Chapter 23

Pre-existing Conditions
Genetic Testing, Causation, and the Justice of Medical Insurance

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Introduction

Ethical controversies regarding the control of science and technology, especially emerging biotechnologies, are among the most contentious of current issues under public debate. Cloning or somatic cell nuclear transfer (even the terminology is controversial) and stem cell research are hot-button political issues, with presidential ethics advisors, presidents and ex-first ladies weighing in, threatening vetoes, or making alliances with strange bedfellows. Every technical advance sets off a new round of discussion and seemingly intractable disagreement. Similar ethical debates complicate public policy decisions regarding the development, regulation and use of genetically modified organisms, gene therapy, and other biological technologies. Research on creating artificial cells and artificial lifeforms is still too early in its development to have reached a comparable level of public awareness, but these areas will also require that we give careful thought to their ethical implications.

Using the term applied ethics for such deliberation is misleading; anyone who has tried to negotiate such practical ethical controversies knows that simply “applying” ethical theory off the shelf is rarely sufficient. At the very least, resolution of ethical questions caused by advances in science and technology will require close collaboration of both scientists and ethicists. Progress on the controversy about human cloning, for instance, may be possible when scientists, ethicists, and philosophers of science use their relevant expertise to sort through the combination of facts and values that is involved (Pennock, 2001). Progress on these kinds of issues may also be easier when we move from a simplistic, generic question about “the morality of X” to a more fine-grained analysis that frames questions in ways that take into account relevant ethical differences between developing just public policies, recognizing professional responsibilities, and respecting and delimiting the
boundaries of personal liberties. In this chapter, I will explore how one may begin to work through such complexities of practical ethics, taking the case of genetic screening technology as an illustration.

As the story is told in newspaper reports from the labs where scientists mine for the gold of genetic knowledge, advances in genetic technology are opening up vast new veins of information about genes that cause disease, bringing us to the cusp of a medical revolution. Spurred by the Human Genome Project, molecular biology has taken on a boomtown mentality. Each week heralds the discovery of yet another purported disease gene. Besides the money being spent by the government, private companies are investing hundreds of millions of dollars to develop genetic tests for disease genes as they are discovered. No one underestimates the vast potential value of the new biological knowledge that is being gained. But the value varies by one’s perspective. Health care professionals, for instance, see this technology as providing new and more precise data, opening the possibility of earlier and more accurate diagnosis, and (eventually) possible cures. For individuals, the possibilities of better diagnosis and treatments are the major values, but they also may simply value the increased self-knowledge and the basis it can provide for life-planning. To businesses and insurers, genetic screening technology is of value in identifying and reducing risks and costs (Waldman, 2004).

However, as in other cases in which a sudden influx of capital enters an economic system, this new wealth of information may drastically upset an established equilibrium. Revolutions always carry with them the dangers of grave injustice during the transition to a new equilibrium, which is of concern from the perspective of public policy. In this case, the new genetic information threatens to upset the balance that so far has held between our (imperfect) knowledge of our health risks and the system of medical insurance upon which we have relied to protect ourselves from those risks.

As tests that can identify genes associated with diseases proliferate, individuals face a new problem: if they test positive for a disease gene they may find that insurers or employers say they have a “pre-existing condition” and cancel or deny coverage, or reject someone for a job on that basis. As I write this, the German government is considering legislation to permit and regulate limited genetic testing for employees in jobs such as construction and public transportation. The question involves how much of a person’s genetic information a potential employer, for example, has the right to know (Tzortzis, 2004). The prospect immediately raised concerns about the potential for genetic discrimination, such as had already been seen under the Nazis and elsewhere. In the United State, cases of individuals being denied insurance by companies because of their genetic risk were among the famous list of cases of genetic discrimination brought to public attention by geneticist Paul Billings (Billings, 1993). A study by the US Office of Technology Assessment in 1992 found that half of all private and non-profit health insurers would refuse coverage to applicants if a genetic test revealed the likelihood of a serious, chronic disease. The same study found that 14 percent of genetic counselors and nurses had clients who had reported having problems about their health
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insurance because of genetic test results (Sanders, 1993). Health care professionals cannot neglect to inform patients of such potential problems when getting informed consent for genetic testing (Conti et al., 2004). In part because of such problems, President Clinton’s failed comprehensive health reform bill had been expected to prohibit the use of any pre-existing conditions clauses (Sanders, 1993). Public concerns about misuse of screening continues to increase, with a 2004 study showing that 92 percent of Americans say employers should not have access to a person’s genetic information, and 80 percent say that health insurers should also not have access (Rovner, 2004). From the insurers’ point of view, however, pre-existing condition exclusion clauses make good sense and it seems obvious to them that when a genetic test reveals that someone has a disease gene this is a proper reason to deny him or her coverage.

