Sickle Cell Disease: A Cultural History

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In April 1973, cinemas throughout America began screening a tearjerking love story, the plot of which revolved around sickle cell disease. 'A Warm December' showcased Dr Matt Younger (played by Sidney Poitier, who also wrote and directed the film) and an African princess called Catherine Oswandu (played by Esther Anderson, who won a National Association for the Advancement of Colored People Image Award for Best Actress for her role). Matt is a charming doctor, father, and recent widower, who falls madly in love with the sensual yet mysterious Catherine. Matt observes that she is always being followed by a couple of shifty men; her regular, sudden disappearances only fuel his infatuation. Eventually, it is revealed that the men tailing her are monitoring her sickle cell condition and giving her blood transfusions to keep her alive. Catherine is in the 'December of her life'.

Let's see a clip from the film. In it, Matt has finally uncovered Catherine's secret and turns up (uninvited) to her home to ask permission from her uncle (an Ambassador) and her physician to take Catherine on a vacation. Because of her health problems, Catherine is reluctant to burden him, but he asks her 'How many times do you pass through?', a veiled reminder of 'passing through life into death'. He reminds her to bring warm clothes (cold weather can spark acute sickle cell episodes) and learns from her doctor that she needs regular blood transplants and takes sedatives for the pain. (See: https://www.youtube.com/watch?v=cpmCMem02Jg, clip between 1:41 and 3.38).

The film is definitely a 'weepy'. Although Catherine is passionately in love with Matt, she turns down his marriage proposal because she wants to protect him and his young daughter from future distress. 'Goodbye, my husband, thank you for a warm December', is the Swahili phrase she uses to bid him farewell. The film was panned by critics, but it brought Sickle Cell Disease to broader public awareness. It also bolstered political consciousness of Blackness and health care disparities.

Why is Sickle Cell condition important? As we will see, it enables us to reflect on 'race' and the racialization of illness in ways that problematise how discussions about Sickle Cell Disease are framed. This talk on sickle cell condition also serves as an internal critique of my own lectures in this series. But first, let me set the scene by discussing some more conventional aspects of the history of a condition known as Sickle Cell Disease.

The blood condition known in medical texts as Sickle Cell Disease is not uncommon. One in ten Black Americans possess the sickle cell trait and one in 375 develop the condition. In England, it affects one out of every 2,000 births and one in 70 babies carry the gene. This makes Sickle Cell one of the most common recessive diseases. Crucially, it is a condition that is highly correlated with specific populations, as is Tay-Sachs disease amongst people of Ashkenazi Jewish descent and cystic fibrosis among whites with ancestry from northern Europe. Sickle cell disease is much more common in people whose ancestors are from sub-Saharan Africa, as well as in people from Hispanic, Mediterranean, East Indian, and Middle Eastern ancestry – although the condition is not exclusive to people with such ancestry. However, since its foundational moments, it has been a racialised disease. In the words of physician Verne Mason, reporting on the fourth case of sickling in 1922, 'the malady has been seen only in [people of African American descent], and, so far as could be ascertained, it is the only disease peculiar to that race'. It is not surprising, therefore, that the condition was well-known in African folk tradition. The enslavement of people from the African continent brought awareness of the condition to white enslavers. In popular parlance, Sickle Cell is known as the 'Black disease'.

It is highly likely that all estimates of its prevalence amongst minoritized populations are huge

underestimates. People with the sickle cell *gene* as well as those with the *disease* often don't display obvious signs of it. Indeed, in the early years, before its molecular structure was known, Sickle Cell conditions were often mistaken for malaria since the symptoms mimicked that disease. Indeed, before the 1950s, it is highly likely that many deaths from Sickle Cell Disease were ascribed to other diseases or to complications of conditions such as pneumonia. This was why some physicians called it the 'great masquerader'.

