

**"Are Carriers of the Genes for Huntington's Chorea and Cystic Fibrosis Diseased?"** The Hastings Center, Briarcliff Manor, NY, October 1, 1991.

ARE PRESYMPTOMATIC CARRIERS OF  
HUNTINGTON'S CHOREA  
AND HETEROZYGOUS CARRIERS OF CYSTIC  
FIBROSIS GENETICALLY DISEASED?

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by

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**1. Introduction**

Technological advances force redefinition of action-mandating concepts and language through complex social, political and economic tendencies that collectively determine what has been dubbed "the technological imperative." The reverse is also true: redefinition of concepts shapes and guides the direction of technological development through shaping public beliefs and expectations. A powerful and far-reaching example of such occurred with the redefinition of "death" and the concept's transformed relationship to transplantation technology.

The human Genome Project is an organized and accelerated phase in another series of technological advances pressing us to "geneticize" the concept of disease. There is a detectable shift in our identification of diseases, from characterizations of them in terms of their occurrent symptoms and signs, through partial differentiation in terms of syndromes, to identification of them in terms of causal mechanisms, including genetic mechanisms. And, genetical thinking proceeds not only to identify genetically driven mechanisms operative in the production of the disease's symptoms; the identification can move to identifying the disease with the genetic substrate.

The potential for dehumanization of both the recipients of technology and the processes of its delivery, particularly in the area of medicine, is well documented. It is a fundamental assumption of this paper that there is a point to resisting surrender to all the tendencies urged by the imperatives of technology and science, a point that falls in the middle ground between opposing all technology and surrendering to its

directions as inevitable. That middle ground consists, in part, on getting clear on the concepts involved, their roles in determining action, and the processes that alter them. Deliberation in these matters may make conceptual change deliberate, creating the possibility that the worst results of conceptual alteration may be avoided, or at least controlled.

The conceptual question to be addressed here is whether heterozygous carriers of cystic fibrosis and other single locus disorders, or late onset disorders such as Huntington's Disease count as "diseased individuals." I shall address this question in a manner somewhat determined by earlier work done in a similar research group at the Hastings Center in the mid and late 1970s.

## **2. Deontological versus Consequentialist Taxonomic Strategies**

There are at least two ways of proceeding in the development of classifications of things like individuals and diseases. The first, which I characterize as the deontological view, might also be called the essentialist or natural kinds view. This is the view that, in the real orders of the world, we have nestings of levels of entities; these entities manifest organization into systems in a manner that is explained by reference to their essential natures; and their essential natures are to be understood in a reductionistic way in term of component systems of entities.

The goal of taxonomy development, therefore, is to mirror in language the nested layers of natural kinds, so that our understanding of a natural kind, such as human persons, should seek to approximate nature with explanations of one level—the whole human organism—in terms of natural kinds at another level—organs and tissue and their processes; this level yields to explanation in terms of natural kinds at the next level—cells, and so on through nested levels of natural kinds—components of cells, especially chromosomes and genes; base pairs; molecules; atoms; components of atoms, and so forth.

This reductionist taxonomy carries with it the baggage of reductionism: emergence of qualities not explicable by reduction becomes progressively incomprehensible as the reducing level is more and more fully characterized. Further, it carries with it the assumptions of scientism; that the result is true, universally applicable, complete, and, below the level of whole persons' behavior, value free.

The second view of taxonomy construction is either atheistic or a least agnostic about natural kinds. It has been called the Humpty-Dumpty view of language: words mean what we want them to; it is just a matter of who is to be master. There are more or less felicitous ways of constructing language in terms of which we think and speak

about persons and their problems, and an important question always to ask in considering a taxonomic shift is, What is to be gained and lost by it?

Both views would acknowledge that taxonomy has important consequences. The natural kind, deontological view, however, sees capture of The Truth to be the driving value and objective, whereas the Humpty-Dumpty view thinks that taxonomy ought to be in service of human interests, and may even doubt that the notion of The Truth, divorced from human interests, is desirable or even possible.

What is sketched here is a meta-ontology of language, a philosophy of sociology of language. It should be clear that, as language is used in ordinary speech and thought, not only are both types of taxonomy at work, but one or the other may dominate in a given institution, tradition or theory. We may illustrate by tracing one way in which the evolution of the concept of “genetic disease” interacts with common view on the beginnings of an individual human life, to produce a term heavily laden with the potential for stigmatization.

