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The risky gene: epidemiology and the evolution of race

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ABSTRACT Alcabes examines how modern epidemiology views race, including a historical review and a critique of methods. Race occupies a central place in health discourse partly because of two misapprehensions. First, contemporary epidemiologic research focuses on individual behaviour as a determinant of disease risk, despite the fact that risk as predicted from epidemiologic data is solely interpretable for large populations, not for individuals. Historically and today, connections between behaviour and disease have been used to fortify racial discrimination, with reprobate behaviours imputed to unwanted races. Second, the advent of evolutionary theory and the recognition that genetics provides a mechanism by which evolution proceeds together have allowed for the social concept of 'race' to be imbued fallaciously with biological determinants. With an epidemiology based on behaviour, a behavioural indexing of race and a racial interpretation of evolutionary theory and genomics, the circle closes: society can ascribe differences in disease occurrence to the genetic make-up of the sufferers or to their behaviour, or both—that is, to race—and avoid having to address social problems that typically underlie disease risk.

KEYWORDS behavioural medicine, epidemiology, ethics, genetics, history, race

The United States is the only industrialized nation that does not collect health data according to economic class.[1] Instead, we report almost every morbidity and mortality statistic by race. Yet, to elucidate how race figures in American epidemiology is a daunting task. We epidemiologists have yet to define what we mean when we examine 'race' (or 'ethnicity', as we usually call it now). And the epidemiologic literature is hot with controversy over how to handle race in health research.

While it would be an oversimplification to declare that there are only two positions in the debate about health and race, two core diametrically opposed positions are clear enough: one side holds that only if health researchers cease to keep track of race can the United States move past

1 Vicente Navarro, 'Race or class versus race and class: mortality differentials in the United States', in Dan E. Beauchamp and Bonnie Steinbock (eds), *New Ethics for the Public's Health* (New York and Oxford: Oxford University Press 1999), 39–44.

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Black–White polarization.[2] The other contends that data *must* be arrayed by race because the field of public health must seek to understand how racism affects health.[3] Much is to be learned from each of the two main positions, and a few commentators have tried to make sense of them in ways that are enlightening. But I see no easy path to resolving the issue of race in health research.

Instead, I hope to show the central place that race occupies in American discourse and that discord has been shaped by two misapprehensions pertaining to disease. First, we have misread the epidemiologic concept of *risk*, thinking it applies to individual behaviour. Second, we have misread *evolution*, thinking it means that some genes are better than others. The concurrent emergence of both epidemiologic and evolutionary science, about 150 years ago, offered a seeming substantiation of race-thinking just when people in western societies were beginning to consider what to do about epidemic illness.

Risk

The epidemiologic meaning of risk is well defined: it is a probability claim, the odds that a member of a given population—Chicagoans, say—will come down with a given disease in the coming year. I can tell you that the risk of someone in that city dying of pneumonia or influenza in the coming year is on the order of 464 in a million. To epidemiologists, risk is a guess. That is why we usually hedge our bets, by stating risk as a *vicinity*: we use the so-called 95 per cent confidence interval, within which the probability of disease probably sits. I might say that the risk of dying of pneumonia or influenza in Chicago lies *somewhere between* 255 in a million and 826 in a million. I produced those probability estimates in the usual epidemiologist's way, the same way that a good player of horses figures out which horse to bet on: by looking at what happened in the past. In my case, I used data

2 The argument was articulated by Mindy Fullilove in 'Comment: abandoning ''race'' as a variable in public health research—an idea whose time has come', *American Journal of Public Health*, vol. 88, 1998, 1297–8. Others have offered rationales for related approaches, for instance, James Y. Nazroo, 'The structuring of ethnic inequalities in health: economic position, racial discrimination, and racism', *American Journal of Public Health*, vol. 93, 2003, 277–84.