One of the earliest accounts was written by Dr Robert Lebby Jr. (a quarantine officer in Charleston, South Carolina) and published in the *Southern Journal of Medical Pharmacology*. Entitled 'Case of Absence of the Spleen' (1846), Lebby reported on an autopsy carried out on an enslaved man who had been executed for attempting an escape. He had numerous leg ulcers and 'bilious intermittent and remittent fevers' and, on autopsy, there was no spleen. Although Lebby did not diagnose it as such, this was probably Sickle Cell Disease in which the spleen had atrophied due to the blood supply being cut off.

A more scientific understanding of the condition was first identified by medical researchers in the November 1910 issue of the *Archives of Internal Medicine* when Chicago physician James B. Herrick (and his intern Ernest Irons, who did most of the work yet was not credited in the published report) diagnosed the disease in a twenty-year-old dental student called Walter Clement Noel. Noel had pulmonary symptoms and anaemia, but what intrigued Herrick was Noel's unusual 'sickle shaped' red blood cells. He hypothesized that these distorted-shaped cells had difficulty passing through blood vessels, eventually blocking the vessels and preventing oxygen from reaching tissues. Herrick's report was followed just three months later by a paper written by fourth-year medical student Benjamin Earl Washburn from the University of Virginia Hospital. In contrast to Noel, who had been born into a wealthy Black family from Grenada, Washburn's patient was female and poor. We now know that this patient was a cook and housemaid named Ellen Anthony, descendant of an enslaved family. Together, Walter Clement Noel, Ellen Anthony, and their physicians transformed what was known about Sickle Cell Disease.

Less than four decades later, chemist Linus Carl Pauling, along with Harvey Itano, Seymour Singer, and Ibert Wells discovered that the condition was caused by an abnormality in the haemoglobin molecule. In their 1949 article in *Science*, they explained that,

> *"The erythrocytes of certain individuals possess the capacity to undergo reversible changes in shape in response to changes in the partial pressure of oxygen. When the oxygen pressure is lowered, these cells change their forms from the normal biconcave disk to crescent, holly wreath, and other forms."*

There was, they concluded, 'a direct link between the existence of "defective" haemoglobin molecules and the pathological consequences of sickle cell disease'. The condition also had a genetic basis: it was recessive – that is, there is a difference between possessing the sickle cell *trait* and having sickle cell *disease* or *anaemia*. Indeed, the sickle cell *trait* is widespread and generally benign. To experience the devastating symptoms, a person had to inherit the gene from *both* parents. In summary, then, the condition known as Sickle Cell Disease is caused by a mutation in the haemoglobin molecule, whereby Haemoglobin A in the red blood cell is replaced by haemoglobin S. As haemoglobin S releases oxygen, haemoglobin polymers stretch the cells into the crescent shape of a sickle. Pauling named this process 'sickling'.

Sickle Cell Disease became the first condition identified as being caused by an abnormality in a protein. By 1956, further research by molecular biologist Vernon Ingram and John A. Hunt meant that Sickle Cell Disease became the first genetic disorder whose molecular basis was known. These were incredible findings for the new discipline of molecular science, holding out hope that basic science could cure disease. As Pauling put it,

> *"I believe that chemistry can be applied effectively to medical problems, and that through this application we may look forward to significant progress in the field of medicine, as it is transformed from its present empirical form into the science of molecular medicine."*

By 1959, Pauling could even be heard contending the radical statement that 'man is simply a collection of molecules' and 'can be understood in terms of molecules'.

These scientific breakthroughs were important contributions to our understanding about why Sickle Cell Disease is so devastating for those who have the condition. Because the 'sickling' of blood cells causes oxygen supplies to tissues and organs to be disrupted, it results in delayed growth and sexual maturation, pulmonary complications, renal failure, bone and joint damage, cognitive difficulties, stroke, and severe pain. To avoid having a stroke, people who develop sickle cell anaemia often require frequent blood transfusions (as did Catherine in the film 'A Warm December'). Many require between 37 and 75 blood transfusions

annually, despite the risk of iron-overload leading to liver and heart failure. The risk of stroke is significant from the beginning of a patient's life: most stroke victims with Sickle Cell Disease are between two and five years of age.

