

## Race and Health part 2

Hi, I'm Jo, I'm Jim, I'm Erik and this is Speaking of Race, the podcast that takes on our misconceptions about what science says about race.

Erik: ...and last time we began a series discussing misconceptions around race and health. Is it just me, but do there seem to be more misconceptions about this than almost anything?

Jim: You might be right. And the scary thing is misconceptions here can have real life-and-death consequences.

Jo: Last time we began the discussion of race and health. In my experience, one of the most misunderstood race and medicine issues of all is sickle cell, so let's talk about that today.

Erik: I agree. I think when we attempt to say that "race" is socio-cultural but not biological, people always come right back with, "Well then what about sickle cell..."

Jo: It's often thought of as a "black disease," right...

Jim: Yep, as I learned while teaching about race. For most of the 15 years I taught the race class at the University of Alabama I gave a pre-test to see what students thought coming into the class. Now it's true that many of them had had anthropology classes before hand so they came in with the notion that race was cultural, not biological. But, over a third of them still thought that, even though races aren't genetic categories, there're diseases that are specific to one race or another. And when asked what diseases, guess what they said?

Erik: Since we're discussing sickle cell today I'm going to go out on a limb here and say ... [is funny]

Jo: Same with my classes. Whenever I introduce the idea that race isn't biological, someone says, "But what about sickle cell?" I've talked about that on the podcast before.

Erik: Can I ask a history question? Do we know how old sickle cell is?

Jo: That's a tougher question than you think.. There's almost certainly mentions of ailments that are likely sickle cell in classic Greek and Roman tales, but it really can't be separated out from other causes of anemia like thalassemias. The first case in the US may be from 1846—diagnosed in a runaway slave who was killed and autopsied—although there is no evidence that the telltale sickle shaped red blood cells were present (Siddiqi, Jordan, & Parker, 2013).

Jim: And as to how old the disease itself is, there's a current argument based on different genetic interpretations, with estimates ranging from 7300 to 22,000 years ago.

Erik: So ... is that where we start the story? 22,000 years ago?

Jim: No, I think we start on September 15, 1904.

Jo: Whew, that's much more manageable.

Erik: [Joke about spending the rest of your life on this episode.] But why such a specific date?

Jim: On September 15, 1904, a 20-year-old Grenadian, Walter Clement Noel, arrived in New York City. He was on his way to attend the Chicago College of Dental Surgery. But he paused on his journey because he had a sore on his ankle. After his ankle had healed up, he continued on to Chicago (Savitt, 2009).

Erik: Wait, wait, what's the deal with the sore on his ankle? That doesn't seem important enough to ... start there...?

Jo: Leg ulcers are a common complication of sickle cell and some other anemias, I know that much. But I have no idea why Jim is starting with this guy.

Jim: Well, I haven't finished the story yet, guys! Noel made it to Chicago to attend dental school. By Thanksgiving that year, he was having trouble breathing but he muscled through until the day after Christmas when he walked across the street from where he was staying to Presbyterian Hospital. He was seen by an intern, Ernest Irons, who took a medical history and performed standard physical, blood and urine exams. In the blood smear, Irons saw, in his words, "many pear-shaped and elongated forms—some small," which he brought to the attention of his attending physician, James Herrick. About a week later, after doing another blood analysis, Irons drew a rough sketch of these red blood cells. These turn out to be the first depiction of sickled cells that we know of.

Erik: Wow! So 1904 Chicago is when we first get real documentation of sickle cell.

Jim: Not exactly. Irons and Herrick followed several of these episodes with Noel over the next couple of years, but never came up with a diagnosis, and they lost contact with him before he left Chicago. In 1907, Noel graduated from the dental school and returned to Grenada to practice dentistry, where he died nine years later.

Erik: And then we get real documentation of sickle cell!

Jim: AHEM. Herrick finally wrote up the case, barely acknowledging Irons, and published it in 1910 with the title, "Peculiar elongated and sickle-shaped red blood corpuscles in a case of severe anemia" (Herrick, 2001 [1910]).

Jo: So, now, finally we get real documentation of sickle cell?

Jim: Still not quite, you eager beaver, you.

Jo: I'm a duck. The Beavers are Oregon STATE. I'm University of Oregon.

Jim: You Overpriced Nike Sneakers, you. No, it took several more cases, including one based on a case in an African American woman from Virginia published in 1911 with the same title that Herrick used (Savitt, 1997; Washburn, 1911).

