Are Pre-existing Condition Exclusion Clauses Just?

Lessons from Causal and Ethical Considerations Regarding Genetic Testing

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THE CURRENT CONTROVERSY

One of the key features of the Patient Protection and Affordable Care Act—the major health care law that President Obama signed on March 23, 2010—was a reform of private health insurance, especially with regard to coverage of individuals with so-called “pre-existing conditions.” The term refers to a health condition that a person has prior to applying for a new health insurance policy. A study by the Department of Health and Human Services estimated that, depending upon the definition used, between 50 to 129 million non-elderly Americans have some type of pre-existing health condition. This represents between 19% and 50% of non-elderly Americans, and of this group up to 1 in 5 is currently uninsured (HealthCare.gov 2010). When an insurer determines that a person applying for a policy has a pre-existing condition, it typically will charge higher premiums, set limits on benefits, or simply deny coverage entirely. The new rules regarding pre-existing conditions in the Affordable Care Act would put an end to such insurance practices by 2014, by which time a system of health insurance exchanges would be implemented and available for all individuals and small businesses. In the interim, individuals who had been denied coverage by an insurer because of a pre-existing condition are able to make use of a special pre-existing condition insurance plan (PCIP). The provision to ban insurance discrimination based on pre-existing conditions was a major centerpiece of the Act, and President Obama especially focused on these in his remarks at the bill signing, emphasizing how they exemplified the “core principle that everybody should have some basic security when it comes to their health care” (Obama 2010).

However, no sooner had President Obama’s signature been set than political opponents of the Affordable Care Act began work to undermine it. Republicans pledged to repeal, overturn, or at least gut substantive provisions from the Act. More than a dozen attorneys general, again mostly Republican, immediately filed suits charging that the law was unconstitutional (Stolberg & Pear 2010). Most of these were subsequently dismissed or had their arguments rejected, but as of this writing two federal judges (in Virginia and Florida) have ruled that the so-called “individual mandate” of the Act, which requires individuals to maintain “minimum essential coverage” of health insurance, is unconstitutional. The constitutional challenge to the individual mandate and to the related question of whether it can be severed from the Act will likely have to be resolved by the Supreme Court (HFMA 2010). Insurance companies also challenged the Act, specifically targeting the pre-existing condition provisions. In the days leading up to the bill signing, President Obama had emphasized that “Starting this year, insurance companies will be banned forever from denying coverage to children with pre-existing conditions,” but some insurers argued that the fine print of the Act only said that they had to cover pre-existing conditions if they chose to cover a child, but that it did not require them to write insurance for someone with a pre-existing condition until 2014 (Pear 2010). Many insurers must hope that the Act will be repealed by then, which Republicans again vowed to do following their retaking of the majority in the House of Representatives in the
November 2010 congressional election (Fox News 2010). In January 2011, the House voted to repeal the law, and in April, as part of the budget deal, Republicans required a new debate and vote on repeal in the Senate as well (Fox News 2011).

With this issue of guaranteed medical insurance and pre-existing conditions once again at the center of political debate, this is an appropriate time to revisit one special class of pre-existing conditions—those identified by genetic tests. This particular issue had become especially pressing by the turn of the millennium because of significant scientific advances in the 1990s, in part the result of the Human Genome Project. It seemed as though each week brought news of some new study that found a possible genetic link to some disease. As tests that could identify genes associated with diseases proliferated, individuals faced a problem: if they tested positive for a disease gene, insurers would say they have a pre-existing condition and cancel or deny coverage on that basis. In the United States, 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 U.S. Office of Technology Assessment in 1992 found that half of all private and nonprofit 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% of genetic counselors and nurses had clients who had reported having problems about their health insurance because of genetic test results (Sanders 1993).

From the insurers’ point of view, so-called pre-existing condition exclusion clauses make good sense and it seemed 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.

In an earlier paper (Pennock 2006), I focused on the ethical implications for the future of medical insurance of regarding genes in this manner. Are pre-existing condition exclusion clauses in insurance policies just or unjust? In particular, I considered whether it is just to deny medical insurance to people who test positive for a disease gene on the grounds that it is a pre-existing condition. This chapter recapitulates my previous analysis that focused on some key causal and ethical considerations regarding pre-existing condition exclusion clauses for pre-existing genetic conditions, and then asks what light that special case may shed on the current debate about pre-existing conditions generally.

I argue that we cannot make a general pronouncement about the justice or injustice of pre-existing condition exclusion clauses; in certain circumstances they are perfectly just, in others not. In the second section of this chapter I defend the justice of pre-existing condition exclusion clauses for the sorts of conditions that have historically been excluded under this heading given the traditional conception of insurance. However, there are both conceptual and moral problems with excluding people who test positive for some "disease gene" under this rubric, and I argue that justice requires minimally that people not be denied medical insurance on these grounds.