3 Nancy Krieger sets forth the argument in 'Does racism harm health? Did child abuse exist before 1962? On explicit questions, critical science, and current controversies: an ecosocial perspective', *American Journal of Public Health*, vol. 93, 2003, 94–9. Camara Phyllis Jones offers a prescription for how to address racism in 'Invited commentary: ''race,'' racism, and the practice of epidemiology', *American Journal of Epidemiology*, vol. 154, no. 4, 2001, 299–304. See also Nancy Krieger,' Discrimination and health', in Lisa F. Berkman and Ichiro Kawachi (eds), *Social Epidemiology* (New York and Oxford: Oxford University Press 2000), 36–75.

published by the United States Centers for Disease Control and Prevention (CDC), showing that there were 1,300 or so pneumonia-influenza deaths in Chicago in each of the last few years.[4] I calculated the vicinity estimate using probabilities figured out a couple of hundred years ago by the French mathematician Siméon Denis Poisson, based on *his* observations of past rare events.

Any good player of horses knows intuitively—and probably without ever having read David Hume on inference—that the past can never tell you the absolute truth about what is going to happen in the future. Sometimes you bet on the favourite and your horse loses to the longshot. This year's pneumonia-influenza death rate might be higher than I predicted, since nobody can predict exactly what is going to happen in nature. But, over time, what happened in the past is more likely than not to happen in the future as well. So, the player of horses is comfortable placing bets based on his scan of the past-performance charts, and the epidemiologist is comfortable making predictions about risk—stating probabilities, that is—when she has had a chance to observe diseases in large and stable populations over a longish period of time.

But we must also recognize what epidemiology cannot do. Our risk estimation process will not work well for small populations, and it does not work for an individual. Based on the data for the city of Chicago, I certainly would not try to predict how many of the baseball players sitting in the dugouts in Wrigley Field on a given day in July will die of pneumonia in the coming year. People are simply too different one from another to allow us to assume that one group's risk is the same as another's. This is intuitive, too: most of us would not assume that we can skateboard down staircases or over concrete barriers without injury to limb or loss of life just because we see our crazy children doing it.

Since epidemiologists do not always communicate well what we mean by risk, though, what we do transmit is misinterpreted much, maybe most, of the time. The biggest reason for this miscommunication is that epidemiology has turned its attention away from what it is best suited to, namely, studying how the forces of nature, the structure of society and the imperatives of economic systems affect the distribution of disease. This is what epidemiology was invented for. The origins of the discipline lay in the work of people whom we would now call social scientists: William Petty, William Farr and William Guy in England, the Marquis de Condorcet and Louis Villermé in France, Rudolf Virchow in Germany, and Joaquín Villalba, who coined the name *epidemiología* for the field in 1802, in Spain.[5] These men were interested

4 '121 Cities Mortality Reporting System', available on the CDC website at www.cdc.gov/epo/dphsi/121hist.htm (viewed 18 July 2006).

5 Nancy Krieger recounts some of this history in 'Epidemiology and the social sciences: towards a critical reengagement in the 21st century', *Epidemiologic Reviews*, vol. 22, 2000, 155–63.

in applying quantitative assessments of disease occurrence and vital data to discern how to reshape society so as to make citizens healthier. In contrast, epidemiologists nowadays spend much more time and energy studying how people *behave*.

Behavioural epidemiology

Today is the heyday of behavioural epidemiology. Eat too much and you can become obese. Smoke and you raise your chances of dying of cancer. Have sex without a condom and you might die of AIDS. Life seems thick with risk, some of it still waiting to be detected by a passing epidemiologist. What did you eat for breakfast? Do you buckle up?