Erik: And then!---

Jo: and then, along came sickle cell.

Jim: Yes, and then, about a few years later, in 1915, two doctors at Washington University-St. Louis published their examination of another case with the same symptoms as those described by Herrick (Cook & Meyer, 1915). They also speculated about the inherited nature of the disease because by then they learned that their patient had three siblings who also showed signs of anemia. And what's more, laboratory examination of the father's blood appeared to resemble the patient's blood -- that funny pear-shaped red blood cell. Also, every one of these individuals was of African descent.

Jo: Ah, okay, so by 1915 they began to recognize that no matter the symptoms it was a disease of the blood they were looking at. And if they learned about the siblings and the father then they knew it was heritable. But given what you just said, I imagine that they recognized the racial connection, too.

Jim: Yep, all the cases in this early medical literature were on people of African descent. And -- here's something else significant. Those two docs at Wash U were working with an anatomy professor, Victor Emmel, who designed a lab test for the cellular identification of sickle cell (Emmel, 1917). While trying to look at the cells under a microscope, he accidentally came up with a blood test. When he prepared cell cultures with blood smears he sealed them with Vaseline between two glass slides. It turned out Vaseline increased the sickling process!

Jo: Wait, didn't this mess up his observations?

Jim: Maybe, but Emmel found that if he made a little divot in the Vaseline and put drops of blood in the divot -- blood cells from people who weren't showing symptoms of anemia -- he might be able to locate sickled cells. So in the case of the siblings I just talked about that Cook & Meyer studied, Emmel took some of the father's blood (who was not showing symptoms of sickle cell) and put it in Vaseline and after a little while saw the cells becoming sickle shaped. He said that he was seeing a *tendency* to sickle, which indicated *latent* disease in the father's blood.

Erik: So at first it was normal -- and he wasn't showing any symptoms of being anemic -- but the Vaseline turned up some sickling cells after a while, so it must have been there, just hiding.

Jim: Exactly. Two other groups started using this technique, too -- in fact they figured out how to speed up the process a little bit. And in the early 1920s, they decided that sickle cell anemia was inherited as a simple Mendelian dominant trait (Huck, 1923; Sydenstricker, Mulherin, & Houseal, 1923).

Jo: OK, hold on right there, cowboy! We need to explain what a Mendelian dominant trait is.

Erik: Listeners at home! Now's the time to grab a pencil and a sheet of paper! We're going to draw Punnett Squares!

Jim: Well it refers to a characteristic that's under the control of a single gene with two forms (or alleles), one that's dominant and one recessive. If someone gets one each of the two different forms from their parents, in this case normal and sickle cell, that person would have sickle cell because it is assumed by these folks to be the dominant form.

Erik: Or as we historians like to say, 'heterozygous.'

Jo: Ha. that was a funny historian joke.

Erik: Wait, I'm confused. I thought that sickle cell was a *recessive* trait.

Jim: You're getting ahead of yourself. We'll get there. Until the late 1940s, sickle cell was thought to be dominant.

Erik: I mean it doesn't make sense, but it does.

Jim: It wasn't until after WWII that people figured out it actually wasn't a race-based Mendelian dominant disease.

Erik: Someone should tell Mr. Murdock, my high school biology teacher/volleyball coach, because I'm pretty sure he taught us it was -- in like 1990.

Jim: You might want to go back to high school and do biology over again.

Jo: I'm pretty sure he couldn't get in.

Erik: I would say that was a sick burn, but I went to public high school.

Jo: I rest my case. Anyway ... back to Emmel's Vaseline test. I can see how it became a way of seeing if someone passing as white was really black.

Erik: Whoa whoa ... What do you mean?

Jim: I know it sounds weird. If a blood test on a white person found sickle cell --

Erik: Ooooooh. They thought that meant that there were African ancestors somewhere in the lineage. Man, that just made me think of something I'd wondered about for a long time -- the anti-interracial marriage bills began spreading across the US right after World War 1 and blood tests in order to get a marriage certificate started right around the same time. Is that just a coincidence?

Jim: Probably not, but there's so much bleeding over from eugenics at that point in time that it's hard to assign blame.

Jo: Back to sickle cell, guys. So in the first couple decades that it was studied, all the arrows pointed to sickle cell being an exclusively black disease. No wonder so many people believe this is so! The idea has been around for over 100 years!

