

Health Disparities and the Ancestral Environment

Health Disparities and the Ancestral Environment:

An Evolutionary Perspective

By

Anthony R. Mawson

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PREFACE

For nearly two decades I was honored to be a professor of epidemiology and biostatistics in the School of Public Health at Jackson State University, an historically black college and university (HBCU). During the years 2006-2010 I was a professor of pediatrics and medicine at the University of Mississippi Medical Center. There I began teaching a graduate school course of health disparities. I was also honored to serve as the first principal investigator of the National Children's Study for Mississippi.

In earlier years at Jackson State University, I helped to organize several national conferences on eliminating health disparities, and two annual training courses on research career survival skills for junior minority faculty. Development of these training courses was in partnership with the University of Pittsburgh's Center for Minority Health. I returned to JSU in 2011 with an investigator-initiated research grant from Pfizer. The research aimed to test a hypothesis on the role of retinoids in onchocerciasis, and it involved a partnership with colleagues at the Tanzanian Institute for Medical Research. I continued to teach a graduate course on health disparities and participated in helping to prepare JSU to become, in 2018, the first accredited School of Public Health at an HBCU in the nation.

These and other experiences deepened my knowledge and curiosity about health disparities affecting African Americans. The theory proposed here also derives partly from my long-standing research interest in the role of vitamin A in health and disease, which began as a graduate student at Tulane University's School of Public Health and Tropical Medicine. Vitamin A is considered an essential nutrient, as the term "vitamin" implies. However, increasing evidence implicates it contributing to the causation of many chronic diseases and to certain acute infectious diseases, associated with reduced risks in people of African descent; that is, health disparities in the "reverse" direction.

During their life cycle, malaria parasites spend over a week in the liver, where over 90% of vitamin A in the body is stored. Malaria parasites selectively absorb vitamin A from the human host, and people of sub-Saharan African descent are at reduced risk of severe malaria compared to people of European descent. In 2013 I proposed the hypothesis that, upon emerging from the liver the merozoite-stage parasites, packed with vitamin A, use it as a membrane destabilizer to invade and reproduce in the red blood cells. On the death of these cells in very large numbers the vitamin A is released into the circulation in toxic concentrations, inducing the signs

and symptoms of the disease. This hypothesis was supported by a study published in 2019.¹ The study confirmed previous findings of low serum retinol in patients with malaria. However, it also reported for the first time that concentrations of retinoic acid, the major biologically active and potentially toxic metabolite of vitamin A, were significantly increased.

How are people of sub-Saharan Africa protected against death from malaria? The foregoing led me to conceive the idea that this protection has resulted from physiological adaptations to the impact of endemic malaria parasitism, related to vitamin A, evolving over the course of millennia. The hypothesized adaptations included: 1) sequestering vitamin A away from the liver to other organs, thereby reducing access to its use by the invaders in causing disease; and 2) slowing the metabolism and breakdown of vitamin A, causing it to accumulate in the body to sub-toxic levels that weakened the parasites and thereby reduced the risk of severe disease.

These adaptations may have served to protect against severe malaria and some other diseases in the ancestral environment. However, they may also have simultaneously increased risks of chronic diseases for people of West Central African descent exposed to the typical dairy-based US diet, which is high in preformed vitamin A. These adaptations are hypothesized to increase susceptibility to chronic inflammatory diseases in which retinoids may play a causative role, notably atopy, vascular diseases, diabetes, and cancer, as well as genetic diseases such as sickle cell anemia and glucose-6-phosphate dehydrogenase deficiency. In short, the same physiological adaptations that are protective against tropical diseases, coupled with exposure to a dairy-based diet, may be contributing to persisting health differences in African Americans.

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I also thank my graduate students in public health at Jackson State University, among whom Juliet Enow, Veronica Hernandez, and Kendrick Walker in particular provided valuable critical comments and help in gathering data for this essay on health disparities; and also Robyn Howard, for preparing the graphics.