### 3. Genetic Disease.

A movement down the natural kinds ladder, through levels of whole-part explanation, is what we mean by “reductionism.” To call a disease “genetic” signals a reductionist slide to a level of nested natural kinds at which many believe lies the individual human identity--the individual’s genetic substrate or genome.

The late Paul Ramsey wrote:

Genetics teaches us that we were from the beginning what we essentially still are in every cell and in every generally human attribute and in every individual attribute. (Ramsey, 1970)

Ramsey here articulated the view that many, religious or not, have believed: that their individual identities are et by, and to be thought in terms of, their inherited characteristics, which genetic science equate with their genomes.

To speak of a genetic disease, then, is to speak of a defect in your essence, your fundamental substrate that connects your body through time back with your existence before birth, just from the point of conception. Graft Ramsey’s articulation onto talk of genetic disease, and the slide from “individual with a disease” through “individual with a genetic disease” to “individual with a diseased genome” to “diseased individual” becomes powerfully persuasive. On this identification of the individual’s identity with the individual’s genome, we cannot sensibly speak of preventing or

curing—in the sense of eliminating—a genetic disease through gene replacement, genetic engineering, or provision of missing gene products, but only of preventing or eliminating genetically diseased individuals. For individuals don't *have* genetic diseases; they don't *acquire* genetic diseases; they *are* genetically diseased, where that “are” expresses identity, not possession or predication.

The Humpty-Dumpty approach, by contrast, encourages us to think first about the consequences for the interests of real, specific humans, situated in their social, ethnic, religious and professional roles and institutions, of certain ways of speaking about their diseases, just as a consequentialist ethics would urge us to think hard about the individual and social consequences of our possible actions for those they will affect before we choose among options. Further, it discourages universal applications of terms like “genetic disease” to individuals having similar “essential” characteristics because such terms are value laden, and values are functions of the life plans and preferences of people situated in their very different environments and lives.

The following discussions of two “genetic diseases” seek to illustrate ways in which essentialist and Humpty-Dumpty taxonomies yield different results in how those conditions tend to fit into patterns of human thought that are frankly value laden.

#### 4. Cystic Fibrosis

Cystic Fibrosis is the most common of the “fatal autosomal recessive diseases” in Caucasians, with an incidence as high as five per cent of the population. It is characterized by irreversible prenatal pancreatic damage with a resultant deficiency in pancreatic enzyme secretion in about 85 of CF patients, who suffer from a consequent malabsorption of protein. Heavy mucus secretion in respiratory tracts is associated with a proneness to respiratory infection. Typical excessive salt content of the sweat, while usually not of clinical significance, has provided the basis for standard diagnostic tests for many years. Hepatic fibrosis in later life frequently occurs. Bowel obstructions, consisting of tenacious plugs of meconium, occur in up to 20% of newborns with CF.

Life expectancy has increased in CF from a few months or years of the early reports of the disease to 2 or 3 decades, due to advances in clinical management of the disease, including the ability to detect individuals with it in utero or prior to full manifestation of symptoms. Ironically, as mean survival time for these individuals has increased, so has the incidence of diabetes mellitus in that population.

The World Health Organization issued a “Report of a joint WHO/ICF(M)A Task Force on Cystic Fibrosis” in Leningrad and Moscow on 29 November 1990. In it, the facts of the genetic basis for this disease are identified:

The disease is caused exclusively by mutation of a single gene, is inherited in autosomal recessive fashion, and is the most common such disorder in populations of Caucasian origin. Although very considerable progress has been made during the past 50 years in its clinical management, with a corresponding improvement in the mean life expectancy in developed countries from a few months to a few decades, it remains incurable . . . . Consequently, attention has been given to the possibility of screening for carriers of the defective gene—who represent up to 5 in some populations—so that they may be given appropriate genetic counseling . . . . Whereas it was previously possible to identify carriers only when they became parents of affected children, in recent year those carriers who were more distantly related to CF patients could often be identified by means of genetic linkage techniques. (World Health Organization, 1990, p. 4)

The report goes on to indicate that it is likely that differences in the expression of CF, including age of onset, are likely due to different mutations in the gene, which consists in some 27 exons and involves about 25,000 base pairs.

A natural question to ask from an evolutionary perspective is, “Why should such a dreadful disease occur?” The explanation o, the occurrence and spread of such a condition is speculative, but it will provide us with some additional salient considerations relevant to the question of our paper.

