QUESTION #1: How can race cause health inequities if it doesn’t exist scientifically?

TROY DUSTER: Let me begin by addressing a core issue behind the question, which is: if in Brazil, race means one thing, and in the Caribbean, something else, and in North Africa, something else still, how can we study race in any way that’s systematic? If something is so variable in its particular empirical manifestation, then how can we come at this topic adequately?

Well, let’s draw an analogy. The family also means different things in different societies. Sometimes it’s biological – a mother and father with their children, a nuclear family. Sometimes it’s matrilineal descent and matriloclal, where the family lives with the mother’s relatives. It could be patrilineal. It could be lesbian or gay couples that adopted kids.

So, family is extremely variable in different societies, and yet we can study the family systematically. We can look and see whether or not, for example, families of lesbian and gay couples have different access to resources than families that are heterosexual. That means that it’s a local question about how we understand the family and its access to resources.

The same is true for race. In Unnatural Causes we see how infant mortality rates between Black and white women in the U.S. differ. That can be studied scientifically. The film shows, for instance, that stress levels are different for these two groups of people and there is therefore a sort of feedback loop between the stress levels experienced by Black women in America and their biological rhythms and their capacity to have a normal childbirth. Similarly, access to housing loans, access to medical care—all these things can be studied in a systematic, empirical way. Therefore, even though race is defined differently by different cultures, you can study how race operates within a culture and within a particular local circumstance.

PILAR OSSORIO: The way the question is framed, “How can we study race if race isn’t a scientifically valid category?” is odd. Maybe the questioner meant to suggest that race is not a genetically definable or biologically fixed set of categories, as opposed to not “scientifically” valid or real. But there are many things in the world that are real even if they are not defined by genetics or biologically immutable traits. We can still study things even if they are not fixed in our genes.

TROY DUSTER: Hence my example about families. You can study the family as it takes different forms in different cultures.

JAY KAUFMAN: I think most of us agree that race is a social distinction that was created as a way of establishing hierarchies in society, as a way of dividing access to resources. It’s a very significant human invention as a way to structure societies and, as such, is a very important generator of health inequalities. That makes it very real in people’s lives without being a part of the natural world. So, we can study how income affects health inequalities, we can study how access to health insurance shapes inequalities, we can study how all kinds of human inventions affect health inequalities and race is one of those.
**QUESTION #2:** I heard that African Americans have higher rates of hypertension rates because they have a salt-retaining gene. Is this true? If so, does that explain other differences in health?

**JAY KAUFMAN:** The basic answer is that we still don’t know all of the genetic causes of hypertension within individuals. We’re still studying blood pressure regulation and we know very few of the genes involved right now. But the ones that we have identified and that we have studied are not differentially distributed by race in a way that would be consistent with this hypothesis. So the quick answer is that we don’t know very much about this yet, but what we do know suggests this is not true.¹

Nonetheless, this “theory” refuses to die. It has been battered around for decades now and is very popular in some circles and keeps resurfacing despite the fact that there’s no evidence for it. So it appears on Oprah’s television program, it appears in the New York Times Magazine, it appears in all kinds of medical textbooks and all kinds of popular accounts. It has a life of its own, even though there’s really no evidence in favor of it. It’s just a really cute, neat, just-so story.

**TROY DUSTER:** Jay, it might be interesting to say a bit about why it’s so appealing. That it’s the Middle Passage, the fact that certain people came over on slave ships.

**JAY KAUFMAN:** Sure. The idea is a Darwinian one, that there was some kind of selection taking place. The background is that people living in societies based on subsistence agriculture have very low measured blood pressures, very low rates of hypertension, whereas in the industrialized world, we have very high rates of hypertension, epidemic hypertension, especially in African Americans.

So some researchers developed a hypothesis that there was a sort of selective mortality pressure operating during the Middle Passage that would explain the high African American hypertension rates today. There was high mortality on the slave ships, very brutal, cruel conditions, and the idea was that if a subpopulation of African slaves survived because they were able to retain fluid, not dying from diarrhea and other diseases associated with fluid loss, that this genetic predisposition might be selected for, and then their descendents in the New World would have a higher prevalence of whatever this genetic trait was that would cause people to retain fluid.