I am especially grateful to Dr. Omar Bagasra, whom I met at a conference on zika, for inviting me to present an early version of the proposed theory of health disparities at the South Carolina Center for Biotechnology, Claflin University, in March 2017. I also thank participants in the Distinguished Seminar Series, Research Centers in Minority Institutions and Center for Environmental Health (RCMI-CEH), Jackson State University, March 29, 2017, for their valuable critical comments on my presentation, "Toward a theory of health disparities affecting people of sub-Saharan African descent."

Finally, I thank my wife Carol Andersen for preparing the Index, for her editorial skills and overall support, and for our two lovely daughters since 2001. My sister Lucy Kinna, also kindly assisted in editing the manuscript for publication.

CHAPTER ONE

GENERAL INTRODUCTION

Human populations have adapted to diverse aspects of their environment, such as climate and pathogens, leaving signatures of genetic variation.^{1,2} This well-established principle may apply to African Americans – people of mainly West Central African descent – who are at increased risk of many vascular, metabolic, malignant, and allergic diseases compared to their European counterparts. In 2021 there were an estimated 40.1 million people self-identifying as non-Hispanic black or African American, representing 12.1 percent of the total United States population of 331.9 million. Blacks/African Americans are the second largest minority population in the United States, after the Hispanic/Latino population. Concentrated primarily in the southern states, African Americans are projected to comprise 15% (60.7 million) by 2060.³

The prevailing view of health disparities affecting African Americans – inequities in health care access, quality, and health outcomes – is that they result from many factors, particularly social, economic and environmental. Racial inequities are estimated to cost the nation a total of \$135 billion per year, including \$93 billion in excess medical costs and \$42 billion in lost productivity.⁴ Disproportionately poor health outcomes – in infant mortality, life expectancy and the prevalence of chronic disease—reveal differences by race and ethnicity, independent of other factors, that are widely attributed to health system, educational, residential, and other forms of discrimination.⁵

“In the past few years, amid a pandemic [COVID-19] that has taken a disproportionate toll on Black and brown people and a national reckoning on racial justice, leaders from several U.S. health systems have named racism as a public health threat and pledged to identify and reverse racist policies and practices in their institutions. As an example, in an open letter published last Juneteenth, leaders from 36 Chicago hospitals said it’s ‘time for action’ and promised to ‘double down’ on efforts to reduce racial health disparities among their patients, create more equitable workplaces for their employees, and invest in communities of color where many of their patients and staff live ... In several institutions, this work has been spurred by medical students or residents demanding change.”⁶

Certain observations on the health of African Americans across the age span are not readily explained by structural racism, discrimination, or other social factors. For instance, it is not widely known that people originally from sub-Saharan Africa are at reduced risk of death from other diseases, notably malaria, dengue hemorrhagic fever, yellow fever, and nonalcoholic fatty liver disease. These mostly infectious diseases are prevalent in tropical West Central Africa, suggesting genetic links between the natural environment and endemic disease and hinting at a role for nonsocial factors in the explanation of health disparities.

Here the case is made that disparities in *health outcomes* affecting African Americans are partly the result of physiological adaptations to the vectors of major infectious diseases in the ancestral environment of sub-Saharan Africa, occurring on an evolutionary timescale, coupled with the impact of the natural and dairy-based nutritional environment of North America. The sub-Saharan African environment has involved continuous exposure to potentially lethal tropical diseases, a major one being *Plasmodium falciparum* malaria, the most dangerous form of the disease. Together with other parasitic organisms, *Plasmodium falciparum* selectively absorbs vitamin A from the human host and uses it in reproduction and disease pathogenesis.

Vitamin A is a major constituent of the dairy-and egg-based diet of North America. Less well known is that vitamin A is a potentially toxic fat-soluble molecule that is stored mainly in the liver as retinyl esters, and it contributes importantly to many acute and chronic conditions and diseases, including parasitic diseases and diabetes.

On the proposed theory, inherited physiological characteristics acquired over millennia from evolutionary adaptations to endemic tropical diseases – including malaria, schistosomiasis, and onchocerciasis – render people of West Central African descent particularly sensitive to vitamin A as a toxin. The proposed evolutionary adaptations were 1) sequestering vitamin A away from the liver, where it is mainly stored, to other organs, to reduce accessibility to *Plasmodia*; and 2) slowing the metabolism and catabolism of vitamin A, causing it to accumulate to subtoxic concentration, thereby weakening the parasite and serving to reduce the risk of severe disease.