This paper will focus on the ethical implications for the future of medical insurance of regarding genes in this manner. Are pre-existing condition exclusion clauses (PECECs) in insurance policies just or unjust? In particular, is it just to deny medical insurance to people who test positive for a disease gene on the grounds that it is a pre-existing condition?

I will argue that we cannot make a general pronouncement about the justice or injustice of PECECs; in certain circumstances they are perfectly just. The first section defends the justice of PECECs for the sorts of conditions that have traditionally been excluded under this heading. However, there are both conceptual and moral problems with excluding people who test positive for some “disease gene” under this rubric, and I will argue that justice requires minimally that people not be denied medical insurance on these grounds. The second section provides the framework for this argument. I present a model of the causal relation – the CaSE model – and apply it to show why it is wrong to consider in general the presence of a particular allele (i.e., form of a gene) as being equivalent to having a genetic disease and why, instead, it should be considered in the same light as environmental conditions. This tells us that the argument of the first section does not apply as a valid reason to deny someone insurance on the basis of a genetic test alone. However, it does not show that it is wrong to charge higher premiums if a gene increases risk of disease and this could lead to a situation in which large numbers of people become genetically uninsurable. In the final section I argue from a Rawlsian viewpoint that, as genetic information proliferates, justice will require a change in the system of health care insurance to protect affordable coverage.

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Originally, when an insurance company declined to insure people with a pre-existing disease condition, that phrase meant that they already had the disease. A miner who was already diagnosed as suffering from black lung disease, say, could...
not then apply for insurance for that condition. Such PECECs made sense in the
circumstances under which insurance organizations arose and on the traditional
model of what an insurance policy is.

The contingencies of the world require us always to act under conditions of
uncertainty, and medical insurance arose as a way to deal with uncertainty about
one’s future health. A health insurance policy allows individuals to take finan-
cial precautions against the possibility of debilitating illnesses and other maladies
without all individuals having to tie up significant portions of their resources saving
for illnesses that might never occur. Some fortunate policy-holders wind up never
being sick a day in their lives and so never collect on their policies, but such people
have no reason to think that they wasted their money. When they bought their
policy they were in the same position as others who bought theirs, none knowing
what the future would bring. For anyone who was acting prudently it made good
economic sense to share the risk with others in a similar predicament.

The advent of mathematical probability and statistics in the seventeenth century,
originally developed by Pascal in response to a gambling problem posed him by the
Chevalier de Méré in 1654, made such decisions under uncertainty more precise.
In 1657 Huygens wrote the first probability textbook, and in 1662 John Graunt
published the first set of statistical inferences based in part upon mortality records
(Hacking, 1990, p. 16). Such data and mathematical tools allowed an actuary to
differentiate people by groups that had differential risks and thus to assign them
different premiums – individuals in groups with lesser risk could then pay less than
those who fell in groups that had a greater risk. Some people would be required
to pay a higher premium than others, but this sort of differentiation seems morally
unproblematic. Prima facie it fits Aristotle’s formal principle of justice, which says
that equals should be treated equally and unequals unequally. Furthermore, it
looks as though differential risk is the relevant property for the case at hand.

If we think of buying insurance on the model of placing a bet in a gambling
game, as early statisticians did and as many people still do, this form of differential
treatment does seem just. If several people are placing bets together on what will
turn up when a pair of dice is thrown, with the winner collecting the whole pot,
then it would be unfair to require the person who bets on snake eyes to pay as
much as the one who bets on seven, since the latter has a higher chance of collect-
ing. Instead, because the latter has a six times greater chance of winning than the
former, fairness requires that his initial bet be six times larger. Once the differential
probabilities of winning are known, mathematical probability lets gamblers cal-
culate what would be fair bets for the different outcomes. Similarly, information
about differential risk of illness (or disability, or mortality) among different groups
allows the actuary to say that an individual in one or another group has a respec-
tively higher or lower chance of “winning” – that is collecting an insurance payoff
by virtue of becoming ill (or disabled, or dying) – and thus to charge them appro-
priately different premiums.

When we consider insurance in this traditional manner it also seems obvious
that the original sort of PECEC was just. To allow John Doe to join the insurance
game and receive a policy for a disease that he already suffered from would be like allowing someone to place their bet after the dice had already been thrown and their number had already come up. In this scenario the PECEC simply prevents someone from being a guaranteed winner. Surely it would be unfair to the other “players” that they pay for those who joined the game only after they had already “won.” It would be like allowing deceased persons’ heirs to sign them up for a life insurance policy post mortem.