An important reason for this shift has to do with a change in what sociologists call the production of scientific knowledge. While the industry of epidemiology strives systematically, scientifically, to generate knowledge, it is no longer in order to learn about the social inequities whose capacity to sicken the populace alarmed epidemiology's nineteenth-century inventors. Now the people who generate such knowledge are the activists and advocates, spokespersons for one or another identity group, or oral historians. Knowledge about how economic mechanisms (the minimum wage, pharmaceutical pricing, 'workfare' in lieu of welfare payments, the male–female wage gap) and social structures (good public schools, integrated neighbourhoods, adequate childcare) link to the health of the public is more a matter of lore, or speculation, than epidemiology. The ten issues of the *American Journal of Epidemiology*, the premier professional journal in the field, that appeared from July to December 2004 contained exactly five research articles on how such structural determinants relate to population health (of which four concerned the control of communicable disease epidemics). But the same ten issues contained thirty-seven research articles on behavioural disease risk.[6]

Is American epidemiology turning away from its original *raison d'être* in order to join in the pursuit of a new American order in which those inequities, like poverty and housing discrimination, that are easily seen to be causes of health problems, are to be ignored? By persuading people to focus on their own behaviour, are we epidemiologists helping them to turn their attention *away* from the pressing threats to health of the modern era: astronomical unemployment among urban youth, the warehousing of the homeless, the withholding of medical care from a quarter of the US

6 The remaining 62 of the 104 research articles published in that period concerned either methodological problems or analysed the relation between individually measured non-behavioural covariates, such as genetic type or serologic markers, with individually measured outcomes.

population by denying them health insurance, and the neglect of public housing (or not building it at all)?

No epidemiologist can tell you what your risk of disease is, based on your behaviour. The first reason is because, as I mentioned, risk does not apply to individuals but only to populations. If you want to know what you should do for yourself, you should ask your mother, not an epidemiologist. Second, behaviour is inherently idiosyncratic and constantly varying: even if we were to assemble a group of people who all ate a high-fibre diet every day, we could not be sure that that group's colon cancer rate would be a good basis for predicting the risk of colon cancer for any other group of fibre-eaters. Which is exactly why you hear so many conflicting stories about whether or not you are supposed to be eating broccoli and Grape Nuts.

Still, the thrust of epidemiology lately goes towards calculating the risk allegedly associated with particular activities. Most of the people I know who do this sort of work are well-meaning: they genuinely believe that they will help make people's lives better if they can demonstrate that those who drink sparingly live longer than people who drink heavily, that smokers who quit are less likely to die of lung cancer or heart disease than those who continue smoking, and that everybody is better off using condoms. Probably they are right, in many cases: it is almost always true that smokers will be healthier and live longer if they quit. Epidemiology is valuable because it points the way to reducing disease by modifying the factors that make disease more likely. But what my colleagues do not realize is that their work also supports the delivery of health-risk-avoidance messages to the public in terms that are heavily moralistic. Their work contributes, albeit indirectly, to a new set of pariahs in our society: smokers, women who drink coffee while pregnant, unhelmeted motorcyclists, people who have 'unprotected' sex. So, while it is true for most people that not smoking is better than smoking, and doubtless safer to wear a helmet if riding a motorcycle fast, I must emphasize that epidemiology has very little to say about whether *you* will be better off if you engage in these 'safe' behaviors. Or any individual. Epidemiology can tell you what is likely to happen, but you must understand that that is only a guide, based on the assumption that you are very similar to other people, and that it is always more likely that the predicted event—injury, illness—will not happen to you than that it will. Perhaps the echoes of old-fashioned race accusation are audible in these new moral messages, and in the ostracism that results when people ignore them.