These adaptations may have helped to enhance survival in the original African environment. However, in the North American environment, characterized by the absence of vitamin A-absorbing parasites and by a dairy-based diet that is rich in vitamin A, derived from the historic dependence of people of northern European descent on grazing animals, these adaptations are hypothesized to cause vitamin A to accumulate, rendering people of West Central African descent sensitive to vitamin A as

a cellular toxin and at greater risk of the same chronic diseases that affect European Americans. The increased concentration of vitamin A in organs outside the liver, coupled with its excess overall accumulation, are proposed to explain the susceptibility of African Americans to organ-specific diseases that were once rare in the original environment and are increasingly linked to vitamin A toxicity via mitochondrial dysfunction and apoptosis.

On this theory, people of West Central African descent are more sensitive to vitamin A as a toxin than people of European descent, and disparities in chronic diseases are partly due to excess expression of the same retinoid-related mechanisms that are responsible for these diseases in general. Among its testable implications, the vitamin A toxicity hypothesis suggests that adoption of traditional or modified West Central African-style diets that are low in vitamin A and high in vitamin A-absorbing fiber could help to prevent and treat these diseases. Subject to testing, they could also serve as a population-based strategy for maintaining overall health and increasing longevity.

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CHAPTER TWO

HEALTH DISPARITIES AFFECTING AFRICAN AMERICANS

An estimated 44 million Americans of African ancestry are now living in the United States, mostly from forced relocation over preceding centuries. The countries of origin were in West and Central Africa and included the Congo and Angola (39%), Togo, Benin, and Western Nigeria (20%), East Nigeria (15%), and the Cameroons, Equatorial Guinea, and Gabon (15%).¹ (Figure 1). For reasons that are not fully understood, African Americans, comprising 13% of the US population, are at greater risk of disease and/or death than Americans of European descent from the following:

- Cancers of the blood and bone marrow, breast, lung, ovary, prostate, colon, and rectum;^{2,3}
- Metabolic and cardiovascular diseases including hypertension, type 2 diabetes, kidney disease, obesity, gout, congestive heart failure, and stroke;^{4,5}
- Eczema;⁶⁻⁸ asthma;⁹ and allergies to eggs and milk.^{10,11}

These conditions were once rare in African countries themselves.¹² Yet today the prevalence of hypertension in African Americans is over 40%, which is among the highest in the world. It also occurs earlier in life and is of greater severity than among other groups.¹³ African Americans are also at risk of certain genetic diseases and conditions that include sickle cell anemia – the most common blood disorder in the US – and glucose-6-phosphate dehydrogenase deficiency (G6PDd), which is found almost exclusively among males from malaria-endemic regions.¹⁴