How does this bear upon the issues raised by the new genetic tests? In a straightforward way genetic tests reduce uncertainty by providing more information. They allow us to take people who previously would have been classified together in the same risk group and to place them into smaller, more homogeneous reference classes. To ignore the information that genetic tests provide would seem to violate the basic principle upon which insurance works. An insurance company spokesman argues the perspective of the insurance industry this way:

Insurance is sold to provide financial protection against unanticipated loss. If people who know they will die at an early age are allowed by law to purchase insurance, then they are at an advantage not only over the insurer but over all the other policy holders covered by that company. As a basic principle, insurance is priced so that those at equal assumed risk pay equally for their protection. If that is not the case, the price of all insurance must change.

(Lowden, 1994, p. 1509)

Here the spokesman was focusing on life insurance, but the same point applies to health and disability insurance. Genetic tests function like a peek at the cards. Losses or wins may no longer be unanticipated and if the law were to prohibit insurers from denying coverage to people who are privy to their genetic information this would be equivalent to allowing those individuals to legally cheat the odds.

Of course, in most cases individuals learn the results of a genetic test in settings where it becomes a part of their medical records and thus is accessible to insurers. If unfair use of genetic information is going to occur it is thus more likely that it will be individuals who are at a disadvantage vis à vis insurance companies. There are already documented cases of genetic discrimination against individuals. Probably the most systematic case of this occurred in the 1970s after some states began to require genetic screening for the sickle cell trait. The original motivation for screening was to provide family planning aid to people with the trait, but a National Academy of Sciences panel noted that it led to a situation in which carriers of the gene were “denied jobs and charged higher insurance rates without evidence that the trait placed a person at a higher risk of illness or death” (Hilts, 1993). The unwarranted assumption of insurers seemed to be that simply having the gene meant that one had an increased risk.\(^1\) In this case the differentiation was unjust because it involved imposing higher premiums without demonstration of higher risk. However, why would it necessarily be discriminatory to deny insurance to someone who tests positive for a disease gene, assuming that we did have good
evidence that it increases the risks? The insurance company position is that it is not unjust to deny insurance in such cases on the same grounds as before, namely, because the disease condition was pre-existing.

Despite the apparent reasonableness of this position, I want to argue now that it rests on a couple of important confusions in the notions of “genetic disease,” “disease gene,” and “pre-existing condition.” In brief, there is an ambiguity in the notion of a “pre-existing condition”; having a disease gene is not the same as having a pre-existing disease. Furthermore, I’ll argue that there is parity between causal conditions that are genetic and those that are environmental, so that, looking simply at the level of causal interactions there is no reason to say that “the cause” of a disease is “genetic” and not “environmental.” In a trivial sense, every disease may be said to have a pre-existing genetic component. One must bring in pragmatic considerations before classifying a disease as genetic rather than environmental. To make this argument I will begin by introducing some general considerations about the causal relation; I’ll introduce the CaSE model as a framework for representing causal relations and then apply it to the case of genetic diseases.

**CaSE Model of Causation**

Causation is an ontic relation – it takes place in the world and involves physical objects, events, properties, processes, and so on. We must distinguish this ontological aspect of the causal relation from the way we speak about causal relations. When we make causal claims we typically speak of causation as though it were a simple two-place relation. We say things like “Pressing your foot on the brake causes the car to slow down and come to a stop” or “Striking a match causes it to light.” In the world, however, causation is not so simple. The world is a complex web of intersecting causal processes converging one upon another and diverging again at points throughout space–time. There are other important features of the causal relation (for example, that it involves production and propagation, and that it has an important asymmetry, that it licenses certain inferences) but for our purposes here the critical feature is its web structure – multiple causal factors are required to produce an effect or effects, and those factors themselves are effects with multiple causes.

Ordinarily it does no harm to think of the causal relation as we usually do as the two-place relation “C causes E” with only a single factor as the cause (C) and another as the effect (E). In most circumstances, explaining to a novice learning to drive that “Pressing the brake causes the car to slow down and stop” is all that is required to convey the causal principle. However, the experienced driver knows that it takes more than pressing the brake (C) to stop the car (E), for that may not work if the brake pads are worn, the car is heavily loaded, or the roads are slick. When considering the causal relations involved in stopping when in the car,
the driver tacitly takes into account these other relevant factors. Clearly, therefore, more information is required to express the causal relation fully and accurately than is included in the single-factor representation.

A more sophisticated representation acknowledges that it is always a constellation of factors that makes up the causal antecedent. With this approach we may say that the antecedent of the causal conditional contains multiple independent relevant variables (that it is MIRVed), and it is their combined force that produces the effect. In the car case, besides the pressing of the brake, the antecedent \( C \) would have to specify that the brake linings are not worn, that the tires are dry, and many other relevant factors. For the representation to be complete, the antecedent \( C \) would have to contain all the factors that are involved in the production of the effect, including negative factors as in the example. Given the effect that I am interested in, such as my coming to a stop in my car at noon yesterday at the intersection of Fifth and Craig, there is a precise answer to the question of what caused it, but that answer is a complex one and requires specification of far more than the mere fact that I pressed the brake.