Jim: Maybe. At least that's the part of the story that's been repeated. But what we don't talk about is that scientists questioned this interpretation not long after Emmel's test was popularized. See, while scientists in Chicago and St. Louis saw a connection between sickle cell and blackness, Thomas Cooley in Detroit saw sickle cell in European immigrants from the Mediterranean region (Cooley & Lee, 1929). What's more, a number of studies in the late-1920s showed that Emmel's Vaseline technique would cause some people's cells to sickle but that they didn't develop any actual symptoms of anemia. So some raised the alarm -- perhaps the technique was creating false positives for sickle cell.

Erik: Oh! ... oh ...

Jo: What are you 'oh'ing about?

Erik: Well, I was about to get happy right then. Like people were going to see that the experiments were probably a little flawed and so that maybe they should give up their preconceived notions about what they thought was going on and rethink it. But then I got the feeling that this is the point in the episode where I'm going to get my hopes up that people began to realize that their racial prejudices aren't actually borne out by science only to have one of you dash them. Kind of like when Charlie Brown is running for the football and then Lucy pulls it away at the last second. Am I right?

Jim: This is indeed that moment! Let me pull away the football. Basically Cooley's findings were seen as aberrations. The very next year, a much more prominent study came out and said that sickle cell was "peculiar to the negro race" (Scriver & Waugh, 1930, p. 380).

Erik: At least I didn't get my hopes up that time. ... But wait. We have to back up. A minute ago, Jim, I asked about them thinking it was a Mendelian-dominant trait and you said they did. But by 1930, they *had* to have realized they were wrong. I mean, if sickle cell was a dominant trait like brown eyes, wouldn't we have a lot more people running around with sickle cell?

Jo: Yeah, we sometimes talk about it being a recessive trait in that old Mendelian sense. So, everyone, pull out your Punnett squares again. It turns out, you only actually have sickle cell anemia if you're unlucky enough to get two *recessive* copies -- the little s's -- one from each parent. And that doesn't happen that often. You can have one copy of the recessive gene and be a carrier, but you won't actually have the full blown disease. That's called having sickle cell 'trait'. But I have no idea when that transition from thinking it was dominant to think it is recessive actually happened.

Jim: That's why you guys keep me around. And why you pay me the big bucks!

Erik: Wait, you're making money on this?! Are we running ads for toothbrushes and mattresses now?

Jo: Why am \*I\* not making any money on this?!

Jim: Hey, I'm retired, I need another income stream. It took until after WWII. The definitive statement on that didn't come until the geneticist James Neel published the results of a study in 1949 (Neel, 1949). He examined the blood of 42 parents of 29 patients with sickle cell anemia and found that every one of the parents had red blood cells that would sickle in the lab. If sickle cell was caused by a dominant gene, only one parent per patient would need to carry it to pass it on. And based on its occurrence in the general population, the likelihood that all 42 would have it was about 1 in 100,000.

Jo: So clearly it couldn't be a dominant factor.

Jim: But then it became "the first molecular disease" when Linus Pauling became involved.

Jo: Who's Linus Pauling?

Erik: Oh, I know that one! Linus Pauling discovered how chemical bonding worked -- he won a Nobel prize for that in 1954 -- then he went on to discover the alpha-helix structure of proteins. He would have figured out DNA's structure too, but the US revoked his passport because they suspected he was a Communist, so he couldn't get to a conference to see Rosalind Franklin's

X-ray photographs of DNA, which is what Watson and Crick did get to see. Then he won another Nobel prize in 1964 for his opposition to nuclear testing that inspired Kennedy's nuclear test ban --

Jim: and then he went crazy on Vitamin C.

Jo: -- Ok, I think you made your point --

Jim: So in 1949, Pauling and his colleagues used a technique called electrophoresis to show that the hemoglobin molecules were different for those without sickle cell (so-called "normals"), those with one copy of the gene (heterozygotes or "sickle cell trait") and those with sickle cell anemia (Pauling, Itano, Singer, & Wells, 1949). Ten years later in 1959, it was demonstrated that the chemical difference between normal and sickle cell hemoglobin was due to a shift in the 6<sup>th</sup> amino acid (those are the building blocks of proteins) from Glutamic Acid to Valine in part of the protein that makes up hemoglobin (Hunt & Ingram, 1959). In the mid-1970s, that shift from Glutamic Acid to Valine was found to be the result of a mutation in the DNA from adenine to thymine (two of the four nucleotides or building blocks of the DNA molecule) (Marotta, Wilson, Forget, & Weissman, 1977).