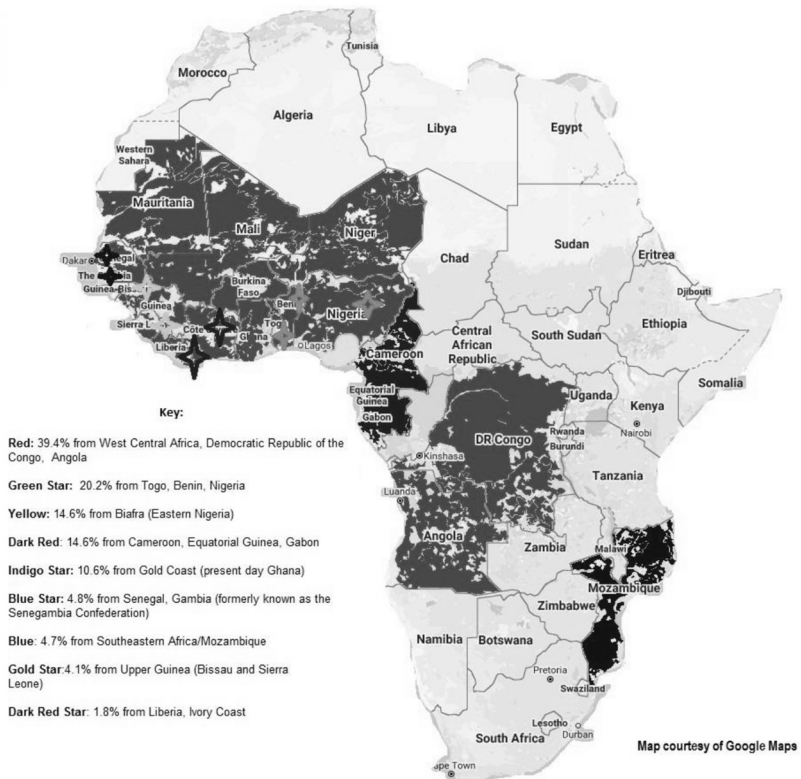
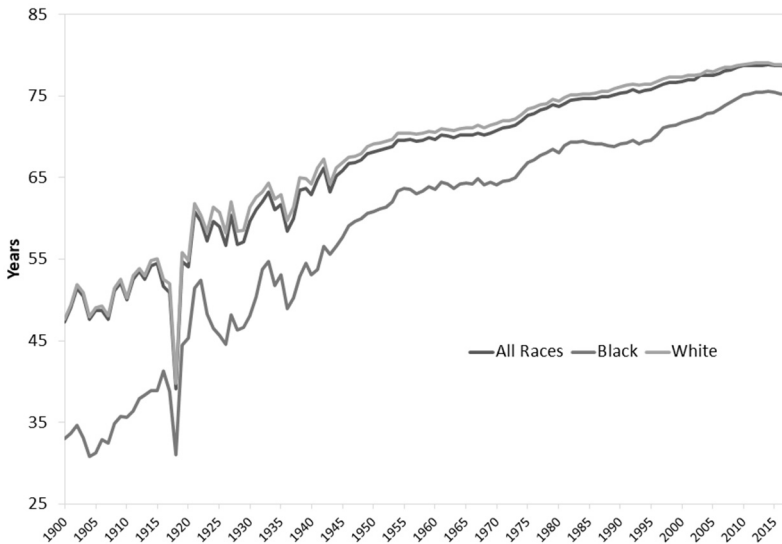


Figure 1. Ancestral origins of African Americans

Life expectancy is also shorter for African Americans compared to European Americans. Although it increased for both males and females from 1980–2014, it was 6.9 years longer for European American than for African American males and 5.6 years longer for European American females than for African American females.¹⁵



■ FEDERAL RESERVE BANK OF ST. LOUIS

<https://www.stlouisfed.org/on-the-economy/2022/january/evolution-racial-gap-us-life-expectancy>

SOURCES: NCHS, National Vital Statistics System and authors' calculations.

NOTES: Data end in 2017. For 1900 to 1969, Black life expectancy is based on data for the nonwhite population. From 1970 onward, the Black life expectancy is based on data for the Black or African American population. Both white and Black definitions include people of Hispanic and non-Hispanic origin.

The infant mortality rate for African American infants (deaths in infants up to 12 months of age per 1,000 live births) is more than twice that of European American infants, due to higher rates of stillbirth, birth defects, low birth weight, premature birth, and Sudden Infant Death Syndrome.¹⁶⁻¹⁸ Rates of maternal mortality among African Americans are also close to three times higher than those of women of European descent.¹⁹

Infant gestational age, an important predictor of death and illness in infants, differs among racial and ethnic groups. Among the five groups measured in the National Vital Statistics Survey (NVSS) in 2014, African American women had the highest percentage of preterm singleton births at 11.1 percent, while Asian or Pacific Islander women had the lowest at 6.8 percent (NCHS, 2016).²⁰

Death rates from cancer, diabetes and heart disease in sub-Saharan Africa are expected to exceed those from infectious diseases by 2030. As many as 46% of Africans suffer from high blood pressure, the highest rate

worldwide, and a growing percentage of people are overweight.²¹ Even in the mid-1990s the prevalence of diabetes exceeded that of the white population in Durban, South Africa.²² Yet less than a century earlier coronary heart disease was rare in many parts of Africa,²³ as it was in western populations before World War I.²⁴ Cancer rates in Africa were also low.²⁵