Having a MIRVed antecedent is more faithful to the ontic relation in that it recognizes equally all the multiple causal conditions that produce the effect, but the approach has a few notable disadvantages. It is rarely practical, since a specification of all the factors could quickly make the representation unwieldy. It also reduces the inferences that one may draw. And it obscures what often appear to be significant differences between the various factors, such as the difference between a triggering cause and background conditions. Also, it does not do as well as the single factor approach in capturing the way we ordinarily speak of causation.

I propose the CaSE model of the causal relation as an alternative representation that incorporates the virtues of both points of view. To accomplish this compromise the model uses a four-place relation in which the pragmatically highlighted factors of “the cause” and “the effect” are placed in their occasioning context or “situation,” giving us Condition \( C \) in situation \( S \) causes effect \( E \). This is abbreviated in the acronym “CaSE.” The capitalized letters are placeholders for the ontic causal factors and the lowercase “a” stands in for the pragmatic elements (often expressed in terms of alternatives, for example, \( C \) rather than \( C \), or \( E \) rather than \( E \)). In the CaSE formulation all the factors of the MIRVed antecedent that had resided tacitly in the background in the single factor two-place relation are put in the situation “\( S \).” So, for example, if we are talking about the striking of a match \( (C) \) causing it to ignite \( (E) \), then \( S \) would include such relevant factors as there being oxygen present, the match being dry, the air being calm, and so on. When precise specification of the factors is not necessary we sometimes think of \( S \) as representing assumed “standard conditions.”

The CaSE model thus makes explicit that it is actually a combination of factors that causes an effect and it also allows us to isolate a particular factor that is of special interest, as we commonly do in ordinary causal talk. What we label “the cause” from among the multiple causal conditions is a salient factor that we choose to highlight because, for instance, we take it to be the triggering factor in stan-
standard background conditions or because of our particular interest at the time. More generally, from among the multiple relevant causal factors, the one we choose to call “C” is based on pragmatic considerations and may change depending upon the question we ask, or the stake we have in the outcome, or the context of the discussion. For example, we are often interested in causal relations because we desire the ability to intervene and to control outcomes and so will typically cite as “the” cause of some given effect that factor that is amenable to our control. In other cases we are interested in unusual or unexpected factors that have significant effects under otherwise standard conditions. For instance, under normal conditions we say that striking causes the match to light. But if striking the match had taken place in what we thought was an air-tight, oxygen-free chamber we would probably say, not that the striking was the cause, but rather that the oxygen that had entered unexpectedly through a faulty valve was the cause. The ontic causal factors that conspired to produce the flame are the same in the two cases; only which of them are taken as “background conditions” and which is taken as “the cause” changes. This parsing into cause and conditions is thus a function of pragmatics rather than of ontology.

In an analysis of causation it is also common to distinguish between necessary causal factors and sufficient causal factors. In the match example the presence of oxygen was a necessary factor in the relation that produced the flame. Yet, in the second match example, because of the unexpected leak in what was thought to be an air-tight valve, the introduction of oxygen was a sufficient factor in the relation that produced the flame. This division between necessary and sufficient factors is common. Nevertheless we notice that the oxygen was once cited as a necessary factor and another time as a sufficient factor, though in both cases the list of contributing factors was the same. If the necessary/sufficient division reflected some important ontic difference then it would seem strange that this could happen. Again, the CaSE model suggests that this is a pragmatic difference. In the first example by re parsing the situational factors (i.e., by conceptually “holding fixed” the striking of the match as part of S), the necessary cause (oxygen) is seen to be also sufficient relative to the background situation. In the second example, holding fixed the original set-up, the introduction of the sufficient cause (oxygen) is also recognized as having been necessary for the effect. In this way we have just reversed what was illustrated in the original examples; how we place the emphasis is simply a pragmatic matter.

To illustrate how the CaSE model may represent different pragmatic parsings of causal conditions, let us take a slight variation of Hanson’s classic example of an automobile accident at the intersection (Hanson, 1958, p. 54). What caused the accident? In the shop, the mechanics say that the cause was worn brake linings. At the station, the police officers say that speeding was the cause. At town hall, concerned citizens say that a tree-branch that partially obscured the traffic light was the cause. And so on. These persons cite things that were among the necessary conditions in the constellation of causal factors that produced the accident, but which were also sufficient given the “normal” or “default” situation assumed by
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their constituency. Mechanics assume that people may drive fast and that road conditions will vary, and focus on what is within their power to remedy. The citizens recognize that drivers occasionally exceed the speed limit and sometimes fail to have their vehicles in the best condition, and they seek to eliminate unusual environmental hazards that could bring a worst-case possibility into actuality. Shared context of discussion, shared community assumptions, usually determine quite clearly what is included in situation S. When discussants do not share common assumptions it may become obvious in the course of conversation and they usually take steps to remedy the misunderstanding by making their assumptions explicit. On an informal level, therefore, we see that the four-place relation does seem to be implicitly assumed in our causal talk and that it is brought to the fore when speakers recognize that they are operating with different conceptions of what constitutes standard conditions.