So it’s a very attractive idea in the Darwinian sense. Alas, there’s no scientific evidence for it. Although a lot of African captives died in these very brutal conditions, there’s really no evidence to suggest that it was because of fluid loss and that the people who survived were selected for their power to retain fluid. Plus, we really can’t see any difference in the genetic predispositions—to the extent that we know about them—between the New World populations and Old World populations.

But the evidence we do have suggests that the prevalence of hypertension among African Americans has a lot to do with the kind of risk factors that we do know about—things like obesity and alcohol consumption and the chronic stress of racism and poverty and eating processed foods. These things differ from society to society, and where those factors are very highly prevalent—as they are in cities in North America—we see high rates of hypertension.

**TROY DUSTER:** One of the graphs I really like from Richard Cooper’s recent study of hypertension across eight different nations showed that Germany has the highest rates of hypertension of any country studied, even higher than American Blacks.²

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JAY KAUFMAN: It’s true. Germany, Finland, Russia, and Eastern European countries have higher rates of hypertension. But you never hear anything about this reflecting something special about the genetic make-up of Germans or Finns. Yet in the medical literature, you frequently see references to some Black exceptionalism, like there’s something different about Black people. So I think it’s a habit of racializing disease that’s very deeply rooted in our medical traditions and the way we teach, but it’s not borne out in the evidence that we have.

PILAR OSSORIO: I want to go back to the question of why this particular genetic explanation for hypertension rates in African Americans as opposed to other Americans. Why is that so appealing?

I think some of it has to do with, number one, that an explanation rooted in the genes or biology rather than society leaves contemporary Americans with less sense of blame, guilt, shame, and responsibility to make changes. Instead, the blame, guilt, shame, etc. fall on people’s ancestors who brought slaves to the New World, relieving us from the responsibility of struggling for racial justice today. It’s just a hypothesis, but I think that has a lot to do with why this same just-so story keeps popping up over and over again and it has such a deep hold.

TROY DUSTER: Of course it’s not just hypertension. We’re talking about everything from diabetes to asthma to prostate cancer. All these health disparities—some of which were discussed rather extensively in Unnatural Causes—have been interpreted conventionally as having a basis inside the body. That allows us to say there’s something different about the biology of different racial groups that makes them susceptible to asthma or diabetes. Or, as Pilar was suggesting, you can start looking for the kinds of forces at play outside the body—toxic waste dumps, patterns of nutritional intake and so on—that have a far more powerful impact upon health outcomes than do these things inside the body.

I’d also like to suggest an additional dimension to this. It’s not only, as Pilar suggested, that genetic explanations are more appealing. But if we can explain what’s going on in terms of the biology of the individual rather than society, then we can somehow go in there and make an intervention with a pill or with a certain kind of scientifically designed strategy at the individual level.3

PILAR OSSORIO: You can make money on medications that lower blood pressure, especially if people are going to be taking that medication for the rest of their lives. But how does a company monetize the eradication of racism and poverty?

QUESTION #3: If it’s really true that wealth equals health, then what about those people that live to be 100 years old? Not all of them are wealthy, and I know plenty of rich people who die young. Isn’t this just genetic inheritance and/or the choices we make or luck?

PILAR OSSORIO: Well, of course there’s always some luck involved. But even after we take into account individual genes and behaviors, at least the ones we can measure, we still see the effects of wealth on people’s health outcomes.

When we measure the impacts of wealth on health, we divide people into different economic groups, and we measure and observe the impacts. And what we measure is the average for each group. And of course some people will have outcomes that are very different from the group average. But on the whole, if you are a member of the wealthier strata, all other things being equal, you’re going to live longer and have better health, than if you were in a lower economic stratum of society.

Now, any time you do something with statistics, you have this problem that of course the statistic that applies to the group doesn’t necessarily apply to each individual person within the group. But overall, all other things being equal, if you are wealthier, you are likely to have better health.