Changes in diet may have contributed to these statistics. Many countries in sub-Saharan Africa are adopting 'western' diets and are experiencing high rates of overweight and death from chronic diseases.²⁶

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Notes

Further notes on health disparities: compared to European Americans, African Americans

- Are at higher risk for heart diseases, stroke, cancer, asthma, influenza and pneumonia, diabetes, and HIV/AIDS (1).
- Are more likely to die at younger ages from all causes (2), including diabetes and stroke (3).
- Have a three times higher rate of kidney failure (4) and account for a third of all patients in the U.S. receiving dialysis for kidney failure, but make up 12.7% of the U.S. population.
- Have the highest death rate and shortest survival from all cancers (5).
- Experienced more serious illness and death from COVID-19 during the 2021-2022 pandemic (6).

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CHAPTER THREE

STRUCTURAL RACISM AND DISCRIMINATION

Health disparities are differences in disease incidence, prevalence, mortality, morbidity (complications), survivorship, including quality of life after treatment, burden of disease, and stage of disease at diagnosis.¹ Disparities in health and health care affecting African Americans are generally attributed to structural racism, which has created limited access to healthcare, poorer quality of healthcare, education, and housing, as well as stressful experiences related to racial discrimination, and poorer overall incomes. The continuing existence of discrimination, stress, and poorer healthcare is well documented.²⁻⁴

According to Williams et al.,⁵ socioeconomic factors alone do not account for racial and ethnic inequities in health. Racism itself is a fundamental cause of adverse health outcomes for racial and ethnic minorities. For each of the primary domains of racism affecting mental and physical health outcomes – structural racism, cultural racism, and individual-level discrimination – Williams et al. present data and suggest priorities for future research to advance knowledge in the area.

The legacy of 400 years of slavery, violent oppression, discrimination, and emotional stress suffered by African Americans and the struggle both to receive and to provide health care, continues to have pervasive effects.⁶⁻⁸ The COVID-19 pandemic in 2020-2022 has also disproportionately affected African Americans. Across the United States, COVID-19 mortality rates in predominantly black counties have been sixfold higher than in predominantly white counties.⁹

African Americans are also at increased risk of diabetes and its complications after controlling statistically for socioeconomic status.¹⁰ Non-Hispanic black women have rates of preterm birth that are 40% higher than those of Hispanic and non-Hispanic white women after adjustment for maternal socioeconomic status and education.¹¹ Chronic worry about experiencing racial discrimination is reported to affect risks of premature birth among African American women, possibly through neuroendocrine, vascular or immune mechanisms involved in responses both to stress and the initiation of labor.¹² Such worry has been suggested to explain the

puzzling observation of greater disparities in preterm birth among more socioeconomically-advantaged women.¹³

While disparities in access to and the quality of health care affecting African Americans remain rooted in structural racism and discrimination, the overall health profile of people of West Central African descent – showing both increased and reduced rates of death from different diseases – suggests a role for additional factors. For instance, in a 25-year follow-up study on the health of medical graduates, Black compared to White physicians had a higher risk of cardiovascular disease, with an earlier onset, twofold higher rates of diabetes and hypertension, a 40% higher rate of coronary artery disease, and a much higher case fatality rate of 52% vs. 9%.¹⁴ Similarly, in a diverse cohort of 927 youth with type 1 diabetes, African Americans compared to other groups had a higher glycated hemoglobin (HbA1c) level after adjustment for many factors, higher rates of severe hypoglycemia and ketoacidosis, higher required doses of insulin per body weight, and a higher rate of hypertension. African American participants also had less residual β -cell function, which likely contributed to a worsened prognosis for type 1 diabetes.¹⁵ Higher HbA1c levels are also reported in African American children with type 1 diabetes compared to white children, after adjustment for socioeconomic status, diabetes duration, and insulin dose.^{16,17} After being diagnosed with type 1 diabetes, African American youth have unexplained adverse outcomes of earlier onset and prognosis for long-term complications.