To summarize, the four-place CaSE model holds all causal factors as ontically equal for the production of a given effect, while providing a way to recognize the striking feature of causal talk – that of singling out a particular factor as being of special interest. This acknowledges the multiplicity of factors that are involved in the production of an effect and also, by means of the pragmatic element and the slot for placing those factors one takes to be fixed as the background situation, makes explicit the ways we may highlight one or another of the conditions as “the” cause. It also makes clear that our labeling of some factors as “conditions” and another as “the cause” can change depending upon our interests and pragmatic choices, as can whether a cause is thought of as “necessary” or “sufficient.”

CaSE study of “Genetic Disease”

What does this model of causation tell us about the concept “genetic disease” and the notion of a “disease gene”? A quick CaSE study provides the answer. For any given symptom set there are necessarily both genetic and environmental contributing causes. It is not the gene alone, but the gene in some environmental situation, S, that produces some effect. For example, the gene called “patched” is involved in the occurrence of basal cell carcinomas. The gene works by inhibiting cell growth, but if both copies of patched in a single cell are damaged by ultraviolet radiation from exposure to the sun then the cell divides unchecked and a tumor – a basal cell carcinoma – forms (Pennisi, 1996). Furthermore, if we are talking about some particular gene, G, the other genes in the genome would also have to be included in S, becoming, as it were, part of its environment. If G appeared together with a different combination of other genes it might have a completely different effect. The CaSE model helps make it clear that this is as true for so-called “single-gene diseases” like sickle cell anemia as it is for polygenic diseases. In many cases geneticists have no idea what additional factors are necessary for a gene to express itself. For instance, a gene for Hirschsprung’s disease – an intestinal disorder – was recently
found on chromosome 10 that appears to be autosomal dominant with incomplete penetrance; some people with a single copy develop the disease but others do not (Fackelmann, 1993). A disease symptom set is a possible effect, so whether $G$ gets expressed in that way or not will depend upon what happens to be in $S$. These considerations let us draw a couple of important conclusions.

First, it tells us that simply having a particular allele in one’s genome is not the same thing as suffering from a disease. To take an extreme example, it would be absurd to disqualify someone from dismemberment insurance on the grounds that they already had the gene sequence that codes for arms, without which they could never lose an arm. Even having the “chalky bone” mutation that weakens bones and predisposes one to fractures only causes that malady under particular environmental conditions, namely, hard and bumpy ones. Furthermore, there is great variability in the occurrence, severity, and course of most genetic diseases. Someone who gets the gene for neurofibromatosis may develop “marked disability of the nervous system, muscles, bones, and skin, while others will exhibit only minor pigmented spots on their bodies” (Gostin, 1994, p. 126). Some people with sickle cell anemia “are seriously ill from early childhood and others show only ‘minor symptoms later in life’” (Cranor, 1994, p. 131). Indeed, a gene that is neutral or even disadvantageous in one situation may turn out to confer an advantage in another; this is the essence of what it is to be a pre-adaptation. Because of this possibility, critical in evolutionary development, it is important that we remember that it can be misleading to call something a “disease gene” – an allele that causes a disease in one set of circumstances could in theory turn out to confer an advantage in another. By itself, a gene is not “for” anything, let alone some malady, but produces effects only in concert with the other causal factors in which it is situated.

What this means is that we must disambiguate two senses of the term “pre-existing condition.” In the initial argument given in support of PECECs for medical insurance we judged that it was unfair to allow someone to join the insurance pool after they already suffered from the disease (the condition) for which they wanted to be insured. But having the disease condition in this way is quite different from having the causal conditions that predispose one to developing the disease condition. To say that one has a disease gene is just to say that one has a given allele that under certain conditions increases one’s chance of getting the disease. It is true that having a disease gene is a “pre-existing condition,” but this is so in the latter sense (i.e., a causal condition that in a given situation may develop into a disease) not the former (i.e., the disease condition itself), which was the sense used in the insurer’s argument justifying genetic PECECs. Thus, we may not automatically infer that that argument shows that it is ethical to deny someone insurance because they have tested positive for a gene that is a causal pre-condition for developing a disease. The argument uses the term “pre-existing condition” in two different senses and so is not valid because it commits the fallacy of ambiguity. Indeed, as we shall see, if that argument were the basis for exclusion, then no one would qualify for insurance at all unless they didn’t need insurance in the first place.
This follows from the second point we should glean from the CaSE analysis, namely, that all diseases are “genetic diseases” and all are “environmental diseases” as well in that all have genetic causes and also all have environmental causes. It makes sense to settle on one aspect rather than the other only relative to other causal factors in the situation. Thus, whether we think of a particular disease as genetic or environmental depends upon which of the occurring causal factors we consider to be the background situation. Again, this is a pragmatic and not just an ontic matter.4