**TROY DUSTER:** *Unnatural Causes* makes a very effective presentation about how even very small differences of wealth levels within populations can have an impact on their health. This goes back to Michael Marmot’s work and the Whitehall studies of British civil servants that observed a continuous health gradient that corresponds with wealth. That aspect deserves to be part of this discussion – that at every level of analysis, a small increment of wealth increases your health.

**PILAR OSSORIO:** Marmot points out that it’s not just wealth *per se* that affects health, but occupational status, power, security, control, features that are usually associated with wealth and class position.

**JAY KAUFMAN:** Getting back to the question about genes though, the important question for health disparities is whether there are differences in the mean for different populations. And there’s no reason to think that any one continental population would have more of whatever genetic trait it is that might allow you to live a longer life. There’s no reason to think that the average for the population of Africa would be higher or lower than that of Europe. We do see variability within a population, tremendous variability. But disparities are about differences between populations. And there’s no consistent evolutionary theory, nor is there any evidence, that suggests that genetic determinants explain any of the mortality differences found between populations.

**QUESTION #4:** Aren’t there diseases that strike specific racial groups and special drugs for Black people with heart disease? How can they get FDA approval unless genetic differences exist between races?

**JAY KAUFMAN:** I’d love to know the answer to that! Because we’ve wondered a lot about the FDA approval process for race-specific drugs. But I should note that there really is only one drug that has gained FDA approval for a race-specific labeling, and that’s a combination of two generic drugs marketed under the trade name BiDil. This was approved in June of 2005 by the FDA just for African Americans for treatment of heart failure.

What happened was, there was an earlier attempt to have this combination drug approved by the FDA based on a study conducted many years ago but that was denied on methodological grounds. So, what to do? Especially with the clock ticking and the original patent running out. And so the study investigators went back and looked at the old data and they saw a slightly larger effect for Blacks than for whites in the original data, and so they decided to conduct a new drug trial that enrolled only Blacks. And in that trial, which involved only self-identified African Americans, the background therapy that it was available to all subjects was different than the previous trials, and the drug was actually found to be quite effective at avoiding a pre-defined set of outcomes. So the company went back to the FDA for approval, but this time only for race-specific approval on the grounds that they had had a trial that only enrolled Black Americans.

But it’s important to note that there was no evidence that there was some physiologic or genetic difference that made this drug more effective in Blacks than in whites. And furthermore there wasn’t even any evidence that indicated that the drug had a differential effect in Blacks and whites since the new drug trial only enrolled Blacks.

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PILAR OSSORIO: I would also add that many people think that this was as much about a marketing and patent ploy as anything else.

JAY KAUFMAN: Right. The manufacturer already had a patent for BiDil. But that patent was due to expire in 2007. But by claiming the drug was race-specific, in this case for African Americans, the company was able to get a second patent, called a “delivery” patent.

TROY DUSTER: And that new patent wouldn’t expire until 2020. So the manufacturers of the drug were very eager for this race-specific approval because of the extra patent protection they already had.  

JAY KAUFMAN: But in this long application that the FDA considered, there was never any suggestion or evidence presented for a genetic difference between Blacks and whites that would make this particular combination of drugs particularly effective in African Americans. There was no genetic evidence presented whatsoever.

TROY DUSTER: In fact, the company even said in their own SEC filings that they were counting on doctors prescribing BiDil “off-label” to other groups as well.

PILAR OSSORIO: The FDA had some choices to make. I don’t think the choice was to not approve the drug at all once the trial had shown some sort of efficacy. The choice was whether or not to approve the drug with some kind of racial labeling, or just to approve the drug, period, which is what they’ve done for all the other drugs that were tested in almost entirely white populations. When they show some efficacy in those situations the drug was just approved; it wasn’t approved with a label that said “For White People.” That’s one of the most problematic aspects of this.