Jo: OK, so now we understand the biochemistry and genetics of sickle cell. But now we have to tie all this to race.

Erik: Yeah, we keep saying that race is not linked to this mutation that shifts Glutamic Acid to Valine to make sickle cell. But we haven't actually connected all the dots. If sickle cell isn't a racial disease, why does it seem to affect so many more black people in the US than white people?

Jo: I love this part. this is the part where we get to talk about malaria.

Erik: That's kind of sadistic-sounding, Jo. Who loves malaria?

Jo: Well, no one. but you know, from a human evolution and public health perspective, it's one of the most important diseases ever, because it's so widespread, deadly, exacerbates health disparities because it disproportionately affects the most impoverished people in the most impoverished equatorial countries, and until just a few weeks ago, there was no vaccine for it at all.

Erik: That's still disturbing that you love it.

Jo: I don't love it, meanie. it's a damned important public health topic!

Jim: But wait, Jo, something you said there is key -- equatorial countries. This, Erik, is the answer to your question about why it is that sickle cell looks racial even when it isn't.

Erik: OK ... more mosquitoes on the equator ... help me out...

Jim: Just after WWII, several people were in East Africa, and they noticed that people with sickle cell *trait* (remember -- "trait" -- isn't "disease" -- "trait" is just one copy of the gene) had fewer malaria parasites in their blood lab work than people with no copies (Beet, 1946). A.C. Allison, a South African physician, traveled to Kenya to collect blood samples for genetic analysis. He noticed that sickle cell trait was *more* common in parts of Africa where malaria was

more prevalent. And he discovered that malaria parasites were *less* common in individuals who had the sickle cell trait. Here is a quote from his 1954 paper.

[silence]

Jo: Erik, you must fulfill your contractual quota of reading at least one quote per episode. Go!

Erik: WE have a CONTRACT?! is that where you're getting all your podcast fortunes, Jim?

Jim: There's such a thing as podcast fortunes?! I thought they were just a time and money drain!

Jo: READ.

Erik: Okay okay. "It is concluded that the abnormal erythrocytes of individuals with the sickle-cell trait are less easily parasitized by *P. falciparum* than are normal erythrocytes. Hence those who are heterozygous for the sickle-cell gene will have a selective advantage in regions where malaria is hyperendemic. This fact may explain why the sickle-cell gene remains common in these areas in spite of the elimination of genes in patients dying of sickle-cell anaemia" (Allison, 1954, p. 294).

Jo: There it is, Erik! The answer to your question!

Erik: So that quote had so much jargon in it that there is no way listeners are going to have any idea what it says.

Jo: There are a few key terms in there, especially "selective advantage" and "hyperendemic." Jim, can you decode?

Jim: So, Allison was describing the phenomenon that fancy rich podcast barons, like myself, call the 'heterozygote advantage.'

Erik: So 'heterozygote' means you have one copy of "normal" and one copy of "sickle cell" in the old Mendelian lingo.

Jim: Exactly. And in an environment where life-threatening malaria is constant, individuals who have one copy of the sickle cell gene (the so-called sickle cell trait) have a malaria survival advantage over people who just have normal hemoglobin.

Erik: So if I get out my handy-dandy Punnett square and I get my big 'A' for normal and my scho'S' for sickle cell and I do 'A/A' that's bad. Because malaria.

Jo: Right. If your blood is completely normal and you have no sickle cell, you're going to get malaria and you're going to have a hard time having kids or, you know, living.

Erik: But if I have 'S/S' that's bad too, right?

Jim: Right. Because that's having sickle cell *disease*, not just sickle cell *trait*. And that also has a low life expectancy.

Erik: ... Filled with sores on your ankles.

Jo: Ew. But yes. In an environment like equatorial Western Africa where malaria is really endemic, in other words, you're getting squeezed from malaria on one end and sickle cell disease on the other. BUT if you happen to have ONE copy of the sickle cell gene, so you have sickle cell trait, you're doing well, because you've got some natural immunity against malaria and you very rarely suffer any of the sickle cell symptoms. That can make the difference between life and death.

Erik: Okay. So lots of malaria in equatorial Western Africa means sickle cell is a disease of people belonging to countries like Benin, Nigeria, and Senegal... how is this helping the argument that it's *not* a black disease?