Epidemics and race

In the era of the industrial revolution, cholera was epidemic in the crowded cities of England and the United States. There were 52,000 deaths in England

in the 1848–9 epidemic,[7] with as many as 1 or 2 per cent of the population in some impoverished riverside neighbourhoods dying in just a few months. In New York City, cholera killed over 5,000 in 1849.[8] Both liberal social reformers and Christian moralists of the day used the disaster of epidemic cholera to promote the causal metaphor of *miasma*: spumes of decayed organic matter allegedly spreading fearsome diseases to people who lived where there were garbage dumps, cesspools and crowds of unwashed bodies. In both London and New York, many of the indigent were Irish. Attributing the cholera epidemic to miasma was a way of saying: *if only the poor would keep cleaner; if only they would stop drinking so much alcohol; if only they would have fewer babies* ... The myths of the day about Catholics in general and the Irish in particular were wrapped up in the miasma theory.[9] It was the logic of victim-blaming: the Irish poor were responsible for their own decimation; they were responsible not because of how economic necessity forced them to live but because of how they acted; and they were responsible not because of how they really acted but because of how the Irish people as a group were rumoured to act.

In other settings where nationality, religion or skin colour was invoked to explain disease disasters, pretty much the same thing happened. The leading killer throughout the nineteenth century was tuberculosis (TB). Here, too, the effects of social structure were displaced on to behaviour, and behaviour was used to define, and then indict, a race. TB is transmitted by the only unavoidable and unmodifiable activity of life, breathing. It is the classic example of a disease whose distribution is determined purely by social conditions. TB incidence is highest where habitations are most crowded and the nutritional supply is the poorest. Unsurprisingly, it was the leading killer among tenement dwellers in the industrial revolution era, and it remains dangerous to the poor in densely populated places everywhere today.

In the United States before the Civil War, TB was responsible for 20 per cent or more of all deaths, and was more pernicious in the industrial cities of the North than in the still agrarian South. But, exactly because of its high profile, TB could also be turned against American Blacks. Proponents of slavery seized on the North–South difference in TB mortality rates as evidence that the southern way of life was salutary and, in particular, cited the lower rate among slaves compared to Northern Blacks as evidence that black people were a race apart: the

7 Sheldon Watts, *Epidemics and History: Disease, Power and Imperialism* (New Haven, CT and London: Yale University Press 1999), 198.

8 John Duffy, *A History of Public Health in New York City 1625–1866* (New York: Russell Sage 1968).

9 This argument is put forth, with a slightly different assessment of its place in the development of a moral agenda in public health, by Charles Rosenberg in *The Cholera Years*, rev. edn (Chicago: University of Chicago Press 1987), see esp. chs 7 and 8.

predisposition of the African American towards TB could be contained only through the benign paternalism of slavery.[10]

Urban TB rates increased further in the later 1800s, as industrialization intensified, immigration expanded and more of the poor crowded into already teeming cities. As Georgina Feldberg shows in her superb book, *Disease and Class*, the prevailing response to TB emerged from Jeffersonian prejudices: Americans held the city *per se* responsible for the high disease rates experienced by its inhabitants.[11] Absent the allegedly salubrious effects of fresh air and the morally uplifting access to agrarian labour and outdoor exercise, the thinking went, city dwellers succumbed to illness. Southern writers focused on the imagined licentiousness of city life as tending especially to raise the risk of TB for black people, whom they imagined to be hereditarily extra-susceptible to consumption.

Of course, removing the shackles from black men and women was dangerous in the southern view, even outside of cities. For instance, the chief medical officer of Mississippi, Dr Henry Rose Carter, definitively asserted that Blacks had brought yellow fever to the United States from Africa and continued to spread it when they were allowed to travel and intermix.[12] The belief that people of African descent were both congenitally resistant to yellow fever (and malaria, another mosquito-borne infection that was a common epidemic problem in nineteenth-century port cities) but ready carriers of those diseases turned race into a catch-22: southern Whites used their conviction that black people were constitutionally resistant to infection to justify assigning black men to work on mosquito-infested docks or in swampland.[13] Naturally, the workers did contract the diseases in such settings. The consequent high rates of disease among the black population validated, for Whites, their assumption that black skin betrayed racial inferiority, notwithstanding the earlier assumption of resistance. And it let white Southerners blame black people's behaviour for transmitting yellow fever and malaria to Whites. Even as they held that black susceptibility to disease was a matter of racial predestination, Southerners argued that the *behaviour* of the black man had to be controlled in order to prevent disease spread.

