
Senegal may be underdeveloped, but we live well...I guess if we had no centenarians, then we might entertain the thought that we too need an overly sanitized [medicalized], American type of life.

—Mr. Seck, a resident of Thiaroye, a suburb of Dakar, 2000 (2)

With this comment made by Mr. Seck whose daughter suffers from sickle cell disease, Duana Fullwiley captures one of several contradictions discussed in her book, *The Enculturated Gene*. Senegal may be “underdeveloped”—reflected in the frustration of those with sickle cell whose government cannot provide adequate health care for their condition—nonetheless people’s experiences of the disease support a Senegalese exceptionalism, consisting of social and cultural practices that enable them to survive, if not exactly to thrive. This may sound like a rationalization of unequal global health care—they don’t need high-tech measures as they are managing without them—and, to some extent, it is. Yet Mr. Seck observes that some people in Senegal do live to be centenarians, suggesting that they are not without the means for addressing health problems. Indeed, his point about “an overly sanitized [medicalized] American type of life” also suggests that simply bringing treatments and equipment used in US hospitals to treat sickle cell patients in Senegal might not be entirely appropriate or welcomed.

Certainly, it would be helpful to have some equipment, such as that used for performing electrophoresis (24), which would enable physicians/specialists to distinguish homozygous sickle cell disease (those with two...
alleles for sickle cell—a dominant trait; HbSS) from heterozygous sickle cell disease (those with one allele for sickle cell—a recessive trait; HbAS). Such genetic information might help women and men contemplating marriage or deciding to have children to assess the risk of giving birth to a child with sickle cell disease, associated with the dominant HbSS.¹ In the absence of such tests, Senegalese women and men who develop the disease and for whom children are vitally important for their social identities, marriages, and kin relations, might object to the primary treatment for sickle cell disease in the US, which consists of a form of chemotherapy known as hydroxyurea. For them, this therapy is particularly problematic as it causes temporary, and possibly permanent, infertility (36).²

Thus, having genetic knowledge may not provide a solution to a particular health problem and, as Lock (2009) notes regarding genetic test results of family members with histories of Alzheimer’s disease, people may not remember or care about this knowledge, particularly when a disease’s etiology is murky and it has no known cure. In the case of Alzheimer’s disease, people may add this bit of information about whether they carry the APOE4 gene to a wider set of beliefs about the disease—that its emergence depends on diet, exercise, mental “exercise,” and sociality as well as hereditary propensity (Lock 2009:168). In fact, the exact relationship with having a specific genetic marker, in this case, the APOE4 gene, and developing the neurological symptoms and range of behaviors associated with Alzheimer’s Disease, is unclear.³ Nonetheless, this lack of exact correspondence does not mean that individuals will not opt for genetic testing as it becomes more widely available and, in some cases (such as breast cancer), act upon this knowledge (Lock 1998). What interests Fullwiley about the possibilities of genetic testing for sickle cell is the range of cultural interpretations that genetic knowledge about this condition has produced—which have varied widely depending on time and place as well as on political and social hierarchies based on race, class, and national identities. She uses the phrase, “the enculturated gene,” to refer to the ways that Senegalese women and men diagnosed with sickle cell trait or disease, along with Senegalese specialists, have interpreted genetic information about sickle cell.

The genetic information on which these interpretations are based derived from the discovery made in 1978 by two US geneticists, Yuët Wai Kan and Andrée M. Dozy (178). They found a type of genetic variation, consisting of differently sized fragments referred to as Restriction
Fragment Length Polymorphisms (RFLPs), which were adjacent to the beta globin gene associated with sickle cell. Kan and Dozy suggested that specific genetic variants of RFLPs (known as haplotypes) could be used as the basis for new studies of sickle cell populations, since they found that certain variants were consistently present in “people of African origin and not detected in Asians or Caucasians” (Kan and Dozy 1978 as cited in Fullwiley 2011:178). Their suggestion that the study of these haplotype variations should be undertaken in Africa intrigued some geneticists who subsequently pursued questions about population genetics distribution.