Jim: Malaria is in areas around the equator. But that's not the only place. Malaria is bad around the Persian Gulf and in eastern India. But there's a big clue about where else it is in the word itself: *Mala Aria*.

Erik: Oh! From the Italian for Bad Air --

Jim: --and sickle cell can also be found around the Mediterranean as Thomas Cooley said back in the 1920s. That includes places like southern Italy, Greece, Turkey, parts of the Arabian peninsula, in addition to India and much of sub-Saharan Africa, but not southern Africa.

Jo: So sickle cell isn't a disease essential to blackness -- just an equatorial disease there in part as an adaptation to another disease. Ironically, hints of what we just laid out were first published in 1942.

Erik: 1942?!

Jo: The University of Chicago MD/PhD Julian Herman Lewis -- the first African American MD/PHD ever, as far as I know -- wrote about this in his book *The Biology of the Negro* (1942). Lewis tried to convince people that many so-called racial diseases were in fact caused by culture and history not essential biological qualities.

Jim: Anthropologist Frank Livingstone ran with the malaria selection idea in 1958. He drew on evidence from archeology, linguistics, and culture history to show how malaria and sickle cell overlap in West Africa. He even gave an account of how malaria became hyperendemic in equatorial Africa. Agriculturalists thousands of years ago left water in open fields, which mosquitoes breed in. And agriculture created stable human host populations for the malaria parasite to thrive. Livingstone showed how the frequency of the sickle cell form of the hemoglobin gene spread as an adaptation (Livingstone, 1958).

Erik: To me this sounds like a pretty solid example of natural selection working in humans. Was Livingstone's 1958 paper the first well-documented case study of it?

Jim: That's right. And the extra benefit is that, once I get this across in class, students really begin to understand why sickle cell isn't just an African genetic disease.

Erik: Nope. Now you lost me again. We said that sickle cell isn't essential to people from Africa, but we've only given an example from Africa. So how does this show that it's not a "black disease"?



Jo: Jim gave me an easy thought experiment that I think helps everyone understand how contingent the story of sickle cell is.

Jim: Yes, we should walk through this.

Jo: So, in order to know about how sickle cell is thought of as a "black disease," you have to understand some history. The physicians and anatomists who claimed it was a black disease in the 1920s through 1950s ... just didn't bother learning American history.

Erik: Why am I not surprised.

Jo: Okay, Historian. Do some history. At the height of the transatlantic slave trade, what do we know about where most slaves came from?

Erik: The "slave coast" extended from Senegal in the north to Angola in the south -- something like 3000 miles of the West African coast.

Jo: About how many people from this region made it to the US?

Erik: Well, over 4 million came to the Caribbean and about another half-million came directly to cities like Charleston, Mobile, Boston, and New Orleans either directly from Africa or from the Caribbean ("Estimates Database," 2018).

Jo: So, first of all, it's remarkable that the vast majority of the millions of African Americans in North America are descendants of those few hundred thousand. But here's the thought experiment: imagine if rather than being colonized primarily by English, French, Dutch, Spanish, and Germans who enslaved people from Senegal to Angola, what if North America was colonized by Italian, Greek, and Turkish colonists who enslaved Xhosa and Zulu peoples from South Africa.

Erik: Does this mean instead of crappy Taco Bell we would have better gyros?

Jo: That might be true... But more importantly, malaria is not endemic in South Africa but *it is* in the eastern Mediterranean basin.

Erik: Ah, so sickle cell would be a condition associated with the descendents of the Italian, Greek, and Turkish slave owners, not the descendants of the South African enslaved peoples!

Jo: And *that* is what we mean when we say that sickle cell isn't a black disease. In this thought experiment, we would be thinking of it as a white disease.

Erik: So it's the historical particulars of slavery and then the discovery of sickle cell in the WW1 era United States.

Jim: And you should mention the social and political climate of the WW1 era United States --

Erik: That was the height of Jim Crow, lynching, the rise of the second KKK, and the passage of anti-interracial marriage laws.

Jo: So it's not surprise that once sickle cell was called a "black disease" by medical researchers, it just got baked into the American psyche.

Erik: Now I get it! That's revolutionary. But it takes people thinking about history... even physicians are bad at that.

Jim: That, AND scientists who are convinced of racial hierarchy simply ignore these facts.