The central symptom of the condition, however, is excruciating pain. Between 70 and 90 per cent of sickle-cell related admissions to hospitals are due to painful crises. When sufferers are undergoing a pain crisis, they require (on average) more than seven days in hospital; in half of these cases, patients have to be readmitted within a month. Patients who survive into adulthood typically die in their forties.

Despite its prevalence, there was very little public awareness of the disease – even within Black communities – until the 1970s. A 1969 article published in *Public Heath Reports* found that only two in ten of Black adults had heard of the disease. Another study, this time of Black families in Boston who had undergone genetic screening for Sickle Cell Disease, found that fewer than half were aware of the condition but none realised that it was hereditary. Only five of 150 Black army recruits in Texas had heard of the disease. In 1981, in a survey of nurses and health visitors in Brent, Kensington, Chelsea, and Westminster (where there were large ethnic minority communities) found that 72 per cent did not know that Sickle Cell Disease was not a form of cancer and only 14 per cent knew that the main treatment were analgesics. As late as 2006 in the UK, there were *no* nation-wide clinical standards for the disease. Given such level of ignorance, it is hardly surprising that misinformation was rife, with patients warned not to exercise, travel on planes, or visit tropical countries.

Clinically, the disease was also neglected. A 1984 survey by the Runnymede Trust (a British organisations exploring issues relating to racial inequalities) found that only a few health districts provided *any* services dedicated to people with the condition. Even districts where more than one-third of residents were BME, almost none employed *any* medical professionals or counsellors for people with Sickle Cell. The most damning account was made by leading British sickle cell haematologist Milica Brozović and nurse Elizabeth Anionwu in 1984. They observed that around 'one third of the adults with sickle cell disease have a severe debilitating disease and require much effort and time in both inpatient and outpatient care'. Yet, management of these patients was 'haphazard, with poor interdisciplinary cooperation and little or no coordination of effort'. Brozović and Anionwu contended that the situation was comparable to that of haemophiliacs three decades earlier.

Treatment or even cure of this chronic condition has been slow. In 1984, the first successful cure of the disease by bone marrow transplant was carried out. The transplant involved destroying the patient's bone marrow and replacing it with bone marrow from a normal, genetically-matched sibling. Many people suffering the disease have no such donors. Similarly, Stem Cell transplantation is a cure but is extremely expensive. Only in 1995, a randomized, double-blind, placebo-controlled trial called the Multicenter Study of Hydroxyurea in Sickle Cell Anemia, showed that Hydroxyurea could prevent the painful crises of Sickle Cell patients. This was the first drug shown to work. Its effectiveness is due to the fact that it increases the synthesis of foetal haemoglobin – and was the result of paediatrician Janet Watson observation that new-born babies with sickle cell anaemia were symptomless because the foetal haemoglobin protected them. Even today, however, most patients continue to be treated with pain management, hydration, oxygen, and antibiotics.

Part of the neglect of Sickle Cell sufferers is due to the fact that most are from minoritized BME communities and poor. Sufferers lack the 'media clout' of many other desperately sick people. They are often uninsured, so are not profitable for pharmaceutical companies. A study in 2019 found that Sickle Cell Disease receives significantly less funding that other diseases. For example, although cystic fibrosis affects fewer than one third the number of people, it receives 3.5 times the amount of funding from the National Institutes of Health and 440 times the funding from national foundations. In the words of Garry Dawson in 1977, 'If the children of company directors, MP's, and disc jockeys died horribly of sickle-cell anaemia, it would be a more popular cause than polio and cystic fibrosis research funds put together'.