The third section presents a model of the causal relation—the CaSE model—that provides the framework for the argument. In the fourth section, I apply the CaSE model to the case of “disease genes” 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 second section of the chapter 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 next section of the chapter, I argue from a Rawlsian viewpoint that ethical considerations of justice require the elimination of pre-existing condition exclusion clauses for pre-existing genetic conditions. The final section of the chapter returns to the current debate and discusses what general lessons may be drawn from the resolution of the genetic case.

**PRE-EXISTING CONDITIONS**

Historically, 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 pre-existing condition exclusion clauses 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 financial 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 policyholders 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 17th century, originally developed by Blaise Pascal in response to a gambling problem posed to him by Chevalier Meré in 1654, made such decisions under uncertainty more precise. In 1657 Christiana 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 collecting. 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 calculate 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 respectively 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 appropriately different premiums.

When we consider insurance in this traditional manner, it also seems obvious that the original sort of pre-existing condition exclusion clause 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 pre-existing condition exclusion clause 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 life insurance policies 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 also accessible to insurers. If unfair use of genetic information is going to occur, it is more thus likely that it will be individuals who are at a disadvantage vis-à-vis insurance companies. There are well-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.\footnote{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 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 representa-
tion to be complete, the antecedent C would have
to contain all the factors that are involved in the
production of the effect, including negative fac-
tors 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 ques-
tion of what caused it, but that answer is complex
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 disad-
vantages. It is rarely practical, since a specification
of all the factors could quickly make the represen-
tation 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 captur-
ing 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
\textit{Condition C in situation S causes effect E}. This is
abbreviated in the acronym “CaSE.” The capital-
ized 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 fac-
tors 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 \textit{salient} factor that we choose to highlight
because, for instance, we take it to be the trigger-
ing factor in 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 dis-
cussion. 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 unex-
pected 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 airtight, 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 \textit{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 \textit{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 airtight valve,
the introduction of oxygen was a \textit{sufficient} factor
in the relation that produced the flame. This divi-
sion 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 reparsing 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 setup, 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 that were also sufficient given the “normal” or “default” situation assumed by 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 or 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 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 (Fackelman 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 pre-existing condition exclusion clauses for medical insurance we judged that it was unfair to allow people 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 diseased condition itself), which was the sense used in the insurer's argument justifying genetic pre-existing condition exclusion clauses. Thus, we may not automatically infer that that argument shows that it is ethically acceptable to deny people insurance because they have tested positive for a gene that is a causal precondition 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 C&SE 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. 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 a 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 which variables of the situation people's interests take to be fixed and which 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 pre-existing condition exclusion clauses 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, the environmental/genetic distinction makes no moral difference in and of itself. The fallacy of ambiguity that we identified in regard to genetic causal preconditions would equally apply to environmental preconditions. In most cases, having a predisposing causal condition that could cause a disease is not 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 precondition 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 flood plain significantly increases 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 pre-existing condition exclusion clauses 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, and 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 precondition, 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 he or she might die earlier in an explosion caused by an oxygen leak. Similarly, someone with the human immunodeficiency 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 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 preconditions 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 to conclude unjustifiably that testing positive for "a disease gene" is the same as having a disease.

The second conclusion we can draw is that under the traditional U.S. 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 came 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 JUSTICE OF MEDICAL INSURANCE**

These causal considerations may now be brought to bear on some of our questions involving the justice of pre-existing condition exclusion clauses and 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 pre-existing condition exclusion clause, but simply because the risks and costs (which can be extremely high for diseases like phenylketonuria, 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 subdivide 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 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 traditional 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. But looking into the kinds of changes brought about by genetic information, we must consider the possibility that the provision of health insurance as traditionally done is simply no longer ethically (or economically) viable. 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, Ch. III)—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 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 under the veil of ignorance in favor of universal health care for all disabilities. However, for genetic risks we have inherited, it would be in our enlightened self-interest to require an institutional structure that would guarantee coverage.

TOWARDS RESOLVING THE CURRENT CONTROVERSY

In the original version of this chapter I went on to briefly discuss ways in which policies and regulations for health insurance might be modified in light of these causal and ethical considerations, and closed with the following recommendations:

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 pre-disposition for disease on the grounds that these fall under [preexisting condition exclusion clauses] is improper. Justice requires that the current system of health insurance be modified in light of these changing conditions. (Pennock 2006, 421-422)

Less than 3 years later, an unusual political meeting of minds resulted in Congress passing (with but a single dissenting vote) and President Bush signing the Genetic Information Nondiscrimination Act (GINA). GINA prohibits discrimination by health insurers based upon an individual’s genetic information (GINA 2008).

Although it may still be too soon to call GINA an unqualified success, it appears that the ban on pre-existing condition exclusion clauses based on genetic information has been implemented without any notable problems; it is now simply a just constraint that affects all health insurers equally.

Do the causal and ethical lessons we have learned regarding health insurance for pre-existing genetic conditions shed any light on the current ongoing controversy about pre-existing health conditions generally?