In our own time, higher rates of both AIDS and heart disease among black Americans have been attributed to habits: African-American AIDS is imputed to the higher number of sexual partners that Black survey respondents admit to, compared to white respondents, to drug injection (although the vast majority of American heroin and cocaine users are white),

10 Georgina Feldberg, *Disease and Class. Tuberculosis and the Shaping of Modern North American Society* (New Brunswick, NJ: Rutgers University Press 1995), 19–35.

11 Ibid., 29.

12 Andrew Spielman and Michael D'Antonio, *Mosquito. A Natural History of Our Most Persistent and Deadly Foe* (New York: Hyperion 2001), 64.

13 Watts, *Epidemics and History*, 215.

and to a rumoured propensity for anal sex. Heart disease is supposedly a function of diet, or an eating 'culture' that disdains low-fat foods, or hip-hop advertising campaigns by McDonalds. Here, too, culpability is fixed by holding African Americans responsible for mythic behaviour: sexual abandon, sexual deviance, primitive eating and dancing rituals, the blood rites of syringe sharing. *If only they could control themselves* . . .

In each of these cases, the victims of disease were seen as having brought it on themselves, through some activity rumoured to be characteristic of their group. Aspersions cast on behaviour are magnified by linking the behaviour to a fearsome disease. And the disease thereby helps to identify people as members of the reprobate group. Double whammy: if you are Irish you must drink too much and have too many babies and you are contributing to the spread of cholera; if you get cholera you must be Irish.

The unspoken predicate of the race accusation—the *then* to follow all those *if only*'s—has always been that we would *all* be healthier. Disease culpability takes the accusation that the victims are responsible, through their behaviour, for their own malaise and expands it into a reason for discrimination: *they* are responsible, it says, for *our* malaise, too. So it is nowadays when we accuse young and dark-complected city dwellers of self-destructive (and, usually, immoral) behaviour associated with sex and drug use. It is really an allegation of antisocial intent: society is damaged by the unhealthy activities of 'those people'. The distance between 'us' and 'them' is highlighted by their supposed indiscretions. It makes them a group apart, a race. And while it incriminates them, it exonerates us. The distinction between the members of the inferior race, with their disparaged and harmful habits, and us innocents—the ones *without* race—helps us to identify the implicated.[14] And it allows us to feel that we are 'innocent' victims.

Epidemiology and evolution

I believe that implicating people in the harming of society, through the allegation that their behaviour causes widespread disease, and imagining that people's behaviour expresses their place in a purported hierarchy of races are two harmful forces in public health. And they have much in common, not least an especial historical concurrence. Evolutionary theory and epidemiology appeared at the same historical moment and virtually in a single place. The London Epidemiologic Society was founded in 1850. In 1855 English physician John Snow illustrated the application of the mathematical methods of epidemiology to studying the cause of disease in

14 I have drawn the terminology of 'implicated' from Richard Goldstein's 1987 article about AIDS in the *Village Voice*, and his 1988 article in *Milbank Quarterly*, titled 'The implicated and the immune'.

his definitive 1855 second edition of *On the Mode of Communication of Cholera*.[15] Darwin's *The Origin of Species* appeared in 1859.

The link between epidemiology and evolution was forged almost immediately. The new principle of natural selection seemed to make sense of the differential disease occurrence that the also-new science of epidemiology discerned: as higher disease rates were observed among certain peoples—the Irish in England, Africans and their descendents in the United States—the inference that those people were less fit, obviously lower orders of life, seemed validated. In my view, it was not Spencer's 'survival-of-the-fittest' take on evolution or its social Darwinian spawn that led people to look at race categories as pseudo-species, with a self-evident higher class (Protestant Whites) and equally self-evident lower classes. That hierarchy already existed in the minds of people who saw disease as the product of the iniquitous activities of people whom they despised, usually the poor and dark. What the mid-nineteenth-century coincidence of evolutionary and epidemiologic theory did was to legitimate the perception that differential disease rates revealed an intrinsic—that is, biological—social ordering.