Sufferers often turn to prayer in their attempts to deal with pain and survive. This is not surprising: 95 per cent of adults in the U.S. say they believe in a God, 88 per cent pray to God regularly, and 72 per cent claim that organized religion is the single most important coping influence in their lives. Black churches have long played important roles in supporting their communities, including promoting physical as well as spiritual healing. Second to God, the other technique used to cope with the agonising pain of Sickle Cell Disease has been dubbed 'John Henryism'. Named after the Black labourer who worked so hard that he defeated a mechanical steel drill, but shortly afterwards died from mental and physical exhaustion, it is shorthand for a coping style demanding hard work and determination against all odds and whatever the cost.

As already mentioned, the most central aspect of the disease is pain, which is caused when the sickle-shaped cells block veins, starving organs of oxygen and leading to hypoxia or oxygen deprivation. As I show in my book *The Story of Pain: From Prayer to Painkillers*, pain is a political and cultural problem as much as a medical one. In the 1960s to the mid-1970s, physicians increasingly became aware of the differences between chronic and acute pain – the two required very different treatments. In many jurisdictions chronic pain specialists established multidisciplinary teams to deal with the chronicity of diseases such as Sickle Cell. This meant the coordination of mental health professionals alongside GPs, pharmacologists, surgeons, and anaesthesiologists. By the 1990s, this had fallen out of favour, in part due to its costs but also due to a greater suspicion of patients. People suffering sickle cell disease were increasingly believed to be seeking drugs (especially if 'male and Black'). Because they built up tolerances for analgesics, they were regarded as particularly suspect. This was due to what Carolyn Rouse called the 'hyper surveillance of sickle cell patients', primary because they were Black. They were routinely disparaged by medical professionals as 'frequent flyers'. As Dr Holly Christopher Lewis, immunologist and public health activist, complained,

> *"In paediatrics, sickle cell patients often get the antibiotics, checkups, and preventive care that they need, but when a black man turns eighteen, he typically can't go to the children's hospital anymore. In an adult hospital, he may get labelled a 'frequent flyer', an addict, all these other connotations. And that's what complicates their access to care and hastens their early death."*

There were disparaging comments about minoritized people having low pain thresholds. Their chronic condition often means that they are forced to take time off work, thus finding it hard to hold down a job. They are routinely denied health or life insurance. Or are offered such at a huge cost. Many people with Sickle Cell conditions stop seeking pain relief because of internalised shame about not being able to 'manage' their pain or not being sufficiently stoical. As a result, patients experience much higher levels of pain than most other chronic pain suffers. 'Good' patients are stoical ones, as we saw with polio patients in an earlier lecture in this series.

Treatment resources are poor. There continues to be a serious shortage of specialist Sickle Cell Disease centres, particularly in low-income communities. In 2010, a U.S. study of over 21,000 people with Sickle Cell conditions found that, although 77 per cent had been hospitalised and 94 per cent had visited an Emergency Department (where waiting times are often long), 60 per cent used Medicare or Medicaid as their primary payer. This is in contrast to between one-quarter and one-third of patients with haemophilia and von Willebrand disease. As researchers LaTasha Lee, Kim Smith-Whitley, Sonja Banks, and Gary Puckrein point out, 'these differences are important because Medicaid limits access to specialized health care'.

Screening, too, is a highly political and racialized act. 'Carrier' status is key to discrimination. It is marketed as important in reproductive decision making in which, for physicians, 'choice is always good' and 'knowledge is power'. But that is not necessarily how it is experienced. For couples who learn that both are 'carriers' so there is a one-in-four chance that their child will 'have' sickle cell disease' (and not be 'simply' a 'healthy carrier') is simply abstract, de-contextualized knowledge.

Furthermore, there are numerous reasons for Black communities to be suspicious of white physicians and geneticists who wanted to established screening programmes aimed solely at them. Might these medical professionals actually be part of a move to reduce the birth rate amongst Black communities? Even in the late-1980s, a 'respected scientist' proposed tattooing a person's sickle cell carrier status on the forehead of every young person who possessed the trait in order to limit transmission. Genetic testing can lead to increased discrimination by employers and others. It affects marriage and parenthood possibilities. Carriers are even blamed for their own illness: for example, pregnant women and their partners are seen as 'difficult' when they refuse testing or get tested too late. But are right to distrust 'genetic medicine' and its eugenic underbelly.