At first glance, much of our analysis seems to argue against the policy. The pre-existing conditions included in the Affordable Care Act involve all sorts of straightforward disease conditions (ranging from AIDS and Alzheimer’s disease to kidney disease and leukemia), rather than the causal conditions for disease that we focused upon. Indeed, the Act is expressly meant to cover a pre-existing condition even when a person already expresses the disease. That is a circumstance I had argued was unjust; given the historical model that insurance is like a game of chance, it is unfair for someone to enter the game and place his bet after the die had been rolled. Put in more Kantian terms, one might say that it undermines the premises of the system and makes it unworkable if someone can wait to buy a policy until after he contracts a disease. However, my main purpose was to show how the changing conditions brought about by new genetic information were already undermining the premises of the insurance game— injustice resulted whether insurers could use the “inside information” of a genetic test against individuals or vice versa—and that the then-current system had to change. GINA changed the rules of the game with regard to genetic information in the right direction, and the Affordable Care Act changes the game in an even more significant way.

One way in which the Act changes the health insurance game, besides the already mentioned general ban on pre-existing condition exclusion clauses, is inclusion of the “individual mandate” requiring everyone to purchase health insurance. Ironically, this current main target of opponents of the Act solves the problem that I had pointed out where individuals could use genetic information but insurers could not. That situation would have allowed people to purchase coverage if they knew their genetic risk was high but to skip coverage otherwise, thus taking advantage of the insurance game. Requiring every individual to carry health insurance resolves that unfair asymmetry.
To rule the individual mandate unconstitutional and to sever it from the Act would again produce an asymmetry that would be to the disadvantage of insurers (assuming that the ban on pre-existing condition exclusion clauses were retained), so it is likely that opponents are really aiming to overturn the law more broadly, not just this one point.

The second way that the Act changes the health insurance game is equally basic and is almost certainly the main reason for the Republican opposition to the Act, namely the institutionalization of the idea that health care should be universal. The Rawlsian argument given above showed why it was just to require universal health care for diseases caused by genetic factors, but the Act extends this to all diseases and makes it the law. As President Obama articulated it in his signing speech, the “core principle” behind the bill is that “everybody should have some basic security when it comes to their health care” (Obama 2010).

Such a principle provides a general ethical framework but leaves many issues still to be resolved. The guarantee of “basic security” for instance, may be seen as a variation of Tom Beauchamp and James Childress’ reasoned proposal for a two-tiered health care 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 & Childress 1994, pp. 348–356). Opponents of the Affordable Care Act charged that it was “Socialist,” but in fact the system of insurance exchanges that it sets up is an attempt to forge a political compromise between public and private insurance, and this is not the place to delve into such matters. The pressing ethical question that remains open by Obama’s core principle is the same that was left open regarding Beauchamp and Childress’ concept of a “decent minimum,” namely what “basic security” comes to with regard to health care. Ethicists now need to focus attention on whether there are principled ways to sort out what conditions and what level of health care should fall under this rubric and what should be properly left to individuals on their own.

The CaSE model of the causal relation and causal equivalence of genetic and environmental factors that was discussed above provides at least a few steps forward in thinking about such questions. Both genetic and environmental causes affect one’s chance of contracting an illness and, epistemically and ontically, there is no difference between the respective probabilities. Thus, just as the Rawlsian argument given above showed why pre-existing condition exclusion clauses for genetic pre-existing conditions are unjust, other things being equal, it should give the same conclusion for many environmental conditions. A morally relevant asymmetry remains in other cases. While any pre-existing genetic factors an individual may have are the result of the genetic lottery, many environmental factors are within one’s control. As we noted, whether or not a factor is under our control is relevant to our ethical deliberations in the Original Position; some environmental conditions that cause illness may be beyond our individual control just as genetic conditions are, and we would agree to jointly guarantee health care in such cases. We might reasonably find it fair (and thus just), however, to limit care for health conditions that persons may bring upon themselves by their own risky choices. Get lung disease because of air pollution and be covered as a part of basic health care security, but choose to smoke and perhaps you ought to buy a supplemental private policy or be ready to pay for care yourself. (Such judgments under the veil of ignorance would also reasonably change as a function of expected available resources; when societal resources are plentiful, we might agree to provide a higher level of care, but when they are limited, we might be less tolerant of imprudent, foolish, or negligent choices and guarantee less.) Finally, it behooves us to look into the future just a bit further and recognize that, as science progresses, genetic conditions will also come under greater and greater individual control, so even those original ethical assessments will become more complicated.

Where does this leave us with regard to the current controversy? With some lessons learned from the previous case of pre-existing condition exclusion clauses for genetic conditions and the precedent set by GINA, we may proceed with a degree of optimism. Although chance is involved in substantive ways in these issues, we should not and need not proceed under the assumption that health care must be evaluated under the ethical framework of joining a game of craps. It remains to be seen whether the Affordable Care Act is a politically viable solution to the ethical complexities of providing just health care, but at least it is on the right track. In working to fundamentally change the rules that govern health insurance, it is building on a rational desire and reasonable conclusion of justice that we should not have to gamble on health care.
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 a 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 “Genetic Causation,” Carl Crantor 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” (Crantor 1994, p. 131). What the CSEP 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. 1510).

References