TROY DUSTER: I want to add another dimension to this story. There was also a political context to this. The company that wanted to have this drug approved for Black people was able to enlist the support of the NAACP Northeastern Corridor, Congressional Black Caucus, and the Association of Black Cardiologists. So when the FDA was hearing about the case, they were also hearing about it in the context of what appeared to be a mobilization of Black doctors and Black political figures, who were all in support of this. So I think we have to also look at the political and social context of what happened with BiDil, as well as the actual trial.

PILAR OSSORIO: It’s also important to remember that when there have been claims made that some racial or ethnic or other socially constructed or identified group is more responsive or less responsive than average to a drug, or that some group is more likely to experience side effects when taking a particular drug, it’s quite rare that there is solid genetic evidence for this sort of claim.

But even when there is, keep in mind that we’re usually talking about a version of a gene that is found in all populations. What differs is the frequency of the gene variant. Maybe some variant of the gene is at 60% in one group and 40% in another group and 70% in another group. My point being that we’re not talking about all or nothing effects. We’re not talking about a variant of a gene that is always found in one racial group and never found in another racial group.

And yet the way these claims are presented to the public implies that all people of a certain racial group have a gene that causes them to respond, or not respond, to a certain drug. So a statistical claim about different probabilities turns into a categorical claim, and it’s very misleading because it tends to make

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http://www.sciam.com/article.cfm?id=race-in-a-bottle
people think that all members of certain racial groups have the same gene variant when it comes to responding to BiDil or something else.

Also, even if a drug is approved by the FDA, that doesn’t mean the drug will work for everybody. And there are lots of reasons why some people may respond quite well and others not at all. It might have to do with a particular individual’s genome, a person’s age, a person’s diet, or other aspects of their life or lifestyle that we haven’t yet identified. But it is very clearly the case that an effective drug may not be effective for a particular individual, even if it’s approved for marketing to people like that individual.

**QUESTION #5: Doesn’t the future of medicine lie in identifying and treating the genetic underpinnings of diseases that strike particular groups (for example, Ashkenazi Jews and breast cancer, or diabetes among Native Americans)?**

**PIÑAR OSSORIO:** Many people believe, and I am one, that knowing more about the genetics of diseases may help us better understand the biology of those diseases, and that knowing more about the biology of diseases may help us to develop better treatments, at least in some cases.

But when we start talking about the biology of disease, then I would stress the word “biology.” Biology is much more than just genes. Biology involves a human organism and that organism’s development from the fertilized egg through the fetus and the newborn and its teens and into adulthood and old age. And development isn’t just the playing out of some genetic program that is predetermined and set on one pathway. The development of a person as a human organism is intimately bound up with all of the interactions that that organism has with its environment, whether the environment in the womb or the environment once that person is born.

People tend to lump together “genes and the environment,” but environment encompasses everything from toxic exposures and diet and exposure to violence and a variety of psychic and emotional stressors, especially factors having to do with a person’s wealth and status. All of that gets lumped together into the “environment,” and is often treated as though it’s a kind of epiphenomenon, as though it’s not really important, and yet it’s **crucial** to the actual human biology.

So we could know everything about a person’s genome and still not be able to provide him or her with adequate medical treatment. We have to know about the **person**, and their genome might be helpful in some cases. But the future of medicine doesn’t **only** lie in genetics and understanding the genetics of diseases. There will always be a lot more that we need to know about a person than their genes before we can provide a person with adequate medical care.

One thing I just want to stress here: A lot of times we hear these claims about “personalized medicine,” and yet in the same sentence we’ll hear a claim about how people of a certain racial or ethnic or religious group have a higher frequency of some disease gene. That seems to be the opposite of personalized medicine, treating people only as a certain statistical average member of a group and **not** as an individual.

So we need to pay attention to those kinds of claims and notice them for what they are. Which is, they’re really not talking about individualized medicine if they start going on about allele frequencies in populations and how that’s going to help provide better medical treatment to individuals. We need to know a lot more about an individual than his or her genes to be able to treat his disease effectively.