Darwinian evolutionary explanation holds that mutations arise spontaneously and are selected for or against in terms of their survival value. We would thus expect that a widespread trait such as that for CF would convey some significant benefit to account for its being selected. We know, for example, that a single occurrence of the gene for sickle cell anemia confers significant reduction in susceptibility to malaria (cf. Murray, Jr. 1974), so that we might envision the gene’s proliferation in a population in which malaria is endemic: the survival of carrier offspring due to malarial resistance selects for the gene despite the high fatality rate of the one child in four who inherits the double dose and is thus susceptible to severe sickle cell anemia.

Not surprisingly, the single gene for CF conveys a high degree of tolerance to cholera, possibly by a partial phenotypic expression in thickened meconium secret

ions, countering the diarrhea and consequent dehydration characteristic of the disease. The explanatory hypothesis, then, is that the CF gene was selected for during periods of pandemic cholera, conveying to carriers a greater resistance to episodes of diarrhea and dehydration, and giving them a subsequent reproductive advantage over the population not carrying the gene.

### **5. Huntington's Chorea**

Huntington's chorea is a lethal degenerative disease caused by a single gene. The name comes from the first person to identify it as a heritable syndrome, and from the Greek root for such words as "choreography," a root meaning "to dance." Symptoms typically begin between ages 30 and 50. Minor motor discrepancies are progressively followed by increasingly severe loss of fine movement, then of major movement control until the sufferer becomes bedridden. In the later stages of the degeneration, emotional and cognitive functions become involved, with characteristic shrieking, mirthless grinning, and laughter; death occurs 10 to 20 years after the onset of symptoms.

The hypothesis of its genetic character was established by examining pedigrees of sufferers and noting that almost invariably there was a parent who had the disease or who died young and who had a parent with the disease. Mendelian genetics accurately predicts that if one parent has the gene for Huntington's chorea, one-half of the couple's offspring will, on average, also have the gene and develop the disease if they survive to middle age; if both parents have the gene, three-quarters of their children will have the gene.

The symptoms of Huntington's chorea seem to follow from degenerative process that start in the basal ganglia, possibly caused by abnormally high levels of glutamate and nitric oxide, or by receptors for these transmitters. An excitatory imbalance follows, destroying brain tissue. The mechanism by which the disease progresses to involve cognitive and emotional functions is not well-understood at this time.

There is one known positive effect of the gene for Huntington's disease. Its survival and spread in the population has occurred because of its late onset, after reproduction has taken place, and because it tends to convey greater fertility to its carriers.

### **6. The concept of disease.**

Hull (1977, 1978) pointed out that the genesis, or root of the concept "disease"

involves a dis-ease on the part of the patient. Subjectively this may be expressed in terms of, abnormal and unwanted feelings, but not every dis-ease manifests in occurring disvalued feelings; “disease” may also be applied to abnormal states or functions of the body that are disvalued by the patient. The irreducibly subjective and frequently teleological character of the concept may be illustrated by noting that we do not speak of infertility a disease in celibates or those who have been voluntarily sterilized, but we define infertility in a couple wanting children in terms of their “inability” to achieve pregnancy during a year of unprotected intercourse; in the latter case, infertility is a dis-ease simply understood in terms of a lack of ease in intended procreation. Infertility in a couple not desiring to procreate but interested in conjugal relations would be not the cause for complaint but for celebration.

Partly under the influence of etiologically-oriented clinical medicine, the concept of disease has undergone a causal shift. This has occurred in two steps: first, through the redefinition of “disease” in terms of “syndrome,” a set of, associated symptoms and signs, with the association to be understood both through frequent joint occurrence and through the hypothesis of a common cause for such concurrent symptoms and signs. It is interesting to note how we proceed from a more-or-less purely phenomenalist sense of “symptom,” as in “Doctor, I don’t feel well.” “What are your symptoms?” to a progressively abstracted sense of the term, in which we speak, for example, of elevated temperature as a symptom of suspected infection. Clearly, the possibility of infection being posited as causally related to the symptom reflects our knowledge that fevers have many causes.

The end step in the conceptual evolution occurs when the term “disease” is shifted from the syndrome to an actual causal mechanism. Medicine distinguishes between merely treating the symptoms, and avoiding or curing the disease by preventing or removing the underlying cause of the syndrome. And, now that we are able to identify the presence of the gene or Huntington’s chorea before the onset of the syndrome, we may come to speak of its carriers as diseased long before they manifest symptoms.