Disregarding the health of minoritized people is hardly a new phenomenon, as we have seen in every lecture in this series. There is a formidable historical literature documenting racist assumptions about Black bodies. For example, Sickle Cell Disease has been used as evidence of Black physiological inferiority. As the insurance analyst Frederick Hoffman asserted in an extraordinary piece of scientific racism in 1899 (this is his book entitled *Race Traits and Tendencies of the American Negro*), Black people were constitutionally weaker than their white counterparts so would eventually decline and fade away. In his words, 'It is not in the conditions of life, but in the race traits and tendencies that we find the causes of excessive mortality.' Or, as he argued in an 1892 article, 'Something must be radically wrong in a constitution thus subject to decay. Even if he be placed on equal grounds [to the white person] he still will exhibit… "his race's proclivity to disease and death"'.

Some commentators even pointed to the condition as indicative of the multiple evolutionary origins of human populations. For example, in the 1943 issue of *The Ulster Medical Journal*, William Dickey asked 'Is race a factor in the causation of disease?' His response was 'no', but he observed that many (white) people would have answered in the affirmative. 'Popularly', Dickey contended,

> *"race is accepted as a self-evident fact; to deny its existence seems opposed to common sense. If we think not only of the differences that seem so obvious between European peoples, but of those, throughout the world, between Europeans, Negroes, Chinese, and Australian Aborigines, we see variations so great that there has been serious discussion as to whether mankind can be included in a single species or must be divided into several."*

Indeed, the idea that 'Black blood' was different from 'white blood' has a surprisingly long life, even after blood types could be categorised as ABO. Not only segregationists but medical professionals, too, (and the two were not mutually exclusive!) were determined to link blood 'types' to 'racial' classification systems. There was a lot at stake in such categorisations. It enabled people to politicize the migration of different populations into the cities. It was a key plank in arguments about what has been derogatorily labelled 'miscegenation', or inter-'racial' relationships. These debates wrongly assume that certain populations are biologically discrete, homogeneous, or 'pure'. Especially when Sickle Cell Disease was believed to be inherited if only *one* parent possessed the trait (which is not correct: it requires both parents to possess the trait), there were exaggerated fears that the 'white' population could be in jeopardy. As historian Carolyn Moxley Rouse explains, in the 1920s, the presumption that Sickle Cell Disease was a dominant trait, meant that 'intermarriage marks the beginning of a rapid spread of the disease into the white population. For many, sickle cell disease proved that antimiscegenation laws were not simply a quaint custom but necessary for [white people's] survival'. When Sickle Cell Disease was found to be more prevalent in African-Americans compared with Africans on that continent, the explanation was said to be the higher levels of 'racial' 'mixing' in the U.S. As Melbourne Tapper explained in *In the Blood: Sickle Cell Anemia and the Politics of Race* (1999) the greater prevalence of the disease in North America was said to be proof of the 'dysgenic effects of race-mixing'. Similarly, prior to the 1940s, when physicians found the disease in patients they assumed were 'white', they responded by seeking 'black blood' in their 'white' patients. Were they 'truly' 'white'? Were people of Mediterranean ancestral origins 'white' or not? Linking Sickle Cell conditions with 'Black blood' enabled commentators to correlate alleged biological unfitness with social, economic, and political inferiority.