**JAY KAUFMAN:** Could I add two things? Several of the questions here refer to people reading things in newspaper accounts. One of the problems with medical journalism is that we hear about these claims when they come out, but then several years later when the claim is not sustained or is actually refuted, the newspaper doesn’t often cover that story.
So a couple of years ago there was an article in the journal “Science” about discovering a gene for big brains, and how Europeans had more of the form of this gene that they claimed led to bigger brains. But then two years later when the article was refuted, there was no coverage of that refutation. Or when an article came out in 2002 about an important component of survival of heart failure, a supposed genetic interaction for African Americans, several studies tried to repeat the finding but they were not able to replicate this as a basis for heart failure survival for Blacks vs. whites. Again, the newspapers didn’t cover that. That’s a big problem.

Another aspect of this question is that it says that “there are differences in these populations or racial groups in disease; is there a genetic basis for these things?” When the environments of these groups change, their disease patterns change, yet people still ask the same questions. So, for example, at the beginning of this century, African Americans had a lower risk of coronary heart disease than whites, and there were all sorts of speculations in the literature about “Black” physiology that must protect Blacks against coronary heart disease. By the 1960s, that had flipped and African Americans had higher coronary heart disease than whites. And suddenly everyone was speculating about the physiologic factors that led Blacks to have higher intrinsic risk. But the genetics didn’t change over the course of 50 years; what changed was the environments people were living in. So these serve as important lessons that the health differences between populations (as opposed to between individuals) are much more likely to be due to environmental factors than genetic factors.

QUESTION #6: Why all the attention, focus and money on medicine and genetics?

JAY KAUFMAN: It’s sexy; it seems more “scientific;” it seems like “hard science” as opposed to the “soft science” that sociologists and psychologists do. It’s high profile… It’s the face of big science now.

PILAR OSSORIO: Also, as hard as it may be to come up with a new drug for disease, that’s still a lot easier to do than to change major aspects of our society that affect health, such as the organization of work, the physical structures and organization of our cities and the spaces in which we live. So I think that as hard and as expensive as it is to do genetics work, to many people that seems to be more tractable.

Even so, I’ve still had many physicians say to me, “Yes I understand that we could know everything about genetics and that would still only explain a few percent of the variation of risk for getting a disease.” So even people whose whole life is spent trying to understand and identify and discover genetic risk for disease will recognize and acknowledge that genes play a very small role in how and why someone gets a disease. It’s a very small contributor to the differences between groups, and even to differences between individuals, potentially.

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But then I’ve had people say things to me like, “Well, it’s a lot harder to get people to change their behavior or to get government to change their programs than it is to just give people a pill.”

JAY KAUFMAN: Yet there is evidence that we can change society and those changes can affect health disparities. There’s a study by Nancy Krieger and colleagues showing that the difference between Blacks and whites in premature mortality narrowed following the Civil Rights Movement and the War on Poverty and Medicare. But those gaps began to expand again in the ‘80s, around the Reagan years.

So here’s something that’s very consistent with the idea that our social programs in the ‘60s and the Civil Rights Movement did in fact begin to ameliorate this disparity between Blacks and whites, and that was reversed through subsequent policies. But this kind of evidence is not found in popular media discussions nearly as often as the latest genetic results.

QUESTION #7: It’s all good and well to suggest that we have a shared obligation to one another and that’s why we need to address health disparities. However, that argument doesn’t mean much to my upper middle class white students who fear that the quality of health care they enjoy will suffer, or that they are bearing the burden for other peoples bad choices.

TROY DUSTER: Well, let me back up a little. In the middle of the last century people began to talk about the revolution in genetics. We also began to observe that different racial and ethnic groups had different risks for those diseases. So the litany begins with Tay-Sachs among Ashkenazi Jews and Thalassemia among Mediterraneans, and cystic fibrosis with select Europeans, and sickle cell and so on. Right around that time some of us made the dire prediction that this was going to fracture the public health consensus. And what do we mean by that?