Of course, in most cases pragmatic factors will likely tend to one side or the other. We judge individual cases relative to what we take to be a standard set of conditions and for ourselves typically we will do this relative to what we take to be the normal healthy bodily state under the normal range of environmental conditions that we live. There are several ways that we might choose to define the normal healthy state – a statistical norm relative to a population, a functional norm relative to our evolutionary history, or a value norm relative to some standard of preferences – that correspond to different theories of disease. If the diseased departure from this state can be traced to a change in the corresponding “normal gene state” then we are likely to call it a genetic disease. If it can be traced to a change in the corresponding “normal environmental state” we are likely to call it an environmental disease.

The story quickly gets more complicated, however, because there may be other features of the situation that may also be of pragmatic interest and thus change which factor we emphasize. We may categorize cases differently depending upon whether and how we can intervene to prevent or cure the disease. Take hemochromatosis for instance, an adult onset disease that looks like end-stage liver failure and carries with it risk of pituitary problems and cardiac failure. The gene that is blamed for the disease is an autosomal recessive. However, because the disease is totally treatable by conventional therapy (phlebotomy to draw off the excess iron store) it is not usually thought of in the category of genetic screening problems, but rather just as a conventional illness. On the other hand, as genetic technologies improve and the promise of efficacious gene therapy becomes more of a reality some scientists are beginning to call a genetic disease anything that could be treatable with some gene level intervention. These alternative classifications work the same way as singling out one or another causal factor as “the” cause of the car accident depending upon what variables of the situation people’s interests take to be fixed and what is under their purview to modify.

There is much more that could be said about the pragmatics of disease classification, but this discussion is sufficient for us to now return to the original question. The foregoing considerations put us in a better position to evaluate the ethical question we originally posed about the justice of PECECs that deny insurance eligibility for individuals who test positive for some disease gene. Is there a moral reason to come down one way rather than the other in fixing the assumed situation and thereby calling a given disease genetic or environmental? For our purposes in considering the justice of exclusion clauses for medical insurance I would argue
that the environmental/genetic distinction makes no moral difference in and of itself. The fallacy of ambiguity that we identified in regard to genetic causal pre-conditions would equally apply to environmental pre-conditions. In neither case is having a predisposing causal condition that could cause a disease the same thing as suffering from the disease condition itself. This showed us that at a merely ontic level there is a parity between genetic and environmental causal conditions. The parity extends in other relevant ways as well.

The most important point of parity involves how changing conditions change risks. Of course, having a genetic pre-condition for a disease, $D$, can make a difference, perhaps even a very large difference, in one’s risk of developing $D$, but this is equally true of environmental causal conditions. Smoking, engaging in certain sexual practices, or living on a floodplain significantly increase one’s chances of contracting various diseases and of dying. There is an exact symmetry between genetic and environmental causal conditions in the sense that either may raise (or lower) one’s risks a little or a lot, depending upon the situation. Thus, if insurance company policy was to have PECECs for any pre-existing causal factor that increased one’s chances of some $D$, then they could insure no one at all. But, of course, this would be absurd since the point of insurance is to take precautions against risks. Driving an automobile, living in a city, or working as a coal miner all increase the chances that one will need medical attention. It would not make sense for an insurer to deny insurance on the grounds that someone had the “pre-existing condition of urban residence” or had already begun working in the mines.

Even in cases in which the risks are extremely high of contracting a disease given some genetic or environmental pre-condition there may be considerable variability in time of onset, and here too there is parity between genetic and environmental factors. In neither case do we know, as we do when a person already suffers from a disease, whether those factors will develop to the point that the person begins to exhibit symptoms and requires medical care. It can be extremely difficult to predict time of onset of a disease because of the extreme variability of genetic disease, even within the same family (Gostin, 1994, p. 126). Even in cases in which the gene could be shown to cause a disease with certainty, with many diseases one still could not say exactly when or even if the onset would occur. A person with the gene marker for Huntington’s or other late onset disease may never collect any insurance for that malady, if only because they may die earlier in an explosion caused by an oxygen leak. Similarly, someone with the human immuno-deficiency virus could die in an automobile wreck before developing any symptoms of AIDS. Even knowing that the effect is inevitable and that it is certain that one could collect on insurance eventually is not in itself a reason to deny insurance because of the variability in time of onset. Everyone is going to die, but that is not a good reason to deny someone life insurance; follow that rule and there would not be life insurance in the first place.