In the nineteenth and early twentieth centuries, Black spokespeople could be heard loudly critiquing the assumptions embedded in medical theorising that Black bodies were inherently weaker ones. W. E. B. Du Bois was one of many leaders who insisted that one of the most severe forms of injustice was the unequal burden of ill-health. He was particularly scathing about the work of Hoffman. In *The Philadelphia Negro* (1899), Dr Bois made the case that so-called 'scientific evidence' for the poor health of Black Americans was actually due to prejudicial observations. Although he did not totally deny that there may be 'some hereditary predisposition' for higher morbidity and mortality amongst Black populations, he made the case that the most important explanations for ill-health amongst Black populations were not their physiological constitution but 'poverty, ignorance and general social deprivation'. This was hardly surprising, he contended, given that the majority lived 'in the most unhealthful parts of the city'. He also noted that the situation was not helped by the reluctance of Black people to seek help from physicians and medical professionals. They had rational reasons for avoiding hospitals, failing to follow through on medical advice, and being less 'compliant' (Dr Bois did not use this term) in the face of allegedly 'expert' opinions. Du Bois explained that their 'superstitious fear of hospitals' had 'some foundation in the roughness or brusqueness of manner prevalent in many hospitals' and the 'lack of a tender spirit of sympathy' by medical professionals towards 'the unfortunate patients'. There was an undercurrent of rage accompanying Du Bois' lament that there have been,

> *"few other cases in the history of civilized peoples where human suffering has been viewed with such peculiar indifference. Nearly the whole nation seemed delighted with the discredited census in 1870 because it was thought to show that the Negroes were dying off rapidly, and the country would soon be well rid of them."*

He expected commentators who believed that 'the [Black] race is doomed to early extinction' to conclude that there was 'little left to do but to moralize on inferior species'. This had to be resisted. Du Bois called for 'increased effort and sound upbuilding' rather than 'passive indifference, or increased discrimination'.

Throughout the century, Du Bois' insights have been echoed by other Black leaders. An important supplement to De Bois' arguments was made by Black physician Julian Herman Lewis. In 1942, he published *The Biology of the Negro*, which in introduced the term 'anthropathology' or 'comparative racial pathology'.

Lewis' writings were a trenchant refutation of scientific racism that insinuate that people were darker skins were 'racially' inferior. Scientific writings were biased, he observed, since they assumed that white patients were the 'normal' and therefore their 'race' did not need to be mentioned, while, when scientists conducted tests on Black subjects, 'race' became a notable feature. He pointed to scientific research on blood types as one example. In 1922, he was able to show that there was no difference by 'race' in the distribution of ABO blood types. In other words, 'racial mixing' had no impact on the 'blood' of children.

Similar refutations were made by physician Charles Roman in his book *American Civilization and the Negro* (1916). Roman maintained that although 'all kinds of varieties are found in all races', nevertheless, 'all men are equal physiologically at their birth, and never cease to be so till they die'. As the editor (between 1909 and 1919) of the *Journal of the National Medical Association* (*JNMA*), a medical journal dedicated to the health of African Americans, Roman was able to categorically assert that,

> *"There is no such thing as racial immunity or susceptibility to disease. Immunity and susceptibility are both products of environment that affect humanity individually and not racially."*

The difficulties in getting this message across was identified in a 1948 editorial of *JNMA*. It warned that little was known about Sickle Cell Disease, sardonically noting that 'nearly all the information that we have about the condition has been obtained by scientists of other races', by which they meant 'white' scientists.

Of course, racism and scientific biases were not the only reasons for the relative neglect of research into Sickle Cell Disease. Even Black physicians might decide that there were more urgent diseases affecting Black communities: TB, for example (as we saw in a previous talk in this series). Sickle Cell conditions also had no cure at this time; it was not irrational, therefore, for minoritized physicians, medical officials, and community workers to focus scientific and medical energies on public health initiatives that could be effective.

The civil rights movements from the 1960s changed everything. Along with every other aspect of Black lives, disease was politicised. Black civil rights leaders and revolutionary political parties saw in Sickle Cell Anaemia a symbol of discrimination against Blacks as well as a way to mobilise Black aspirations for better lives. For the Black Panther Party for Self Defense (founded in 1966), the failure of governments to address Sickle Cell Disease was simply another example of distain for Black lives. They instigated major public health programmes, eventually shifting from ideas of self-defense to those of self-help. Their clinics educated Black people about Sickle Cell Disease, provided genetic screening, and spread the view that the politics of Sickle Cell was the politics of institutionalized racism.

