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A Call to Action-The Need to Address Obesity in the Black Community

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Over the last 3 decades, we have witnessed the emergence of yet another disease which disproportionately impacts patients of color—obesity. While the overall prevalence of obesity in the US is 38.9%, non-Hispanic blacks have a significantly higher prevalence of 47.6.[1] Even though overweight and obesity affects nearly one half of non-Hispanic blacks, obesity is a disease that is often ignored because clinicians tend to focus on the complications of obesity, including cardiovascular disease, type 2 diabetes, and cancers, among many other diseases. We do this at our own peril because many of those affected begin with an elevated and unaddressed BMI, which if treated early, could potentially decrease the millions of deaths as a result of cardiovascular disease and other obesity associated diseases.[2]

Currently, 17.3 million deaths per year occur as a result of cardiovascular disease, many which result from chronic excess adiposity.[3] The surplus of adipose tissue initiates the release of inflammatory mediators including tumor necrosis factor α (TNF-α) and interleukin 6 (IL-6), while simultaneously reducing the production of adiponectin.[4] This cascade predisposes the body to a pro-inflammatory state and oxidative stress which lay the foundation for cardiovascular disease including coagulation, atherosclerosis, metabolic syndrome, insulin resistance, and Type 2 diabetes mellitus (T2DM).

Another manifestation of excess adiposity is the association between many types of cancers and obesity. There is a wealth of research demonstrating the causal link between obesity and cancer at 13 anatomic sites including the esophagus, colon, rectum, liver, gallbladder, pancreas, prostate, postmenopausal breast, uterine, ovarian, kidney, meningioma, thyroid, and multiple myeloma.[5] Notably, overweight in youth and young adulthood has also been observed to increase risk of many cancers linked to adult excess weight.[6,7] As childhood and young adult obesity rises, we continue to witness a rise in obesity related cancers as adults live a larger portion of their lives in an obesogenic and inflammatory milieu. Currently, many of the obesity related cancers have a higher prevalence in the African American population including prostate, colon, pancreatic, ovarian, kidney, meningioma, and multiple myeloma.

The higher prevalence of obesity in African American communities has been well established and the causes are multifactorial.[8] Many of the factors that are associated with obesity in African Americans are not unique to this population, but the confluences of genetic, behavioral, and environmental factors in the prevailing sociocultural context creates a unique risk pattern that contributes to the observed prevalence. Fundamentally, one should begin by examining the role of genetics in the obesity epidemic as experienced by African Americans. Much genetic research has uncovered the potential role of the desensitization of leptin, a satiety hormone which acts on the Proopiomelanocortin (POMC) pathway located in the lower ventromedial hypothalamus. The differences in leptin hormone production, response, and release may explain higher obesity rates in different racial/ethnic groups including the higher prevalence in the African American community.[9] An additional genetically determined pathway that demonstrates a link between the greater prevalence of obesity in African Americans is the role of Brain Derived Neurotrophic Factor (BDNF). BDNF acts as a downstream regulator of the leptin-proopiomelanocortin pathway. A lower expression of BDNF in the lower ventromedial hypothalamus has been associated with higher levels of adiposity and higher BMI in pediatric and adult patients.[10] A specific allele, rs12291063 CC genotype, has been found to be associated with lower levels of BDNF. This allele has been found to be significantly higher in African Americans than Caucasian cohorts and to be positively associated with an increase in BMI. Additional genetic research discovered the association between the higher levels of protein SEMA4D and a greater prevalence of obesity after studying the serum of both West Africans and African Americans.[11] Carriers of the SEMA4D C Allele were also found to be 4.6 BMI units greater than individuals with the T allele which was found to be greater in those of European and Asian descent. The interplay between the environment and genetics has been linked with the A allele of the OXTR gene, elevated BMI and adiposity when children were raised in low socioeconomic status (SES) environments.[12] When children who were carriers of the same allele, were raised in high SES conditions, the resulting elevated BMI was not noted thus magnifying the association between genetics and the environment. While it is clear that body size is heritable, body size is a complex genetic trait; for example, a meta-analysis of approximately 340,000 individuals identified 97 genome wide significant loci that were associated with BMI. These 97 loci accounted for 2.7% of the variation in BMI.[13] This underscores the fact that common obesity is often a combination of a genetic predisposition and environmental and behavioral factors that lead to the presenting phenotype.