Well, you go back to the beginning of the 20th century, there was really a consensus that smallpox, tuberculosis, typhoid, cholera, yellow fever – all of these things were putting all of us at risk as a society. So there’s something called the common wealth. When seen through this lens, it has an important dimension, which is our common health. So the common wealth was parks and museums and public works and clean water and so on. Now, that argument about the public health, the public wealth—a common wealth—was a lot easier to make in that early part of the century when we begin to put resources into producing a cleaner public space. By that I mean in contrast to the filth and stench of city streets in 19th century England that Charles Dickens describes.

Now, move up to the last part of the 20th century. We all have running water, toilets; the streets aren’t full of feces. Now genetics comes along and fractures that consensus about our otherwise common interest in the health of all members of the society. As I said, we begin to see all these different patterns in different illnesses. So it’s very hard when people say, “Well, my group is not at risk for cystic fibrosis, so what do I care about it?” If it were smallpox, since we’re all at risk, then it’s a public health problem. So the issues around genetics play a vital role in this history.

But while we are mesmerized by some of the high technology around genetics, we can still see developments like TB, which has a new strain now that’s very resistant to drug treatment. If this kind of strain becomes much more common, we’re back to an early 20th century version of what it would mean to have everybody involved in the common public health. When I talk to my students about this, especially my upper-middle class white students, I tell them that they may think that the whole war on health is over in terms of their own group, but in fact lurking behind all of this is the possibility that there could be a strain of TB or other kinds of health issues which, like in the 20th century, could affect all of us.

PILAR OSSORIO: I would add to that, no matter what group of people you study, every group, whether for genetic or non-genetic reasons, has slightly greater risk than average for some diseases. So it might not all be the same disease, but everyone’s at risk for some. We’re all going to have some diseases that need to be studied for which we need additional or new interventions of some kind or another.

JAY KAUFMAN: Well, the questioner actually says that he or she is concerned about paying for the health care of people who make bad choices. And I think that there are people who make bad choices, but there are people who make bad choices in every single group.

PILAR OSSORIO: This sort of individualized mindset goes far beyond issues of health. I think it’s really a huge problem in our ability to conceptualize any kind of collective interest and collective, community well-being or anything else. And that’s a much huger problem that I can’t begin to address….

JAY KAUFMAN: Well, one way to address that is to look at those societies that do have a more communitarian approach to the public’s health. They are spending less of their GNP on health care yet they’re having longer life expectancy and healthier life, less chronic disease, less disability. So this perspective in which people are pooling resources to make sure that there’s not a segment of the population that’s falling off the bottom end of poor health, the whole society is doing much better.

PILAR OSSORIO: One way I have thought about getting at this, in part, goes to the global economy issue. Which is that you need all of your people to be productive, and to be able to do that, they need to be relatively healthy and well-educated. But we’re ignoring the determinants of health and our schools. We’re privatizing them more and more and those that can’t afford them do without and end up on the bottom end of the educational ladder and the health hierarchy. They are not going to be contributing what we want them to contribute to make this country competitive.

TROY DUSTER: That was the exact argument that was used back in about 1845 at the beginning of the whole movement towards public works. It wasn’t made on the basis of health; it was about getting the workers to work longer hours. If they were healthier, they could work better. So everyone agreed, ok, let’s put in running water and an underground sewage disposal network.

PILAR OSSORIO: Jay mentioned that other countries have better health outcomes on a lot of very important measures despite spending less on health care. But those same countries also tend to have far less inequality in wealth, and the people at the bottom aren’t nearly so far down compared to the top. And if you measure wealth not just as a function of what the individual owns, or what she earns, but also what resources she has access to, then those people in some of the European countries and even some countries in Asia, Taiwan would be an example, they have access to a lot of wealth through government services because of this idea of a collective good that we don’t have.

So part of the reason they might not spend as much on health care is not just a better organizational structure for health care delivery, but also because they don’t have these vast inequalities that seem to have a really bad impact on people’s health. So maybe they don’t have people entering into the system with as severe health problems as we have.

QUESTION #8: Should doctors take race into account when treating their patients?