The main conclusion we should draw from this discussion is that genetic and environmental causal pre-conditions are on a par. For that reason, it is wrong to
deny someone insurance by counting having a “disease gene” as a pre-existing condition. We have misled ourselves in thinking that there is something critically different about genetic causal conditions. Probably this is because we have begun only recently to be able to identify and understand genes and we still have only marginal abilities to intervene to control them. Genes still seem mysterious. It has not helped that scientists have done little to discourage the popular press in its tendency to speak of genes as though they alone determine our lives. People have developed an attitude that might be described as “genetic fatalism.” The fatalistic attitude about genes is unwarranted; it is based on a simplistic understanding of the causal web within which a gene is just one factor among many. This over-generalized view of the power of genes can lead people unjustifiably to conclude that testing positive for “a disease gene” is the same as having a disease.

The second conclusion we can draw is that under the current US system of medical insurance based on the gambling analogy of players freely choosing whether or not they will agree to play, it is acceptable for premiums to reflect differential assumed risk, assessed by a genetic test. *Prima facie,* it is the degree of risk and not whether that risk is introduced by genetic or environmental causes that should make a difference in insurance rates. That is, the morally relevant consideration is not where the causal inputs come from but rather what their known effects are and how information about those inputs allows one to anticipate differential losses. In a fair game of chance the price of the bet should reflect the known odds. That is why the case we mentioned earlier of insurers raising rates for people who tested positive for the sickle cell gene was unjust genetic discrimination, because the premium increase was imposed without evidence that carrier risks were indeed higher. It is fair to charge more when risks are higher, but it should not make a difference that the risk is genetic rather than environmental.

**The Future of Medical Insurance**

This leads us to look into the future of medical insurance. One implication of the previous argument is that insurers may charge proportionately higher premiums for greater risks, and we know cases in which the increased risks may be substantial. Indeed, if we maintain a complete parity between genetic and environmental factors, we should even allow insurers to deny insurance to people with a “disease gene,” not by virtue of a PECEC, but simply because the risks and costs (which can be extremely high for diseases like PKU, Parkinson’s or cystic fibrosis) make the policy a bad bet for the insurers. Previously these costs were spread out over everyone who was insured since there was no way to sub-divide the assumed risk group. But with the advent of genetic testing, the new information allows insurers to refine reference classes. Such groups will often be too small to make the premiums affordable to individuals when the costs are divided. With over a thousand
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genetic tests already available and more being developed every day this will lead to a situation in which a large proportion of the population simply cannot afford medical insurance. As developing genetic technology pushes us inexorably in this direction new considerations of justice arise.

Would it be just to maintain the current system of medical insurance in which only the genetically lucky can afford health insurance? Up to this point we have been considering the question of justice within the current institutional structure of health care. As we look into the future we must consider the possibility that the provision of health insurance will have to be reformed. Putting this another way, it may no longer be proper to judge the fairness of insurance on analogy with placing bets in a gambling game as we did before, taking for granted the current rules of the game. Justice may require that we change the rules.

An argument in favor of this view may be made on Rawlsian grounds. Briefly, Rawls tells us to think of justice as fairness, and proposes a framework – the original position (Rawls, 1971, Chapter 3) – for evaluating the justice of social institutions. What institutional and legal structures would we, as free, equal, and rational people, agree to set up and be governed by if we had to make that choice from under a “veil of ignorance”? That is, what structures would it be rational for us to agree to if we did not know in advance which personal characteristics, values, position in society, and so on we would have in that society?

Naturally, under the veil of ignorance we would be ignorant of the specifics of our genome. We would not know whether we would have genes that confer a high degree of disease risk. Nor would we know whether we would be wealthy enough to afford the high insurance premiums concomitant with high risk under the current system of medical insurance. Given that adequate health is a prerequisite for the pursuit and enjoyment of what one values, health care is a basic good, and it would be in our rational interest to see to it that it was guaranteed to all for such conditions. When we consider the justice of health insurance institutions and rules in light of these considerations we see an immediate reason to question the earlier analogy to the game of chance. We cannot chose not to be born and we cannot chose whether or not to roll the genetic dice. Though the current system looked fair initially, it is unfair from a more global perspective. This conclusion connects with another Rawlsian insight, namely, that issues of justice involve compensating for the inequalities of life’s “natural lottery” (Rawls, 1971, p. 74). We would want to be sure that our access to health care was protected, particularly if we should be born with a predisposition for some disease.