A study of hypertension among West Africans in Africa and their descendants reported a stepwise increase in hypertension as groups moved from rural to urban Africa to the Caribbean, and from the Caribbean to the US. People of African descent in the US had hypertension levels that were twice as high as those in Africa, and higher than among mainly white populations in Europe.¹⁸

Another study comparing hypertension rates in West Africa, the Caribbean and the US, based on 10,014 individuals in seven locations and using standardized data collection methods, reported a consistent gradient of hypertension prevalence, rising from 16% in West Africa to 26% in the Caribbean and to 33% in the US. Mean blood pressures were similar among those aged 25 to 34 but the increase in rates of hypertension with age was twice as steep in the US as in Africa.¹⁹

Age-specific mortality rates from chronic diseases were often several-fold higher in younger black adult age-groups overall than in most high-income countries. Even more surprising, age-specific mortality rates from chronic diseases are higher in sub-Saharan Africa than in virtually all other regions of the world, both in men and women.²⁰

These findings pose the following questions: How does the North American environment contribute to increased rates of chronic disease in all population groups that have been studied? Why are African Americans more susceptible to these diseases than European Americans? And why are chronic disease rates rising rapidly in Africa when they have historically been low? In short, why were Africans healthy in their original environment but unhealthier than Europeans in North America, and increasingly in Africa itself? In what way were Africans well-adapted health-wise in the West Central African environment, but fare less well in the United States? The thesis presented here suggests answers to these questions.

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Notes

A summary of “Systemic Racism and Health Disparities: A Statement from the Editors of Family Medicine Journals”. Sumi M. Sexton, MD, American Family Physician and other editors. Published ahead of print, October 15, 2020: https://www.aafp.org/pubs/afp/content/Racism_statement.html

The year 2020 was marked by historic protests following the deaths of George Floyd, Ahmaud Arbery, Breonna Taylor, and many other Black people. These protests heightened awareness of racism as a public health crisis, its profound adverse effects on physical and mental health, (1,2) and on negative patient experiences in the healthcare system.(3,4) They noted that race is a sociopolitical construct that continues to disadvantage Black and other People of Color, (5-8) and to affirm that family medicine is a specialty that has emerged as a counterculture to reform mainstream medicine, (9) – both to confront systemic racism and eliminate health disparities – “so we can better serve our patients and our communities.”

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Professor Paula Lantz, University of Michigan, writes that growing numbers of states have banned the teaching of Critical Race Theory (CRT) and other frameworks about structural racism in K-12 schools. Her view is that the scientific tenets that underlie CRT are crucial for understanding and addressing racial inequality in important social outcomes, including population health; and CRT provides a framework for understanding racial inequality, supported by decades of multidisciplinary scholarship. The tenets of CRT are essential in seeking to address racial inequalities in all social outcomes, and that “there can be no objective or effective teaching, research, or practice addressing the large racial health disparities (e.g., in life expectancy, infant/maternal mortality, and almost every type of illness or injury) that persist in the U.S. that does not implicitly or explicitly incorporate the main tenets of CRT.”

Regarding population health science, these tenets are: 1) Race is a social construction in the way that race is defined and experienced and is the result of social and political thought and actions that change over time. “Race” is not a biological imperative driving disease processes and longevity, but rather a marker for how individuals experience physical and social exposures, including discrimination, that influence health over the life course. 2) Racism and its outcomes are perpetuated through social processes above and beyond individual actions through cultural norms, institutional rules, and laws and regulations. Racism is not primarily person to person, but rather the processes built into institutions, systems, and policies that reinforce, codify, and perpetuate exposures and opportunities that differ across socioeconomic and racial groups. 3) Because racism is commonplace, understanding structural racism within our institutions and policies – on education, income, housing, food, criminal justice, the environment, and health care – are key for addressing population health inequities. 4) Listening to and understanding the lived experiences of

individuals is essential for understanding how racism works to create inequities in individual outcomes, including health, and that there is a need to better represent in research, the media, and policy reform work, how racism in all its manifestations is experienced by people in ways that matter, including for physical/mental health. Lantz concludes that those committed to health equity through population health science should publicly defend the tenets of CRT and their long-standing contributions to population health in teaching, research, community-based efforts and policy reform, to improve health and well-being for all. (Professor Lantz defends Critical Race Theory, May 2022 <https://fordschool.umich.edu/news/2022/lantz-defends-critical-race-theory>)