The hypothesizing of a genetic cause on grounds of pedigree may not be sufficient for this shift. Bodmer & Cavalli-Sforza (1976) wrote, “Huntington’s chorea is a severe and rare disease with a late age of onset . . . . In many of the pedigrees in which a patient with this disease is found, one of the parents of the patient had the same disease. In others, a parent died well before the usual age of onset and so might have had the disease, had he or she lived long enough” (p. 72, emphases mine). Here, a distinction between having the disease and the onset of its symptoms seems struggling to emerge, but has not done so yet.

However, note that if it becomes possible to confirm reliably that you will have the symptoms in your future, we might expect to come to speak of the possibility of diagnosing the (presence of the) disease prior to the onset of its symptoms, signaling that “disease” is now being applied to the genetic substrate of the disease and not to the onset of the syndrome. Here is an example of an article in which “disease” is going through a dance between identification with a syndrome, with a genetic substrate, and a neutral point in between.

Huntington’s disease is the first “adult-onset” genetic disorder for which testing can determine, years in advance of symptoms, whether a person has inherited the defective gene. (neutral)

As scientists wander along chromosomes in search of specific genes, many more diseases will be detectable long before they’re preventable or treatable. (genetic substrate; the disease is detected solely on the basis of the genetic substrate, implying that the latter is sufficient for the presence of the former)

Testing positive for the HD gene replaces the ambiguity of whether the disease will appear with the ambiguity of when. (genetic substrate: “the disease is present, but hasn’t yet appeared” seems to be the sense)

How people react to the news that they’re destined to develop a disabling or fatal disease is a critical question as scientists labor to make more tests available. (syndrome: the disease is in the future, although certain) (*Health*, 1990, p. 54)

But an even clearer movement of the sort of which I am speaking occurs in the evolution of the following terminology. At the level of symptoms, trading on a superficial resemblance to people of Mongolian ancestry, we first spoke of “Mongolism” or “Mongolian Idiocy.” Later, there was a shift to syndromic terminology, named after the clinician who first integrated symptoms and signs; “Down’s Syndrome” or “Down Syndrome.” The third shift occurred at the point at which the syndrome became reliably associated with the presence of three twenty-first chromosomes: “Trisomy-21.”

I shall return to the question of the moral significance of this shift to identification of a disease in terms of its genetic substrate.

Such a shift in usage, if my supposition is correct that it is to be expected and in fact is occurring, raises the question of what we mean when we speak of “the” cause of a disease or event. Clearly in some cases, the designation of something as for example, the cause of death, involves identifying one of a number of possible candidates as “the cause,” where that designation, in turn, reflects some particular interest. Philosopher of science Michael Scriven once put it, “A Cause is a non-redundant member of a set of conditions jointly sufficient for the effect . . . , the choice between the several candidates that usually meet this requirement being based on considerations of context.” (Scriven, 1962, p. 215)

### **7. The moral significance of the shift from disease symptoms through disease syndrome to disease cause.**

If Scriven is correct, namely, that designating something as the cause of some event, taken as the effect, involves picking out a non-redundant member of a set of conditions jointly sufficient for the effect, with the choice among the members of that set being dictated by considerations of context, then we should look to the factors that set the context that dictates which member of the set of non-redundant jointly sufficient conditions will be picked in order to understand how it is that something comes to be called “the cause of the disease.” And, if my hypothesis about a shift of the term designating the disease from signs and symptoms to syndrome to cause is correct, we should expect a high degree of influence of those factors of context on a disease being designated in terms of its genetic substrate.

Lippman (1990, 1991) and Lippman et al. (1991) describe a phenomenon they call “geneticization,” an ongoing process by which differences between individuals are reduced to their DNA code, with most disorders, behaviors and physiological variations defined, at least in part, as genetic in origin. It refers as well to the process by which interventions employing genetic technologies are adopted to manage problems of health. (Lippman, 1991, p. 15)

I believe that this process can be examined to discover those contexts in which selection of genetic components of the set of non-redundant conditions jointly sufficient for diseases arise. For purposes of illustration and comparison, let us take two contexts: that of the clinic, and that of the work place.

Clinical medicine seeks to develop treatments that, when applied to individuals with maladies, enable them to overcome those maladies or to limit their disruptive effects on a normal life. For many years, insulin shots have enabled diabetics to extend their lives; the insulin micro-pump has decreased the associated

inconvenience and moved diabetics towards more normal lives; and the possibility of transplantation of adrenal tissue containing functional islets of Langerhans promises even greater chances of normalcy.