This is not to say that we would necessarily decide in the original position in favor of universal health care for all disabilities. However, for genetic risks we have inherited I conclude that it would be in our enlightened self-interest to require an institutional structure that would guarantee coverage.5

What we have seen is that the new genetic information may lead to a situation in which health care coverage is not offered to everyone. These changing circumstances may force a reform of current insurance practices.
Pre-existing Conditions

Conclusion

Once information becomes readily available it will also affect the economy of exchange. New genetic information has already begun to upset the established equilibrium that allowed the current system of medical insurance to work, and something must change if we are to find a viable new equilibrium. Before genetic tests became available, people with a genetic predisposition to some disease still had access to coverage because there was no way to identify their increased risk in advance of its expression; since everyone was in the same state of ignorance no one had an unfair advantage in the insurance game. We are no longer in that situation and, unless we want to leave a large percentage of the population uninsurable, either we will have to change the rules of the game or we will have to start playing another game. As we have seen, this policy issue involves issues of justice and sits squarely in the middle of public morality.

I have not argued here for particular changes in institutional structure. Some philosophers, such as Philip Kitcher (Kitcher, 1996, pp. 135–6), have argued persuasively that in these new circumstances it will be necessary to have universal health care and drop the private medical insurance system entirely, but this is not a foregone conclusion. Perhaps the insurance industry could find a way to offer genetic “as is” health insurance or find a sufficiently broad classification scheme that would allow health insurance to be generally affordable. For this to work it might be that the government would have to institute some industry-wide regulations regarding genetic pre-conditions to maintain fairness for companies. There is already a precedent for such regulations in laws that prohibit differential premiums based on racial classification and legislation along these lines for genetic conditions has already been proposed. Such legislation could be a boon, not just for individuals, but for biotechnology itself (Anon., 1996). Perhaps we would have to move to some mixed system of public and private health insurance. Tom Beauchamp and James Childress, for reasons independent of the genetic considerations we have discussed, have proposed a two-tiered system that would provide a public guarantee of a “decent minimum” of health care and allow for optional private insurance that people can purchase for supplemental coverage (Beauchamp and Childress, 1994, pp. 348–56). Perhaps under the framework of the original position we could decide on a pragmatic norm that could be used to classify diseases and disease pre-conditions. For example, relative to a standard situation, we could agree that diseases resulting from inherited factors should be covered by universal insurance whereas conditions arising from environmental risks that one takes on by choice should fall under the traditional form of insurance.

These and other possibilities will require serious deliberation as we adjust to the availability of new genetic information. In the end, personal, professional, and public ethical perspectives have to work together. Scientists and health care professionals must be aware of both their research duties and their clinical duties.
As they push the technology forward they must be careful to explain the utility of genetic tests without the loaded "disease gene" terminology, and be sure that patients understand the nature and implications of the information, so they can assess the costs and benefits. Furthermore, a policy must be developed to change (somehow) the rules of the medical insurance game through either legislative or judicial action so that genetic tests are not used unjustly. Minimally, we can conclude here that the practice of denying health insurance to people with a genetic predisposition for disease on the grounds that these fall under PECECs is improper. Justice requires that the current system of health insurance be modified in light of these changing conditions.

Notes

1 In fact, we know that the sickle cell gene has a heterozygote advantage in conferring resistance to malaria, so in some circumstances having the gene actually lessens one's overall risk of ill-health.

2 Of course, we are here speaking not of causes as logically necessary and sufficient, but as physically or productively so, since we are still dealing with ontic causation.

3 As a first approximation we might think of $S$ as a *ceteris paribus* clause – $C$ causes $E$, *other things being equal*. However, $S$ need not include all other things, but just a restricted subset. Specifically it should include those factors that make a difference to, and thus can be taken to be significant in, the production of the effect. The fact that the car in the accident had brake linings that were worn made a difference. The fact that the driver had on a vest with worn lining did not. The former factor gets included in the specification of $S$. The latter is irrelevant and is omitted. *Situation $S$ should thus include all and only those factors that are causally relevant (that is, actually make a difference) for the production of the given effect $E$, relative to the factor of interest, $C$.*

4 Other philosophers have mentioned that there may be pragmatic reasons for picking out a gene in some contexts as "the" cause of a disease. For example, in his useful article on genetic causation Carl Cranor writes, "The fact that a complex set of conditions is sufficient to produce an event does not detract from drawing attention to one of the contingencies as 'a' or 'the' cause for certain purposes. What matters is the context and the purpose and that we do not lose sight of the complexity of the processes involved" (Cranor, 1994, p. 131). What the CaSE model shows is that pragmatic partitioning of causal factors is a general feature of the causal relation that must be recognized for all causal claims, so its application to questions having to do with "genetic diseases" is to be expected.

5 Interestingly, Alexander Lowden, who had argued for the insurance companies' perspective, appears to accept this idea. His concluding argument against legislation that would have limited insurance company's use of test information is that such legislation "will add to the cost of a product that should be available to all" (Lowden, 1994, p. 15).
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References

Hills, P.J. (1993), Genetic testing can lead to discrimination, science panel warns, *Austin American-Statesman*, November 5: A16.

Suggestions for Further Reading


