to low birthweight as compared to White American infants<sup>80</sup>.

#### *Autism*

In 2017, Vinkhuysen et al.<sup>81</sup> found that maternal 25(OH)D levels at mid-gestation of less than 10 ng/mL compared to more than 20 ng/mL were associated with a more than two-fold increased risk of autism spectrum disorder in the child. Children of Black American mothers are 14% more likely to be diagnosed with autism spectrum disorder than children of White American mothers<sup>82</sup>.

#### *Schizophrenia*

In 2018, Eyles et al.<sup>83</sup> found that neonates with 25(OH)D less than 8 ng/mL compared to more than 21 ng/mL have a 44% increased risk of schizophrenia in later life. Black Americans are more than twice as likely as White Americans to be diagnosed with schizophrenia<sup>84</sup>.

#### *Covid-19*

A recent meta-analysis of data from 54 studies involving a total of 1,400,000 individuals found that persons with 25(OH)D less than 20 ng/mL compared to more than 20 ng/mL have a 138% increased risk of hospitalization from COVID-19 and an 84% increased risk of death from COVID-19<sup>85</sup>. Black Americans are 2.8 times as likely to be hospitalized for COVID-19 as White Americans, and 2.0 times as likely to die from COVID-19<sup>86</sup>.

### **Amount of Sun Exposure Needed**

Serum 25(OH)D levels of less than 30 ng/mL have been associated with all-cause mortality<sup>47</sup> and increased risk of colorectal cancer<sup>50</sup>, stomach cancer<sup>52</sup>, LRTI<sup>59</sup>, SIDS<sup>69</sup> and preterm birth<sup>78</sup>, indicating that all persons should have serum 25(OH)D levels of at least 30 ng/mL. Levels higher than 30 ng/mL may be beneficial, since as noted above persons still living traditional pastoral lives outdoors have levels of 46 ng/mL<sup>45</sup>. Therefore, Black Americans need enough sun exposure to maintain their serum 25(OH)D levels (as shown on a standard vitamin D test) at 30 ng/mL or higher. The amount of sun exposure needed to accomplish this objective will be different for each person.

### **Confounding**

The studies cited herein correlating increased risk of various diseases and adverse health conditions with low levels of serum 25(OH)D have adjusted such increased risk for potential confounding variables as set forth in such studies. However, findings based on observational epidemiology remain susceptible to the possibility of residual confounding. No determination has been made by the author regarding the portion of racial health disparities caused by insufficient sun exposure and the portion caused by the social determinants of health or other factors.

### **Why More Sun Exposure Rather Than Vitamin D Supplements**

The conclusion expressed herein that the remedy for Black Americans is more sun exposure rather than vitamin D supplements is based in part on the fact that UVR exposure produces nitric oxide<sup>27-30</sup> and other beneficial biomolecules<sup>31-34</sup> in the body in addition to vitamin D, whereas vitamin D supplements produce only vitamin D. Nitric oxide has been shown to lower blood pressure<sup>27,28</sup> while randomized controlled trials have shown that vitamin D supplements do not<sup>87</sup>, and hypertension is one of the greatest risk factors for good health<sup>7</sup>. Ames et al.<sup>5</sup> notes that vitamin D supplementation has been found to have no effect on reducing all-cause mortality, and cites Manson et al.<sup>88</sup> for its conclusion that "Vitamin D supplementation did not reduce invasive cancer incidence or cardiovascular events."

### **Skin Cancer**

Black Americans have far less skin cancer than White Americans. The incidence of melanoma in Black Americans is 0.8 per 100,000 compared to 25.1 per 100,000 for White Americans<sup>89</sup>, and most melanomas in Black Americans are a subtype of melanoma called acral lentiginous melanoma (ALM). ALM is not sun-induced and occurs on the palms of hands, the soles of feet and under nails<sup>90</sup>.

### **Conclusion**

Insufficient sun exposure coupled with the larger melanin content of Black Americans' skin is highly associated with the racial health disparities between Black Americans and White Americans. Black Americans could reduce their risks of a wide range of debilitating diseases and premature death by obtaining considerably more sun

exposure. Vitamin D supplementation is not an effective substitute for sun exposure because vitamin D supplementation does not produce the important biomolecule nitric oxide which is critical in reducing the risk of cardiovascular disease, the leading cause of death in the United States. The evidence suggests that obtaining enough sun exposure to raise blood levels of 25(OH)D to 30 ng/mL is needed to attenuate these risks. Public

health messaging and policy should reflect these realities.

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