Consider tuberculosis again. Once Darwinian theory had come to enjoy wide acceptance in science and after the causative organism of TB had been isolated by Robert Koch in Berlin in 1882, an evolution-inspired definition of race explicitly entered the tuberculosis picture. The American view shifted to what Feldberg calls the 'seed-and-soil' approach: granted TB is triggered by a bacillus, but the disease only develops if the bacterial seed falls on fertile ground.[16] And one of the leading reasons for imagined 'fertility' to TB was evolution: Blacks were held to be uniquely, hereditarily susceptible as a people. As a race, that is. If you think that higher disease susceptibility is an expression of lesser fitness, then you can place the susceptible group—the people at *high risk*, to use modern terminology—lower on the evolutionary ladder.

In the case of yellow fever and malaria, evolution solidified the slavery-era contention that black Americans were uniquely susceptible to disease, and seemed to provide a reason for the lesser fitness that white Americans imputed to black people.[17] Ronald Ross, who claimed to have discovered how malaria was transmitted, wrote while in Sierra Leone that 'the native is . . . nearer a monkey than a man'.[18] To Ross and like thinkers, what

15 The full text of Snow's book, as well as maps and statistics on cholera in nineteenth-century London, can be found at Ralph R. Frerichs's encyclopaedic website on Snow, at www.ph.ucla.edu/epi/snow.html (viewed 18 July 2006).

16 Feldberg, *Disease and Class*, 42–8.

17 Watts, *Epidemics and History*, 241.

18 Ibid., 256, quoted from Gordon Harrison, *Mosquitoes, Malaria and Man: A History of Hostilities since 1880* (New York: Dutton 1978), 94. Watts says that Ross was not the discoverer of Anopheles mosquitoes as the vector of malaria; Ross took credit for the work of his assistant Muhammed Bux.

American physicians came to call *diathesis*, a mysterious predisposition to disease, arose because Africans were phylogenetically retarded.

Translating a genetic explanation for disease susceptibility into facile racial accusations sometimes has extreme results, as anyone knows who is familiar with the Nazi euthanasia programme that murdered over 70,000 'Aryan', that is, non-Jewish, Germans beginning in 1939. For our purposes here, what is most significant about the German euthanasia programme is why it ended: it was subsumed, in 1941, within the larger-scale extermination of Slavs, Jews, Gypsies and other *lebensunwerten Lebens*, 'worthless life', the racial offenders who, according to the Nazis, were polluting the German *Volk*. As Götz Aly and colleagues explain in their disturbing book *Cleansing the Fatherland*, the euthanasia programme originally was aimed at reducing the German population's susceptibility to disease and dysfunction by extirpating the genetically compromised.[19] But eliminating the socially dangerous gene turned out to be exactly the metaphor needed to rationalize the Nazis' Final Solution to the race problem. I do not mean to suggest that genetic thinking will make such exterminations happen again. But I do see genetic determinism as a modern version of the seed-and-soil metaphor, continuing to influence how people view disease and having clear links to race. Today, our health officials do not talk about 'worthless life'. But they do connect hereditary disease susceptibility to race when they assert that people of African descent are especially susceptible to specific illnesses.

The diseases to which black Americans are supposedly 'predisposed', or 'at higher risk' of contracting,[20] include such widely disparate conditions as heart disease and stroke,[21] hypertension,[22] prostate cancer,[23] asthma,[24] hepatitis B[25] and HIV infections,[26] infant mortality,[27] and low birthweight,[28] to name only a few. We do know what causes hepatitis B infection, and I

19 Götz Aly, Peter Chroust and Christian Pross, *Cleansing the Fatherland. Nazi Medicine and Racial Hygiene*, trans. from the German by Belinda Cooper (Baltimore: Johns Hopkins University Press 1994), 39, 23 and 44ff.