Fullwiley focuses on a 1984 study conducted and led by the French genetic researcher, Dominique Labie, who compared sickle cell haplotype markers in Senegal, Benin, and the Central African Republic. Labie and her colleagues concluded that a particular haplotype sequence found in those with sickle cell disease in Dakar, Senegal possibly contributed to the production of a type of hemoglobin, known as fetal hemoglobin (HbF). While hesitant to draw a direct connection between this haplotype variant and HbF, some sickle cell specialists pondered the possibility that the presence of HbF provided some protection from painful sickle cell crises in adults. This association led to the subsequent characterization of Senegalese sickle cell as a relatively “mild” form of the disease, even while the researchers involved in this study took pains to discourage such a direct causal connection.

Yet the fact that many people in Senegal have taken up the idea that there is a distinctive form of Senegalese sickle cell and have interpreted the 1984 haplotype study findings which suggested that a “mild form” of Senegalese sickle cell exists reflects a particular local interpretation of genetic information. In the context of reduced levels of healthcare associated with neoliberal economic reforms of the 1980s, where treatment consists of folic acid tablets and, when needed, acetaminophen (an analgesic), genetic evidence suggesting that Senegalese sickle cell is associated with the continued production of fetal hemoglobin is reassuring. The presence of fetal hemoglobin in adults is believed to reduce the severity of the excruciatingly painful, periodic crises associated with sickle cell disease that are caused by the bunching up of sickle cells, particularly in joints. The presence of sickle cell also reinforces ideas about traditional medicines, such as fagara (Fagara xanthoxyloïdes), a plant widely used for treatment, which some consider to be more effective than most Western (“modern”) medicines (85). Indeed, after being treated by the renowned
healer Gaoussou Sambou, two sisters attributed an increase in “F” cells (HbF) in their blood, documented by electrophoresis, to medicines that consisted mainly of fagara. Yet fagara has not been conclusively shown in clinical tests to increase HbF. This situation puts Senegalese sickle cell specialists in the awkward position of being optimistic about the possibilities of fagara in the production of juvenile hemoglobin while also being hesitant about their comparison with “traditionalists” by their European colleagues who are skeptical of such low-tech, herbal remedies. In such circumstances, people make do, relying on both specialists’ promotion of “mild sickle cell” and on healers’ use of herbal medicines, and, of equal importance, on various forms of kin and association support to get by.

Stacey Langwick (2011) observes that healers and medical professionals in postcolonial places such as Tanzania or Senegal cannot help but be influenced by Western biomedicine to which they are exposed through medical education and government training programs, in hospitals and in clinics, as well as through radio and television broadcasts. Nonetheless they often interpret biomedicine in culturally specific ways. In the case of sickle cell in Senegal, because diagnosis has primarily been based on the serological identification of sickle hemoglobin in the blood, many people—perhaps ten percent of the population identify themselves as drépano (sicklers) when they have a single recessive gene, HbAS—and hence, the sickle cell trait. Younger Senegalese sickle cell specialists do not consider this to be a serious health condition since it does not express itself as full-blown sickle cell disease (HbSS). Yet their patients describe symptoms such as headache and fever which they identify as sickle cell, even when these conditions may indicate something else, such as malaria. Fullwiley argues that these patients’ understandings of sickle cell, and its possible psychosomatic expressions, reflect connections made between individual bodies and the body politic. For them, bodily illness reflects the state of the Senegalese economy—brought low by the implementation of a structural adjustment program and loan repayments which have impoverished many ordinary Senegalese citizens. Even with increased electrophoresis testing for genotypes, people with the sickle cell trait also want to receive treatment for their perceived health problems.

Consequently, women and men who see themselves as having sickle cell rely on practices that reflect local understandings of the body, illness, and cure(s) in which the social relationships of patients—with health care providers (both sickle cell specialists and traditional healers), a range of
family members, and those who have sickle cell—are seen as valuable substitutes for prohibitively expensive biomedical treatments. Indeed, the health benefits attributed to extended social networks of families and associations are reminiscent of those made in Durkheim’s (1951) study of suicide in Europe. Here, he argues that suicide is more common in societies where individualism is stressed, as opposed to those where extensive family, religious, and associational connections are maintained. Fullwiley’s assessment of Senegalese women and men with sickle cell who have formed the Association sénégalaise de drépanocytose (ASD) as a source of mutual support and as a way to put political pressure on the Senegalese state to fund sickle cell treatment agrees with Durkheim’s findings.4

Thus Fullwiley considers a constellation of practices and things, not only genotypes, as contributing to the experience of sickle cell in Senegal: “These aspects of life itself integrate genes, poverty, hope, religious faith, and constraints in care, as well as autochthonous plants, scientific aspirations, and human survival strategies that cannot be parsed as separate” (12). And while the complexity of specific workings of haplotypes in relation to the varied expressions of sickle cell underscore the many unknown aspects of genetic research on this disease, these unanswered questions which frustrate researchers, may be differently interpreted: “Many people I met in Dakar viewed “holes” in science…as spaces where possibilities could happen” (29).