CHAPTER FOUR

OTHER CLUES TO EXPLAINING HEALTH DISPARITIES

Health Disparities in the Reverse Direction

A major clue in explaining the origin and causes of health disparities affecting African Americans is that disparities also exist in the reverse direction; that is, people of West Central African descent are protected from severe disease and less likely than European Americans to die from malaria, yellow fever, and dengue hemorrhagic fever.¹⁻⁴ In earlier centuries, African workers were highly prized in the Caribbean because they were less likely than Europeans to succumb to these diseases, all of which were highly prevalent and lethal in the region at the time.⁵ African Americans are also at lower risk of non-alcoholic fatty liver disease (NAFLD),^{6,7} which is defined as hepatic fat accumulation (steatosis) >5% of total weight of the liver and affects >30% or more of older European Americans.⁸ African Americans have less hepatic lipid content, lower plasma lipoprotein lipase, and less overall liver disease.⁹ NAFLD is a growing global epidemic, affecting millions of people worldwide. The burden of NAFLD is widely underestimated.¹⁰ In the US, Hispanics are the most disproportionately affected whereas African Americans as a group are least affected by hepatic steatosis and elevated aminotransferase enzyme levels. Considering the vital role of the liver in metabolism and in overall health, the anomaly of lower rates of NAFLD in African Americans suggests an underlying biological process or mechanism related to the liver, which potentially explains the pattern of health disparities affecting African Americans.

African Americans also have the lowest risk of osteoporosis of any racial group at younger ages, as well as higher bone densities and lower bone turnover rates,¹¹ (p. 71) and have half the nonvertebral fracture rate of European Americans.^{12,13} However, when hip fractures do occur, African American women are more likely to die than European American women; they also have longer hospital stays and are less likely to be ambulatory at discharge.¹⁴

The thesis proposed here is that differences in health outcomes affecting African Americans, both negative and positive, are due in part to evolutionary physiological adaptations to the ancestral environment of West Central Africa. This environment was and remains broadly characterized by exposure to potentially lethal parasitic (protozoan and helminthic) organisms, most notably those causing malaria, schistosomiasis, and onchocerciasis.¹⁵ It is suggested that these adaptations were successful in enhancing survival in the original ancestral environment, but they are potentially disadvantageous in the environment of North America, as explained below.

About 3.3 billion people are at risk of malaria due to *Plasmodium falciparum*, the most dangerous malaria parasite, making it one of the world's most significant health problems.¹⁶ There were 214 million new cases of malaria worldwide in 2015, with Africa accounting for most (88%), and an estimated 438,000 deaths from malaria worldwide, mostly in Africa (90%) and in children under age 6, i.e., about 0.2% of the 214 million cases, indicating the comparative rarity of lethal outcomes.¹⁷

Schistosoma mansoni, a waterborne flatworm trematode parasite known as a schistosome, is the leading cause of schistosomiasis and the second most common human parasitic disease after malaria. It is found in areas with water contaminated with freshwater snails, the intermediate host for the parasites. The disease causes liver and intestinal damage and the worms can live in the bloodstream for decades, even if the host has a healthy immune system. In 2016, 207 million people in 75 countries had the disease and about 200,000 people, mostly in Africa, died from it.¹⁸

Onchocerca volvulus, also known as “river blindness”, is a filarial (arthropod-borne) nematode (roundworm) that causes onchocerciasis, the second leading infectious cause of blindness worldwide after trachoma. The disease is transmitted by repeated bites from blackflies that live near fast-flowing streams and rivers in remote rural areas. In addition to causing visual impairment that can progress to blindness, onchocerciasis is also associated with severe skin disease, including intense itching, skin atrophy, rashes, or nodules under the skin. About 18 million people are affected by the disease.¹⁹

Environmental Exposure to Vitamin A-Ingesting Pathogens

A second major clue to explaining the health profile of people of West Central Africa is that both *P. falciparum*²⁰ and *Onchocerciasis volvulus*²¹ selectively absorb vitamin A from the human host. Mizuno et al.