Recent work on the CF gene indicate the possibility of restoring lung tissue of persons with CF to normalcy by infecting the lung with a virus altered to carry the normal gene that is alternative to the CF gene. In animal models, there is strong evidence that normal secretion can be achieved using such a viral transport; the viral transport inserts the normal allele into the cell of the lung so that secretions of normal consistency are produced. We can even imagine that the alteration would be restricted to lung cells, so that the protective benefits of the CF gene in populations exposed to the cholera bacillus would be preserved, but without the harmful effects. So, in the context of contemporary clinical medicine, geneticization of cystic fibrosis may be enabling the creation of an effective, one-time somatic therapy for persons with the CF gene that will eliminate or prevent the symptoms of the disease. The analogy with vaccination is strong: a routine, one-time treatment prevents the occurrence of a disease by providing an effective, on-site counter to the, in this case, genetic agent that causes the disease.

Here, the context involves the interaction of clinical medicine and advanced genetic technology. The moral argument for geneticization in the resultant context is that it provides a compelling rationale for directing research towards development of an effective one-time preventive treatment for the individual who is at risk for the symptoms of the disease—effectively the same rationale as for preventive vaccination.

Contrast the context created by the union of clinical medicine and genetic technology with that created by the union of the workplace and genetic technology. We are already in the early stages of the tendency of employers to use genetic screening to identify employees or potential employees who are at elevated risk for injuries resulting from exposure to work place chemicals and other physical factors, or at elevated risk for increased frequency of absence or worker's compensation claims, due to the effects of genetic traits on general health. The Congressional Office of Technology Assessment conducted a survey in 1982 that found a number of large companies that had at one time or another tested employees and applicants for sickle cell anemia, glucose-6-phosphate dehydrogenase deficiency, or alpha-1-antitrypsin deficiency. (OTA, p. 133)

When the arguments for such screening by the companies were examined against their actual practices, it appeared that genetic screening was being used to identify individuals at risk and to exclude them involuntarily from either employment or jobs in which such risks would be heightened by exposure to chemicals. (Holtzman 1989,

p. 202). Moreover, the practices were discriminatory inasmuch as not all conditions predisposing to similar injuries and identifiable through genetic screening were tested for. Black applicants to DuPont, for example, were screened for the sickling trait, but other traits that would also predispose to anemia were excluded from testing, as were conditions that would produce borderline anemia for reasons unrelated to non-redundant genetic factors. (Ibid.)

The moral argument against geneticization in this context is that it renders employers able to avoid improvements in work place safety by excluding individuals from it whom the natural lottery has predisposed to harm at levels of work place exposure tolerable by others. Fundamental features of justice are thereby denied to individuals through no fault of their own. Rawls (1972) discusses how the obligation of fairness requires altering external circumstances to provide employment opportunities for persons who are naturally disadvantaged.)

Geneticization involves a deliberate focus on the individual who is at risk. In some contexts the focus enable development of means for reducing that risk through treatment of the individual in ways that are consistent with our moral principles of non-maleficence and justice. In other contexts, the focus on the individual is a focus away from other factors that are amenable to change at some expense, where our moral principles of non-maleficence and justice are violated. Geneticization can result in an illicit equation of human biology with human genetics. Lippman, 1991, charges this; whether such an equation is pernicious depends on how moral principles are preserved or abused.

The question of whether asymptomatic carriers of the Huntington's disease, or heterozygous carriers of the cystic fibrosis gene are dis-eased would normally seem to involve a conceptual confusion but for the predictive power of diagnosis. For, if to be dis-eased fundamentally involves "dis-ease," and an individual lacks a normally necessary condition for the presence of a disease, such as abnormal or distressing symptoms involving either increase in discomfort, or impairment of a desired functional capacity, it is nonsense to speak of such an individual as diseased. However, if you can now know, on the basis of factors already present, that your future predictably contains such severe complaints, that their occurrence is "only a matter of time," or that your children will, with a certain degree of likelihood, suffer severe medical problems as a result of your genetic transmission, the question of whether you are diseased arises meaningfully. For, to be diagnosed as diseased in some way evokes automatic changes in your socialization, life planning, and so forth, that fall severely outside of the normal patterns of health life.

Worst case scenarios galvanize action when they become more than bare logical

possibilities. A diagnosis of an active disease prompts actions to curb the spread of the disease if infectious, to minimize or avoid symptoms, and, where possible, to eliminate causes. It provides a powerful excuse from the responsibilities normally associated with health in a social setting. And it imposes special burdens of care and accommodation on family, friends, employers, and insurers of the bearer of the diagnosis. Since diagnosis of carrier status of an asymptomatic individual predicts a probable future in which similar personal and social disruption will take place in a manner relatively free of further contingencies, the application of “diseased” to such individuals involves a powerful and tempting extension of the concept.