20 See, for example, the homepage of the Office of Minority Health at the CDC, available at www.cdc.gov/omh/AboutUs/disparities.htm (viewed 18 July 2006).

21 US National Center for Health Statistics (NCHS), *Health, United States, 2005*, Tables 36 and 37, available at www.cdc.gov/nchs/data/hus/hus05.pdf (viewed 19 July 2006).

22 Ibid., Table 69.

23 Ibid., Table 53.

24 NCHS, 'Asthma prevalence, health care use and mortality, 2002', available at www.cdc.gov/nchs/products/pubs/pubd/hestats/asthma/asthma.htm (viewed 19 July 2006).

25 Charles Marwick and Mike Mitka, 'Debate revived on hepatitis B vaccine value', *Journal of the American Medical Association*, vol. 282, no. 1, 1999, 15–17.

26 NCHS, *Health, United States, 2005*, Tables 42 and 52.

27 G. K. Singh and S. M. Yu, 'Infant mortality in the United States: trends, differentials, and projections, 1950 through 2010', *American Journal of Public Health*, vol. 85, 1995, 957–64. See also NCHS *Health, United States, 2005,* Table 20.

28 NCHS, *Health, Unites States, 2005*, Tables 13 and 14.

surmise that it is more common in African Americans for reasons that have to do solely with social clustering: a kind of founder effect—in which the people most likely to acquire an infection and most likely to pass it on are those who are socially linked to the individuals who first contracted the infection—tends to sequestre and amplify sexually transmitted and needle-borne infections like hepatitis B within small social groups. But we do not know completely what the causes are of asthma, prostate cancer, low birthweight or a myriad of other conditions. Claiming that African Americans are predisposed to such conditions explicitly attributes disease risk to genetics and implicitly attributes fitness to race.

Race as metaphor

Metaphors for disease risk, like miasma or seed-and-soil, reinforce popular notions of who is better and who is worse. I see three reasons why genetic determinism is a particularly persuasive metaphor when it comes to explaining disease differences. First, genes do seem to explain some diseases quite well. Tay Sachs, haemophilia and thalassaemia remind us of that. Second, and more perversely, genes seem to offer a biological mechanism by which to justify all those suspicions about racially programmed behaviour and risk, and it is a mechanism that does not require that we mention 'race' at all. And third, genetic determinism allows for very simple explanations about cause.

As for the first reason, the recent finding of differences between 'European' and 'African' populations in the prevalence of a gene that confers prostate cancer risk[29] was widely publicized as implying that it explained Black–White differences in prostate cancer rates.[30] However, a close look at the data reveals that the gene that is reportedly twice as common among the sample of 'African' men in the study than among the 'European' men is found in only 8 per cent of prostate cancer cases. When it comes to pancreatic cancer, stroke, low birthweight or the many other conditions whose occurrence is said to be characterized by racial 'disparity' (to use the term of art), the genetic background that causes these conditions to occur is not clear and simple. Here, and with most conditions, the alleged race correlation of disease occurrence invokes the second and third reasons. And it is the latter, the propensity to adopt simple causal explanations that concerns me here.

29 Laufey T. Amundadottir, Patrick Sulem, Julius Gudmundsson, Agnes Helgason, Adam Baker *et al.*, 'A common variant associated with prostate cancer in European and African populations' (letter), *Nature Genetics* (online), 7 May 2006 (print publication in vol. 38, no. 6, June 2006).

30 See, for instance, Nicholas Wade, 'Scientists discover gene linked to higher rates of prostate cancer', *New York Times*, 8 May 2006.