The role of the environment is further emphasized by the frequency of food deserts and areas with lower quality food options throughout lower SES and largely minority neighborhoods. Such neighborhoods, while frequently saturated with fast food options and convenience stores, are void of adequate supplies of lean proteins and fresh fruits and vegetables. African Americans are more likely to reside in these types of neighborhoods due to longstanding social injustices and structural discrimination that has limited access to more affluent neighborhoods or adequately developing areas that were predominately minority.[14] As a result of these constraints, individuals residing in these communities are exposed to obesogenic environments with higher frequency and durations. Minorities residing in lower SES communities consume a greater amount of calorically dense foods void of optimal

nutritional value than those who reside in wealthier SES communities.[15] African American and Hispanic children consume greater amounts of sugar sweetened beverages and fast food by the age of 2 than their Caucasian counterparts.[15] While socioeconomics appears to play an active role in the development of the obesogenic environment, there are fewer fresh food options, less recreational options, and more fast-food choices in African American neighborhoods when compared to white neighborhoods with similar SES suggesting that more than income is at play.[16] Inadequate access to plentiful and affordable healthy options combined with a potential increase in genetic predisposition has undoubtedly exacerbated the increased prevalence of obesity in communities of color.

Physical activity is another important lifestyle behavior associated with obesity. Increased activity, access to organized sports and play grounds is associated with a lower prevalence of pediatric obesity.[17] Additionally, African Americans were less likely to meet public health recommendations of than were non-Hispanic whites: 36.4%and 52.2%, respectively according to the 2012 National Health Interview Survey.[18] Lower SES neighborhoods frequently have fewer physical activity resources than medium to high SES neighborhoods. Children living in in lower SES communities were 50 percent more likely to be physically inactive, 52 percent more likely to watch television more than 2 h per day, and 65 percent more likely to engage in recreational computer use of more than 2 h per day than children living in most favorable social conditions.[19] Such conditions lead to greater inactivity of neighborhood residents.[20] Economically disadvantaged minorities reside in areas of low SES with less access to safe areas for healthy physical activity at a greater rate than Caucasian families.[21]

Psychosocial factors including stress and sleep, play an integral role in the increase prevalence of obesity in communities of color. As a response to stress, leptin causes an increase in the production of inflammatory mediators such as IL-6 and C-reactive protein (CRP), both of which have been implicated in adult and pediatric obesity.[22] A myriad of stresses including racism, are experienced with greater frequency in African American women according to The Black Women’s Health Study. Likewise, the beneficial anti-inflammatory effects of adiponectin have been found to be decreased in patients with obesity. There has also been a significant amount of research demonstrating the relationship between sleep deprivation and obesity in the adult population.[23] In the pediatric population, African American and Hispanic children sleep less at all ages with the greatest deficit noted between 6 months and 7 years of age. Contrary to sleep patterns seen in people with obesity, lower obesity risk has been associated with healthy sleep patterns including in children and adolescents.[24]

As with many chronic diseases, people of color are affected at greater rates than Caucasians. Despite the noted increase in prevalence of obesity in minority communities, patients of color are less likely to be formally diagnosed with obesity and subsequently less likely to have a conversation with their provider about obesity treatment.[25] It has been noted that most patients of color receive their health care from physicians of color. As such, it is plausible that physicians of color therefore play a key role in reducing the disparity between those patients with obesity and the less than 50% that are actually diagnosed with the disease. Likewise, we can contribute to reducing the disparity between those that are

diagnosed with obesity and the less than 25% that actually receive follow up care. Perhaps we can lead the change towards curbing this epidemic that disproportionately affects patients of color. Treating obesity does not mandate being an obesity specialist. The burden is far greater than the number of American Board of Obesity Medicine (ABOM) certified physicians.[26] Treating obesity begins with a mere conversation about our patient's weight status and any complications due to obesity. It begins with providing support, education, and referral when needed for patients who struggle with the chronic disease of obesity.[27] Treatment begins with providing practical guidance on lifestyle changes and reflecting upon potential triggers of excessive caloric intake and weight retention, inactivity, and sleep quality and duration, weight promoting medications, and circadian rhythm disturbances. In cases where a provider is not prepared or equipped to fully implement a treatment plan, treatment includes referring to an ABOM certified physician or a provider who has proven track record of providing comprehensive care including lifestyle modification, behavior therapy, anti-obesity medications and surgical referral when appropriate. No longer should we accept as adequate the idea of only treating the complications of obesity without addressing the root cause of those diseases, i.e., the obesity. It is therefore incumbent upon us who care for these patients that bear the greatest burden of obesity to begin the conversation. We must rise up, educate, and act in order to combat the disease that has affected so many persons of African descent.

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