It is important to note, however, the contextual character of the predictive import of a diagnosis of carrier status. For a confirmed celibate, being a heterozygous carrier of the cystic fibrosis gene has no negative predictive value, in that there will be absent, for example, offspring resulting from a rape or from your role as a gamete donor no individual created at risk for transmission of the disease. Similarly, for a carrier married to a non-carrier, there is, all else being equal, no negative predictive value associated with the status of carrier. And, for a carrier married to a carrier where one or both are sterile by choice or naturally sterile and comfortable with the fact, there is no negative predictive value associated with carrier status. In fact, we can imagine contexts (for example, the prospect of becoming a missionary or health worker in a region with endemic cholera) in which there is positive predictive value associated with the status of carrier. I would submit that in all of these cases, and perhaps others, it would be not only pointless but conceptually confused to call such individuals diseased. For, given certain simplifying assumptions I have made about expressions of the lethal recessive heterozygous gene, there is no lack of ease they will ever experience that would ground the subjective root of the concept.

By contrast, a couple intending and able to have their own biological children who are both heterozygous carriers of the gene for cystic fibrosis are, on my analysis, reproductively diseased; their reproduction will not be easy. On average, without special measures, 25 percent of their children will be severely affected by a disease that they have given them. Their lives will be negatively affected for a substantial period of years; the future of other members of their families, their friends, their employers, their insurers will be affected by the combination of their carrier status and their reproductive objectives. It will be appropriate for them to consider extraordinary measures: the possibility of using donor gametes; the possibility of employing reproductive technologies to preselect against fertilization of carrier ova with carrier sperm, or against implantation of embryos with both alleles. If their carrier status is known in advance of marriage, it may be appropriate for them to seek

other mates, individuals with whom neither will have a reproductive dis-ease.

But even these observations hold in an uncomplicated way only given a context in which the advantages conferred by the CF gene are not operative. Cholera is currently at epidemic proportions in a number of South American countries. Given that the appropriate environmental factors hold for a couple at risk for children with CF, they may expect greater reproductive success and less infant mortality than a couple neither of whom is a carrier. Of course, improved sanitation to eliminate the epidemic is of far greater utility than spreading the CF gene.

Contexts in which being an asymptomatic carrier of the Huntington's chorea gene would have no negative predictive consequences are difficult to imagine, but not impossible. Certainly, having no interest in, or ability to have, children will relieve such a carrier of a substantial portion of the negatively valued, predictable consequences of that status. The person who knows he or she will die before the earliest point of onset of the symptoms, for example a convict on death row, or a terrorist who has committed to a suicidal mission, lacks any negative consequences of that status. Indeed, possession of the status of asymptomatic carrier for Huntington's chorea may prompt a person to undertake meaningful risks normally avoided by an individual with the prospect of a long and healthy life.

On the other hand, most carriers of the gene for Huntington's chorea do face and, if they know of their status, contemplate, very negative predictable futures. Reason would seem to dictate that, for them, certain activities, such as undertaking the long preparation for a career as a neurosurgeon, ought to be avoided. In these individuals, the status of one who has a disease may well galvanize appropriate responses to a future that is far more determinate than that of most. Such judgments will be possible with far greater precision with the technology for identifying the particular defective allele that is productive of the degree and age of onset of the symptoms of cystic fibrosis; we may then anticipate greater precision in identification of individuals in terms of the disease's genetic substratum. Given a "moral context" of such identification, no objection would seem to attach to regarding such individuals as diseased.

## 8. Conclusion

The decision to regard individuals who carry the genetic substratum for Huntington's Disease or Cystic Fibrosis as diseased is a decision with an undeniable valuational component. In some contexts, such a designation promotes discrimination that is unjust. In others, such designation empowers, as when the

claim of a diseased individual for preventive treatment is honored. Such designation is not merely a matter of application of scientific facts; science itself is not value free, in that its directions are determined for human purposes. But where given science's identification of actual constellations of conditions jointly sufficient for various effects, the decision to designate one of those conditions the cause is a matter of context, itself determined by human values.

As we design social policy for absorbing and integrating the results of the human Genome Project into our practices, we would do well to keep foremost in mind that the scientific language of causation is designed to serve our purposes, and not the reverse.

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