When we say we do not entirely know what causes pancreatic cancer, stroke or low birthweight but that black people are at higher risk, we are saying that we are ready to accept a particular simple causal narrative. Whether we call it 'heredity', 'race' or 'ethnicity', we are saying that something about those people confers differential susceptibility. That simple narrative of cause and effect lets our critical political faculties off the hook. When we accept such simple explanations for disease occurrence we are saying that we do not deem it necessary, as part of the pursuit of better public welfare, to examine the structure of our social arrangements or the economic policies that decree that some people's lives will be straitened and austere while others will live luxuriously. We are saying that we can attribute the higher susceptibility to the things that make *those* people different from *our* people. We do not have to fix the housing, provide the health insurance or reopen the clinics. The problem is in the genes. There is nothing to be done.

Both the epidemiologist's model and that of the geneticist take on extra power when translated into today's science-permeated culture. The modern-day epidemiologist insists that behaviour underlies disease statistics, the molecular biologist ascribes all variation in nature to genes, and the two views together strengthen the race lens through which Americans examine their own culture today.

This lens is a weak one. Even if it had no other faults, the race viewpoint would be suspect because it makes invisible to us the economic constraints—the effects of wealth and social status—that really and irresistibly worsen the public's health. Presumably, many forms of social stratification—not least economic wherewithal—underlie differences in disease rates; to encapsulate in the simple term 'race' the rich explanation of disease rates offered by complex social differences is simplistic. Further, if our desire is to find and then modify the real determinants of disease, the race lens is also beside the point.

It is a dangerous lens to use, too. Constructs like 'race' freight scientific theories with socially volatile cargo. The end result is to disconnect science from the political agendas and social aims that undergird it. The explosiveness of the race issue forces scientists to take cover in an easy pretense of 'objectivity'. When scientists pretend to be objective, they reinforce science consumers' prejudice that scientific findings are specialized knowledge. And when science is thus reduced to a specialty craft that is carried out in isolation from the ebb and flow of social life, political and economic goals seem disconnected from scientific means.

Given the metaphors about risk and race that epidemiology and evolution have inadvertently provided, people who do not want to face up to the tasks of solving social problems and altering the economic policies that contribute to the disease rates in the first place have some convenient options. They can seek to explain why some diseases are more common in some groups of people and less common in others by assigning the cause to genetics.

Increasingly—especially with what Troy Duster calls the 're-biologization of race' through racial interpretations of human genomics findings[31]—attributing causation to genetics means attributing it to race. Those who claim that differences in disease rates are caused by racial differences in the genome can add credibility to their assertions by using the language of biology and evolution. Besides concretizing the concept of 'race' in a genetically-caused-disease foundation, it is a view that absolves its holders of any responsibility to act.

Or (the other option), in the crazy interconnection of morality and morbidity by which our society explains to itself why some people die and others live, the cause of disease can be assigned to individual behaviour. Identifying behavioural causes of disease produces more investment in yet more campaigns to get people to alter their behaviour and live the healthy life. And that allows us, as a society, to continue to ignore the social problems, economic policies and other forms of social stratification that underlie differences in disease susceptibility.

Race is, often enough, where these two options converge. 'Race' is one of the names we give to discontinuities in disease rates. In the United States, 'race' is often the name we give to such discontinuities when they really come from class. In general, the race allegation refers to deficits in wealth or access to power. But it also, nowadays, resonates with assumptions about fitness. You are part of a race if you are identifiable as a member of the *lesser* classes, not just of the lower strata economically, but of the groups whose disease rates are higher.

We can say that the differences we observe in disease rates arise from genetics, or we can say that the differences arise from engaging in improper behaviour. But in either case what we mean is that those people—the ones who are 'at higher risk', the ones who are 'hereditarily' susceptible—are the *loci* of disease. They carry risk in their genes. They are socially suspect. They are implicated. And since we are innocent, we do not have to solve, or necessarily even examine, the vexing problems of modern society that truly make some people sick while others are well.

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31 Troy Duster, 'Buried alive: the concept of race in science', *Chronicle of Higher Education*, 14 September 2001, B11–12.