Genetic research on sickle cell haplotypes and the conclusion that there is a national—Senegalese—haplotype also reflects earlier research projects in which researchers sought to associate levels of sickle cell hemoglobin with particular races, conceptualized as separate races distinguished by ethnic differences in Senegal as well as with distinct racial groups in the United States. This desire to identify and classify disease according to specific genotypical or phenotypical traits underscores the underlying, and often unacknowledged, political and economic bases of these processes. Thus in the 1950s, French colonial medical officers sought to associate sickle cell hemoglobin levels with particular races/ethnic groups (see 166, Figure 5.1). Although they were ultimately unsuccessful, this counter-evidence was construed as a “lack of ethnic purity” due to research conducted “too late.” Such efforts to differentiate and distinctly map people onto particular places reflected a variation on the strategy of divide and conquer.5 And while French officials were more concerned with classifying Africans serotypically by race and ethnicity, their
finding that lighter-skinned Peul (Fulani)-speaking people [had] equal [or higher] level of sickle cell hemoglobin as compared with darker-skinned Wolof speakers was considered surprising (65).\textsuperscript{6} In other words, ideas about racial hierarchy came into their evaluations of disease as well.

Indeed, in the US context, sickle cell represents a classic case of the association of racial categories and disease. Sickle cell disease was viewed as a disease “of Negro blood” (Wailoo 2003:236), despite counter-evidence that sickle cell hemoglobin was also found in people classified as “white.” Sickle cell was construed as an indication of black ancestry—defined by the “one drop” rule (x). The seeming arbitrariness of these classifications of disease in the US, which nonetheless effectively reinforced racial stereotypes, may be seen in other diseases as well. If sickle cell has been characterized as a black disease by US medical researchers since the 1940s, poliomyelitis (or infantile paralysis) was conceptualized as a white one. In her study of African Americans’ experience of polio, Naomi Rogers (2007:786), notes that polio experts in the 1920s viewed the predominance of confirmed cases of polio among white children as evidence of “the complex and delicate bodies of the ‘civilized’ peoples of Northern European heritage,” while black children were considered to be less susceptible and few were documented with polio. This assessment not only coincided with prevailing mainstream views about race but also justified the lack of medical care as blacks had fewer cases and hence did not need treatment. As was the case with the inconvenient evidence of phenotypically white Europeans with sickle cell hemoglobin contradicting the classification of disease by race, the fact that white southern children, like many black southern children, lived without access to centralized, filtered water and hence were often exposed to the wild poliovirus as infants and had low levels of polio paralysis, was ignored.

More recently, racial difference based on genetic testing has shifted from phenotypic classifications to genotypic ones. Now, when everyone is at risk for one disease or another, racial distinctions continue to be made in several ways. First, knowing one’s risks enables the state to reduce its responsibility for health care, by holding individuals accountable for their own health, which may be easier for white or black Americans living in the suburbs than for those living in the inner city. Second, individuals of African descent who, statistically, may have a greater genetic risk for particular diseases such as sickle cell, may pressure the state for approval of race-specific drugs. Similarly, pressure on the federal government for
race- or ethnically-based funding for biomedical and genetic research reinforces ideas about racial difference (El Haj 2007:293-94).