Federal and local politicians began to listen; at the very least, they had a lot to gain electorally by aligning themselves with (strictly limited) aspects of the civil rights movements. Even President Richard Nixon recognised the political value of drawing attention to Sickle Cell Disease as signalling his support for Black causes. On 18th February 1971, he mentioned the disease in his health address to Congress. A year later, the National Sickle Cell Anemia Control Act was passed, providing funds for research into the disease. Ironically, Nixon initiated these measures at the same time as he introduced laws that adversely affected Black and other minoritized communities. Increased funding for Sickle Cell research was well-meaning but was effortlessly co-opted into a 'politics of pity' and the 'politics of electoral egoism' rather than anything more substantive.

The resurgence of interest in Sickle Cell Disease lasted less than two decades. By the 1990s, the condition had not only fallen back into obscurity (taken over by more 'white' afflictions such as breast cancer, AIDS, and dementia, as we have seen in other lectures in this series) but had arguably become even more stigmatised than it had been prior to the 1960s. After all, Carolyn Moxley Rouse has observed in her book *Uncertain Suffering: Racial Health Care Disparities and Sickle Cell Disease*, the U.S. was 'substantially closer to racial equality [in life expectancy] in 1945 than it was by the end of the century'. Sickle Cell neglect is part of this rising inequality in health care. The increased stigmatisation of the disease was the result of a huge conservative backlash focussing on 'welfarism', 'malingering', and drug addiction. The 'war on drugs' was incredibly harmful for sufferers of Sickle Cell conditions. Not only did Sickle Cell patients find themselves labelled 'druggies', but its *'racialized'* element meant that they were also accused of dealing in drugs and faking Sickle Class Anaemia crises in order to defraud health systems and insurers. In 1997, a survey of American medical practitioners found that 53 per cent of Emergency Department physicians and nearly one-quarter of haematologists believed that more than one fifth of the adults suffering an acute Sickle Cell episode were addicted to pain-relieving drugs. Even children with the condition were labelled addicts, according to between one-fifth and one-quarter of haematologists and Emergency Department physicians, respectively. For patients in the midst of an excruciating Sickle Cell crisis, this was devastating since Emergency Department physicians were their primary carers. In reality, their situation was even worse. After all, most

Emergency Department physicians only saw the patient once so had no insight into how their patient's pain progressed, waned, and flared up again. This meant that they tended to discharge patients from the ward 'with an inadequate supply of analgesics' which increased the risk of the patient having to return and, consequently, be regarded as either addicted or attention-seeking. Or both.

One of the most insightful analysis of this process is that of Keith Wailoo in his influential study *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Death* (2001). Wailoo argues that Sickle Cell Disease and syphilis (as most notoriously represented in the Tuskegee Syphilis Study) were the main illnesses used to denigrate both the biological and social standing of African Americans. For Wailoo, diseases are commodities – they have a

> *"place in a network of exchange relationships, where – much like any object – the disease concept and the illness experience acquired value and could leverage resources, money, or social concessions."*

In particular, Wailoo shows how hospitals, medical professionals, and politicians, as well as patients themselves, use Sickle Cell Disease to further their own concerns. Interestingly, though, there is a geopolitical history to these uses. In Memphis, for example, physicians were particularly anxious about addiction so tended to under-treat the pain of Sickle Cell; in contrast, physicians in Oakland and Chicago believed that the main aim of treatment was to eradicate or blunt pain as much as possible. In this way, where a person lived had huge implications for pain treatment and disability allowances.

Although similar dynamics were observed in Britain, there were other factors that dominated the British scene: empire, decolonization, and migration. The best analysis is that of Grace Redhead, in an article (which I hope will soon be a book) entitled '"A British Problem Affecting British People": Sickle Cell Anaemia, Medical Activism and Race in the National Health Service, 1975-1993'. Redhead's arguments are subtle and complex (please read her incredible articles and blogs), but one of the chief points she makes is the irony of the fact that migrants from the former British empire 'made the welfare state possible and shaped its forms of assistance in myriad ways', yet the same people were accused of being 'welfare parasites' when they used it. By the end of the 1960s, nearly one-third of nurse pupil vacancies were being filled with Commonwealth migrants. By the mid-1970s, between 18 and 32 per cent of people working in hospitals were born outside of Britain. Yet, these workers were reproached for bringing the 'Black Blood disease' into a 'white England'. Migrant nurses and other NHS workers were even accused of spreading the condition throughout the wards.

In Britain, as in the U.S., neither racism nor the neglect went uncontested. Local haematologists, nurses, and patients' rights activists began organising. These included the Organisation for Sickle Cell Anaemia Research (OSCAR), which was founded by patient activist Neville Clare in 1975. In 1979, the Brent Sickle Cell and Thalassaemia Centre was founded by haematologist Milica Brozović and nurse Elizabeth Anionwu, who also founded the Sickle Cell Society that year.

In my final reflections, I want to turn to a problem that is pervasive in too much of the debates about Sickle Cell – including in my work. Particular ideological discourses (specifically medical sciences and anthropology) have historically claimed that there is a link between sickle cell and *'race'* or racial distinctiveness. By identifying 'race' as an indicator for diseases such as Sickle Cell Anaemia, we risk cementing the false idea that 'racial' groups are biologically determined rather than simply an arbitrary way of distinguishing different populations. There is more variation within groups bundled into 'races' than between them. In other words, the racialization of diseases such as Sickle Cell reinforces the idea that there are immutable biological and genetic properties. It ignores the insight that 'race' itself is a social construct. This point was made forcibly by the American Association of Biological Anthropologists in their statement of 2019. They explained that,

> *"Race does not provide an accurate representation of human biological variation. It was never accurate in the past, and it remains inaccurate when referencing contemporary human populations. Humans are not divided biologically into distinct continental types or racial genetic clusters. Instead, the Western concept of race must be understood as a classification system that emerged from, and in support of, European colonialism, oppression, and discrimination. It thus does not have its roots in biological reality, but in policies of discrimination."*

'Race' is a way of 'sorting individuals and populations into units based on historical contexts and social, cultural, and political motives'. It is 'real' to the extent to which it 'affects our biology, health, and well-being'. In other words, while 'race' is 'not a scientifically accurate biological concept', it 'can have important biological consequences because of the effects of racism'. The Association recommended using 'populations' instead.

This is not to deny that Sickle Cell Disease is much more common in certain populations – specifically, people whose ancestors were from sub-Saharan Africa, as well as people from Hispanic, Mediterranean, East Indian, and Middle Eastern ancestry. But it is not exclusive to people with such ancestry. The gene is important in many areas because it acts as a preventive mechanism against malaria, which is why it is found in areas where malaria is endemic. Melbourne Tapper points out that this association with malaria has been important in the racialization of Sickle Cell. After all, he points out, the fact that sickling has 'historically occurred in those individuals, or their ancestors, inhabiting malarial water regions… has been used to establish a linkage between the sickle cell gene, the black [sic] body, and natural selection'. But, of course, malarial waters are found elsewhere in the world. This 'suggests a linkage not between the sickle cell gene and certain peoples, races or bodied (such as the black body) but, rather, between the sickle cell gene and certain geographies'.

In conclusion. Diseases are meshed in social worlds. Medical and insurance cultures *matter*: worlds are changed depending on funding of the NHS or Medicare; it *matters* how genetic counselling is conducted. As we have seen in this lecture but also throughout this series of six lectures, the role of activists make a difference. But although more research into Sickle Cell Disease and improved health care facilities for minoritized communities are urgently required, if a real difference is to be made for people who suffer this condition more attention must be paid to ways of tackling systemic inequalities at every level.

References and Further Reading

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