Jo: So that's the story about sickle cell. What should we do for the third part --

Erik. --- Wait wait wait. Before we wrap things up -- right at the beginning I asked how old sickle cell was and Jim said there was this enormous range for the age of sickle cell. Like it might be as young as what we think of as civilization. Or much older--22,000 years old. I wanted to ask why the spread was so large.

Jim: It's going to require us to go into the weeds a bit -- you okay with that?

Erik: Well, yeah, I mean our thing is explaining race and science -- so let's throw some science on it.

Jo: Well, if we're tying up stuff that's more in the weeds, I have a question, too. Jim, I remember you saying that you taught that there were five *separate* points of origin of the sickle cell mutation in Africa and Asia?

Jim: That was true ... a few years ago. Actually, to answer both of these questions we have to go back ... back ... way back to 1995. In 1995, America was introduced to the Restriction Fragment Length Polymorphism or RFLP in the O.J. Simpson trial.

Erik: The Juice got loose because no one understood the DNA analysis technique the prosecution used to show that the blood in the footprints leaving the murder scene and on *THE GLOVE* were a one in one million match to OJ. But what does it have to do with sickle cell?

Jim: We called RFLP tests "DNA Fingerprinting" in the 1990s. It was a pretty good way of figuring out similarities and differences at the time, but our genomic sequence analysis has come a long way. Anyway, starting in the late 1970s researchers used RFLP testing to define differences in the genetic sequences near the mutation spot for sickle cell. Right around the time of OJ's trial, scientists had identified 5 different types of genetic sequences.

Jo: Right, that's what you said, that there were five origins corresponding to India, Benin, Cameroon, Bantu or Central African Republic, and Senegal.

Jim: Yep, and the origin of the mutation was thought to be very recent, within the last few thousand years.

Erik: So you're saying that all of that has to be thrown out now?

Jim: Hey, that's science—we have new tools and new evidence and so our interpretations change. So it's exciting that just within the last 13 months two papers have been published using newer genomic analysis instead of the cruder RFLP technique. They suggest a couple of things. First, all of the haplotypes derive much earlier from a single original mutation in Africa.

Jo: How do they know that?

Jim: The older studies found markers around the sickle cell mutation, but they weren't limited to showing up with sickle cell, they also were common with the A gene. In the newer study, the researchers found 20 different groupings that were highly associated with the sickle cell mutation and when they plugged them into a "family tree" type analysis, they showed how all the different combinations are derived from a single ancestral gene sequence (Shriner & Rotimi, 2018).

Erik: So where does the "much earlier" shift from Adenine to Thymine in the DNA come from?

Jim: The oldest direct evidence of sickle cell comes from the genetic analysis of ancient Egyptian remains dating back over 5,000 years. The researchers also used Y-chromosome markers in their sample that have known ages to guesstimate an age close to 10,000 years ago. Finally, their own direct estimations based on modeling the mutations necessary to come up with the surrounding genetic sequences that led to the family tree of the 20 different groups suggested an age of about 7,300 years for the mutation.

Erik: Well, which is it? And, I thought you said something about more than 20,000 years ago!

Jim: That's right. A second study published two months ago looked at sickle cell in African hunters and gatherers and they suggested that the mutation likely occurred about 22,000 years ago. They used slightly different assumptions (about heterozygote advantage selection) to model how long their family tree of different genomes would take to complete. They're suggesting that the mutation and its relationship to malaria pre-dates agriculture, with fatal human malaria showing an age of about 40,000 years in Africa. Both studies propose that sickle cell spread first through central Africa, then to the middle east and India, and finally to southern Europe (Laval et al., 2019).

Jo: And there's that selection pressure in those areas because of the threat of malaria, so the sickle cell gene increased in frequency.

Jim: That's right. Selection due to malaria has pushed very high frequencies of the sickle cell gene in some Greek villages and some parts of India.

Erik: Aha! So that's where your example came from! I can see how that would be good to use in class or when talking to people about why even sickle cell anemia is not a race-based disease.

Jim: Right—usually gets students thinking about how changeable our ideas about race are, especially if they missed that question on the pre-test!

Jo: Hopefully it can change some minds because our listeners spread the word! -- right listeners? Have you had interactions with people over race and health stuff? We want to hear from you about this episode! You can find us on Facebook @ SORPodcast, on Twitter @speakingofrace, and on instagram @speakingofrace.

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