PILAR OSSORIO: Doctors should treat each individual patient. A doctor-patient relationship is a dyadic relationship. It’s not a relationship that has to do with groups of people. It’s those two people. So to the extent that race is part of an individual’s lived experience, it may be perfectly appropriate for a
doctor to do that. But there are lots of ways in which race might come into play, and one of the worst ways a doctor could use it would be to infer what somebody’s individual genotype is likely to be.

So taking race into account doesn’t just mean that you take somebody as an instance of a particular racial group. If it’s done, it needs to be done in the context of what the lived experience of race means for that particular person.

TROY DUSTER: The same is true for ethnicity. If you look at the two, ethnic and racial categories, under what conditions might you take them into account? Well, if you’re in Minnesota, and you know about the migration of Scandinavians, then it’s quite legitimate to think that perhaps hemochromatosis in this population would be higher than in south Texas. So your knowledge of that history of migration, a little bit of sociology, culture, and geography, would come in handy in terms of racial and ethnic categories. That doesn’t mean, however, that you think in terms of “white,” because people in rural Texas are white as well; they just didn’t come from Scandinavia.

JAY KAUFMAN: Although I agree with the logic there, unfortunately most of our empirical evidence suggests that physicians don’t actually handle those distinctions very effectively. What we often see is a kind of categorical thinking, where we say, for example, cystic fibrosis is more common in European Americans than it is in African Americans. As a result cystic fibrosis in African Americans often goes misdiagnosed because there’s this kind of categorical thinking: well, if you’re Black you can’t have this disease. And that does a tremendous disservice to patients.

So in most of the empirical studies that look at the physician-patient relationship, we see that race plays a role that puts the patient at a disadvantage. There’s a nice study by Kevin Schulman\textsuperscript{12} where he had actors read a case presentation to see if they would be referred for right heart catheterization. The script that the actors were reading was exactly the same. Whether the actor was black or white or man or a woman, they were reading exactly the same words. The study asked the physician, in addition to whether or not the patient should be referred for treatment, to rate the patient on a variety of other characteristics. And the African Americans were rated to be of lower socioeconomic class, less likely to comply with their recommended therapies, less likely to show up for their next appointment… all sorts of significant differences between the patients in terms of how intelligent they were and how nice they were and all sorts of things about the patients that were completely out of the physicians’ imagination or stereotypes. And those are the problems with taking race into account in the doctor’s office.

QUESTION #9: If you could pick one thing to change in order to improve health outcomes what would that be?

TROY DUSTER: Jay already addressed this when he talked about how in different societies, reducing the wealth gaps and assuming a collective responsibility for health are probably the most important things in terms of reducing health disparities. So if I could pick one thing to change? Income distribution.

JAY KAUFMAN: That’s a big one!

PILAR OSSORIO: Maybe the tax structure.

http://content.nejm.org/cgi/content/brief/340/8/618
JAY KAUFMAN: But all those things are related to the degree to which we take collective responsibility for health and well-being in a society rather than just leave each to his or her own. And, again, it’s more than just changing a policy; it’s changing an attitude, changing our system of health care, changing our system for providing a lot of basic services, and it requires a real different way of thinking. It’s more about us all being in there together.

QUESTION #10: Name three things every person can do to work towards health equity.

TROY DUSTER: You know, it seems to me that the way in which this gets sorted is as an individual actor. But there’s a way individuals can become part of something called a social movement, or an insurgency of collective actors. So, individuals shouldn’t just be saying, “What should I do, maybe I can get more exercise, I can eat healthier.” But if individuals can see themselves as part of a collectivity that’s trying to change policies, that would seem to me a first and major step.

I can give you an example. It’s right there in *Unnatural Causes*. The difference between what happened in Richmond, CA, and the example in Seattle where people could and did come together in Seattle to make this change in their neighborhood. It wasn’t as individuals; it was as a collectivity.

PILAR OSSORIO: Individuals can—and should—improve their own health behaviors. But we have data suggesting that doesn’t happen very often. But to improve the healthcare system, or to improve the way our cities are being built or the way our buildings are being built, that requires people coming together and making collective decisions and that means engaging in the political process.