Fullwiley’s remarkable study of the “enculturated” sickle cell gene suggests how access to genetic information and its distinctive interpretations in Senegal may be similarly fraught. This is not to say that biogenetic research does not have health benefits or that it is inherently flawed. Rather, studies such as those of Fullwiley, Wailoo (2003), and Rogers (2007) demonstrate the ways that political, economic, social, cultural, and historical factors influence just how research results are interpreted. In the case of genetic research, such interpretations are further influenced by the complex technologies and associated techniques used for identifying genetic material such as haplotypes. Gell’s (1992) observations in the context of art are also relevant here, in that technology, such as the complicated equipment used for assessing genetic material, may astound those—most of us—who do not understand how it works. Gell argues that such technologies “enchant” us with their unfathomable mystery, predisposing the recipients of genetic test results to accept them as fact. Furthermore, genetics research benefits not only from the allure of technology but also from its scientific precision in ostensibly predicting the future, which supports the hope that a specific cause of disease can be identified and addressed. This particular form of explanation and its potential for curing disease is especially attractive as it isolates geneticists working in laboratories from the messy, imprecise aspects of social life.

Yet it is this messiness—reflected in marital disputes, government graft, and inequities in international research, as well as memories of particular colonial pasts and in contemporary experience of global economic inequality—which also contributes to explanations of how diseases are identified, treated, and cured. Indeed, the skepticism of European knowledge concerning sickle cell and the valorization of a Senegalese way of life—extended family networks, occupational associations, religious practices, and historical knowledge of local medicinal plants as voiced by Mr. Seck at the beginning of this essay are part of these messy dynamics.

Duana Fullwiley has produced an extraordinary work that incorporates the insights of anthropology as well as science and technology studies of genetics and race. It is also exceptional in its multi-sited focus on Senegal and France, since many similar studies of genetics have tended to focus on the US and Europe. While there is some repetitious overlap between the different chapters, the richness of the fieldwork and the depth
of Fullwiley’s knowledge about sickle cell, from both the perspectives of biomedical-genetic sickle cell specialists and researchers in Senegal and France, and from the perspectives of sickle cell patients in Senegal, enables readers not only to understand but to also:

take...seriously how scientists themselves construct and put into place reductionistic, and at times racializing, genetic categories and therefore have a hand in eliding the biological outcomes they observe and the genetic distinction in bodies that may or may not be meaningful. (35)

Endnotes:

1 In June 2010, the Senegalese National Assembly announced a new prenuptial-testing law that would require those intending to marry to have blood tests to test for sickle cell (260). Nonetheless, Fullwiley (82) notes the refusal of one man to this prenuptial testing, who referred to it as an “affair u tubaab la” (That is, something that Whites do, not us).

2 Alternately, transfusion therapy requires routine blood transfusions along with iron chelation therapy to reduce the excess iron that transfusions entail.

3 Fullwiley makes a similar point with regard to fetal hemoglobin (15).

4 Durkheim’s (1951) study is also instructive as an early example of efforts to map associations of disease and place. The map of suicide numbers by arrondissements in France (1887-1891) suggests later mappings of race/ethnicity and place were used in an attempt to link sickle cell with particular groups in French West Africa (166, Figure 5.1).

5 In the case of sickle cell in Senegal, colonial officials sought to categorize human groups as “distinct” based on serological difference, while later researchers categorized “sample populations’ genes as naturally linked to their assumed geographical differences,” in this case a national identity (36).

A related but different example of mapping genetic differences and disease may be seen in the genomic sequences in poliovirus identification. As part of the Global Polio Eradication Initiative, certain genomic sequences of the poliovirus have been associated with particular populations and nations. For example, during the 2008 outbreak of wild poliovirus in parts of West Africa where locally identified wild strains had been eliminated, genomic sequencing of these strains found in Togo, Niger, and Ghana were identified as Northern Nigerian poliovirus (Renne 2010:86). There have been various reactions to these national classifications of poliovirus. On the one hand, a Northern Nigerian man was indignant when I told him that cases of polio in Ghana had been identified as Nigerian variants of the disease. On the other, a Ghanaian health worker did not blame Nigerians for the outbreak of the eight wild poliovirus cases in the Northern Region of Ghana. “If Nigeria is [exporting] this thing—they still have the virus, if we reach the level of immunization that is higher we won’t be affected” (Renne 2010:100)—i.e., Ghanaians were responsible for the outbreak due to immunization gaps in the Northern Region. Yet these sorts of “geographies of blame” nonetheless have their uses, not only in tracing the spread of wild poliovirus, but also in shaming Nigerian government officials to step up their immunization campaigns.

6 It was also considered somewhat suspect, with one colonial scientist suggesting that people lied about their ethnic backgrounds when it was advantageous (171).
References: