"Designing Humans versus Designing for Humans: Some Ethical Issues in Genetics," Philosophy Department Colloquium, SUNY College at Geneseo, April 6, 1978.

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by
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At a meeting of the American Society for Value Inquiry in Chicago last spring, and again at a conference on biomedical ethics last fall in London, Ontario, David J. Roy, Head of the Institute for Medical Humanities, University of Montreal, described a developing situation in the biomedical technologies about which he and many of his colleagues in the profession share an enormous apprehension. The biomedical sciences have in their possession, in development, and on the drawing boards a technology that has the potential of enabling us to alter much of what has to date been seen as fundamental givens and fixed points of the human situation, from the forms of human reproduction, through the frequency of distribution of various human characteristics, to those very characteristics themselves.

His question , in the form of a plea, was this: it is becoming desperately urgent that scientists and technologists in these fields be given guidance about what they are doing; the sense is that what *can* be done ranges far beyond what *should* be done, and that the technological imperative (do what technology makes possible) and the epistemological imperative (find a use for what we know) are so strong that, in the absence of a normative consensus of what it is to be human, these sciences and technologies may well have a transforming impact upon a society that is not prepared to control them. When it becomes possible to eliminate the traditional and biological form of human reproduction, with the development of in vitro fertilization and the artificial placenta, shall we? When it becomes possible to eliminate deleterious genes from the human gene pool (or to limit their occurrence to the level of chance mutation), shall we? When it becomes possible to enable parents to select in advance characteristics of their offspring, from sex to hair and eye color, and perhaps even to influence the polygenic determinate and determinable characteristics of race, stature, aggressiveness, intelligence and talent, to the point that we can influence the distribution of propensities and abilities in the population to fit more closely the manpower needs of our society, shall we? Shall we breed astronauts, musicians,

artisans, using either techniques of gene replacement or cloning? Shall we, in short, make mankind itself the subject of the design professions? And if we do, for whose or what sake shall we design humans?

Roy's concern is fairly uncommon among scientists; rather more common is the attitude that scientist engage in basic research and that what is done *with* their discoveries is not within their proper sphere of concern. This attitude underlies, for example, the remarks of Bernard Davis of Harvard University Medical School's Bacterial Physiology Unit at a conference sponsored by the New York Academy of Sciences last year. He wrote:

> ...(R)omantics of the counterculture and the New Left have accused scientists of dereliction of duty in not personally preventing their discoveries from being put to bad use. But this criticism is based on two tacit and very questionable assumptions: that scientists could have the power of such control if they wished, and that good and bad are self-evident. In fact, such ethical decisions ultimately involve the whole public. (1)

Most scientists who smart under the sting of public criticism perceive themselves as unfairly called to task for the misuse of their discoveries, where that misuse occurs at a level of exploitation of those discoveries over which the scientist — at lest the "pure" one — perceives himself as having no control and little influence.

At the other extreme, however, is the scientist who recognizes the potential of eugenics and the new genetic technology for alteration of the human condition, and welcomes it. One is perhaps inclined to forgive Charles Darwin, writing 106 years ago before our more modern examples of technology out of control, for saying:

> We civilized men . . . do our utmost to check the process of elimination; we build asylums . . . We institute poor-laws . . . . There is reason to believe that vaccination has preserved thousands, who from a weak constitution would formerly have succumbed to small-pox. Thus the weak members of civilized society propagate their kind. No one who has attended to the breeding of domestic animals will doubt that this must be highly injurious to the race of man. It is surprising how soon a want of care, or care wrongly directed, leads to the degeneration of a domestic race, but excepting in the case of man himself, hardly anyone is so ignorant as to allow his worst animals to breed. (2).

Yet if anything this attitude is even stronger in very influential individuals today. The authors of the standard text on population genetics have written:

> The aim of eugenics is the improvement of the species by decreasing the propagation of the physically and mentally handicapped (*negative* eugenics) and by increasing that of the "more desirable" types (*positive* eugenics). It is, in other words, the application to man of the methods developed by breeders for improving their stocks by artificial selection . . . . (s)ome genotypes can do very well in certain environments, while they do less well, or worse than average, in others It would be a terrible waste to force a potential Bach to become a bricklayer or an engineer, or a potential Einstein to be an accountant. (3)

Cavalli-Svorza have strengthened Darwin's call by calling for the application of present eugenics and the genetic technology that is with us and just over the horizon to alter human reproduction so as to fit better the needs of the species. But to be fair, it is more common to find much less extreme proposals and praises of eugenics and the new genetic technologies by scientists. Consider, again, Bernard Davis's observations:

> . . . the genetic cure of monogenic diseases would still not bring us closer to the dangers of genetic manipulation of technologies . . . . (For,) most phenotypic traits are (polygenic and) governed by gene-environment interactions, rather than determined by either alone. (1)

Davis's logic is puzzling: his argument appears to be that development of the ability to manipulate monogenic phenotypic traits according to our wishes will not bring us closer to the ability to manipulate polygenic phenotypic traits (which seems false), and will thus not bring us closer to manipulating traits that result from the interaction of a number of genes with factors in the environment. In thin veil, this is the standard position that the techniques and tools of science are in themselves innocuous; how else can we understand him except as saying that developing the tools of genetic manipulation won't bring us any closer actually to manipulating with them?

I want to argue in this set of remarks against the suggestions of Cavalli-Svorza and Darwin that we favor eugenic policies, and I want to argue against Davis that the technology we are developing, indeed the very language we use to characterize it, is value-laden. My examples will be drawn from the literature on (so-called) genetic disease, but I intend the implications broadly, as falling under the general rubric of this paper: Should we de sign humans, or (only) design *for* them? So, if I may, I'd like to classify human genetics as a design profession (which it most certainly is), and inquire into the question of whether it is value-free or not. If not, I want to raise the questions of how and under what conception human genetic research should be continued. My suggestion will be that genetics be relegated to the position of a subdiscipline of euthenics, the study of the ways in which the environment acts upon the individual and can be

altered so as to improve the individual's lot.

There is a cluster of words and phrases that are continually employed in genetic counseling, therapy and research, but whose careful analysis has been generally neglected. The more obvious of these (with the explicit valuational component italicized) include expressions like "*mal*adaptive trait, congenital *mal*formation," "*defect*," "*deleterious* gene," and "inborn *error* of metabolism." Even such terms ad "genetic *ab*normality" and "chromosomal *aberration*," indicative of only a deviation from the normal range of variability, are, I suspect, most frequently used in application to disvalued divergence.

Less obviously, there are even valuational components tot he meaning of the important and constantly-used expression, "genetic disease," and I want to examine them in this paper. These components are in part etymological, stemming from the origins of the term, "disease." But they also derive from two other sources: the social and psychological results of identifying a disease through only one of its causal conditions, and the consequences of the selective focus on only one particular range of possible therapies, prevention, and research stemming from the decision to call a disease "genetic." In the following sections, I will trace out these sources of the valuational aspects of the expression "genetic disease," to show the ways in which they come into conflict in the genetic counseling situation.

Etymology of "genetic disease": some shifts in meaning

A. "Disease"

Conceptually, the notion of disease originates in the patient's complaints about bodily discomforts. A disease is identified, first and foremost, in terms of the dis-ease involved. In the subjective sense of the term, we speak of symptoms, or abnormal feelings which the patient has. But a disease is also identified in terms of abnormal states or conditions of the body which are publicly observable; these are sometimes also called symptoms, but I shall refer to them as signs. Some signs may be so obvious that they become part of the patient's complaint (such as rashes, paralyses, etc.), but others may require more skilled observation to detect (such as dulness of some portion of the lungs under percussion).

Apart from physiological knowledge of how an individual symptom or sign arises and may be made to recede, often little else is required for mere palliation. But the notion of disease has also come to involve, first, the idea of a syndrome, and second, the idea of its cause. A syndrome is a set of *associated* symptoms and signs, associated both through their frequent joint occurrence and through the notion that they have a common cause. For we are frequently not satisfied with mere palliative measures: they must be constantly administered throughout the disease's natural course to suppress the occurrence of signs or symptoms, and they are frequently not fully successful. They involve conceiving of and

treating symptoms individually, whereas we suspect that in most cases constellations of signs and symptoms can be thought of as manifestations of a common cause.

But it is overly simple to speak of "the cause" of a disease. One of the benefits of philosophers' investigations into the term is to show how idiosyncratic our use of "cause" can be. In its fullest sense, of course, the cause of something is the total set of its individually necessary and jointly sufficient conditions. In talking of the cause of a disease in more ordinary circumstances, however, this definition of "cause" is not employed, in large part because of the difficulty of satisfying its demand. Rather, "some one or more conditions within that set which were novel, unusual, or controllable" are usually cited in giving the cause. (4) Michael Scriven once described this common notion as follows: "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." (5) What strikes us as novel, unusual, or controllable will be singled out as the cause, because our interests lie in uncommon, novel, or controllable phenomena.

The notion of a syndrome — a set of associated signs and symptoms thought to have a common cause — is not quite sufficient to capture the concept of disease as it is most commonly employed today. Indeed, there are some dramatic departures from the notion of disease as rooted in the patient's characteristic complaints together with associated signs. In its efforts to understand, control, and avoid disease, modern medicine has incorporated into the very identification of a disease the notion of the cause of the syndrome. This permits the individuation of similar syndromes with distinct causes into different diseases. (6) For example, the general classification of epilepsies along syndromic lines (petit mal, gran mal, temporal lobe) is augmented by classifications amounting to discrimination by cause: each type is further divided into ischemic, traumatic, and ideopathic. Thus, there are now three kinds of petit mal epilepsy; the symptoms are no longer definitive of the disease, but constitute manifestations of the disease; the disease is not identified with the underlying cause of the symptoms; and ischemic and traumatic gran mal epilepsy are spoken of as distinct diseases which manifest similar symptoms. (7) What has happened is that the root notion of disease — the syndrome — is replaced in its identification by the syndrome's cause. I shall return to this significant point.

B. "Genetic"

There are two things to be said about the "genetic" component of the expression "genetic disease." The first can be put in a couple of ways. Every non-genetic disease has a genetic component, describable in terms of the presence (or absence) or one or more genetically-determined traits. Susceptibility or

resistance to infection by a given micro-organism, injurability, toxic reactability, are each in part a function of genetic endowment (although doubtless more frequently polygenic than monogenic in character). We may well suppose that individual susceptibility, say, to smallpox is a function of genetic endowment, but we don't classify smallpox as a genetic disease. Why? Another way of putting the same point is this: every non-genetic disease has both genetic and non-genetic members of its set of non-redundant, jointly sufficient conditions. This raises the question of why and how some diseases come to be classified by appeal to their genetic component(s) and others by appeal to their non-genetic component(s). Whence our interest in only certain members of those sets of jointly sufficient conditions, and not others? I shall return to these points.

The second observation is this.. Many so-called genetic diseases have known environmental components; the associated syndrome is manifested only in the presence of specific environmental factors or agents. For example, the hemolytic anemia associated with glucose-6-phosphate dehydrogenase deficiency is manifested only upon exposure to certain foods (such as Vicia fava, the common broad bean) or certain drugs (such as primaquine, used in treatment of malaria). (8) Xeroderma Pigmentosum skin cancer is triggered by sunlight and ultraviolet radiation, but otherwise would not occur in persons homozygous for the recessive gene. Further, many genetic diseases involve the deficiency, or deficiency at a crucial stage of development, of some enzyme or hormone

that plays a developmental or maintenance role. Insulin, whose absence produces the hereditary metabolic disease diabetes mellitus, can be artificially supplied, thereby allowing the patient to live a relatively normal life. Homozygotes for phenylketonuria are unable to convert phenylalanine to tyrosine because of the lack of the liver enzyme phenylalanine hydroxylase. (8) The weight of evidence indicates that the presence of phenylalanine and its metabolites in large quantities hinders the laying down of myelin. Hence, a low phenylalanine diet, instigated at birth, allows more or less normal development of myelin to occur, thus avoiding or minimizing the associated mental deficiency. The diet may be relaxed after the first several years, since most myelin is laid down then and only maintenance of the neural sheaths is carried out thereafter. The point is that in these cases it is the absence of the enzyme which is "the cause" of the disease; but the genetic abnormality is not necessarily itself sufficient for the disease, for the enzyme could (in principle) be supplied artificially, or the results of incomplete metabolism avoided by dietary control. Sodium cyanate has been used clinically in treatment of sickle-cell anemia (although it is a suspected toxicant of the central nervous system); and a recent report indicates that dimethyl adipimidate may act on hemoglobin to increase its affinity for oxygen and restore the red blood cell membrane to normalcy without affecting red cell metabolism. (9)

Thus, even diseases such as sickle-cell anemia, which seem under normal

conditions to possess causally-sufficient genetic antecedents, frequently are manifested through intermediate steps that are capable of reversal or interruption.

Finally, a number of genetic diseases that are thought to be due to a single (dominant or recessive) maladaptive allele may in fact involve the absence of some offsetting additional mutation. For example, several cases have been reported of healthy adults in families having Tay-Sachs or Sandhoff's disease, in which the healthy adults appear to have a double dose of the associated lethal recessive gene. (10) (11) (12) The evidence suggests that the effect is due to the presence of an offsetting mutation in the gene. So, if the presence of the second mutant is a sufficient condition for the non-occurrence of Tay-Sachs or Sandhoff's disease, its absence is as much a necessary condition for each of those diseases as is the commonly associated so-called lethal recessive in double dosage. What at first appears to be a classic case of the presence of a genetic factor homozygously fitting our definition of cause, so that we could without embarrassment identify it as *the* cause of the disease, turns out not to be so unequivocally.

This latter sort of case suggests a way to view even the most genetic of diseases in a non-genetic way. Our general understanding of such disorders indicates that in them the presence or absence of certain gene products in the internal biochemical environment of the body are the causes of those genetic diseases with no identifiable external environmental factor. But this raises the theoretical possibility of modifying the internal environment by means of introducing the essential product at its crucial pont of interaction in some metabolic chain, or of suppressing the production, or otherwise offsetting the effect, of some gene product. Thus, strictly speaking, the presence or absence of the gene itself is not the cause of the disease, but rather the presence or absence of one or more chemicals that serve as partial determinants of the inner environment. Tot he extent that such products theoretically can be suppressed or supplemented by externally-originating action, we may fairly characterize even the most "genetic" diseases as having non-genetic components.

These cases suggest the possibility that no disease (or at most very few diseases) has only genetic factors involved in its total set of individually necessary, jointly sufficient conditions. It is evident that in most cases of genetic disease the term's application reflects a choice having been made among the causal factors to emphasize the genetic component and de-emphasize the environmental component. And there is some inductive reason to suppose that even the hard core of what now seem truly "genetic" diseases may shrink, as our knowledge of euthenics engineering increases, and we become able to control expression of the existing genetic information of individuals affected with diseases having genetic components, so as to lead to more desirable phenotypes. (13) Hence, I am tempted to endorse the generalization that every "genetic disease" has non-genetic components, whether presently known and understood

or not.

Putting together the results of our enquiry, we see that the decision to call a disease "genetic" involves its identification with a non-redundant or individually necessary member of the set of conditions jointly sufficient for the associated syndrome, where there may be alternative necessary conditions with which it is not identified due to our relative lack of interest in them; further, the decision to call a disease "genetic" involves shifting the application of the term "disease" from the syndrome to "the cause," and the designation of the syndrome as a *manifestation* of the disease.

As Sheldon has observed, "the basis of the categorization of disease fundamentally determines what gets done about disease, even more than the organizational structures which develop to deal with it." (14) If so, the decision to identify a disease as genetic may well determine a preference of a particular range of possible foci for therapy, prevention, and research. It thus becomes legitimate to ask, What are the moral dimensions of this "decision"?

Uses of "genetic disease": some policy implications

The positive benefits are by now obvious and familiar. They involve (I) furthering our knowledge of human development and variation as determined by the fundamental processes of human reproduction; (ii) demythologizing the occurrence of various kinds of disease (in particular, providing more cogent explanations of various disorders than their supposed visitation on persons as punishment for sin); (iii) the possibility of eradicating various kinds of disease (or reducing them tot he level of chance mutation) by elimination of their genetic determinants from the gene pool; (iv) the possibility of improving the species and thus exerting control over evolutionary processes; and so forth. Rationales of these sorts generally hold up select long-term consequences as justifications of the pursuit of genetic research, expressed in terms of the elimination of certain diseases, the advancement of knowledge, perhaps even the enhancement of desirable characteristics of the species, as likely future benefits.

But of course all such goals can be pursued whether we employ the typology "genetic disease" or not. Given that employing the term diagnostically does contribute in some way to a social and policy-making climate favorable to those goals, whether they provide adequate justification for such a use depends on whether there are significant negative considerations relevant to their employment. (I ignore, for present considerations, the question of whether even these goals may not have negative aspects.) We may first observe that identifying any disease in terms of a causal factor has profound effects on our perceptions of alternative causal accounts. To build one causal factor into the very conception of a disease and thereby to prefer one causal hypothesis over others is to invite stagnation of research and treatment, and distortion of funding priorities. (A case in point is that of the viral conception of the etiology of

cancer, a concept that has held sway in the funding of cancer research to the detriment of other approaches that have a higher likelihood of payoff in increased rates of cure.) (15)

Settling upon causal specifications discourages alternative conceptions of disease. A theory-neutral conception of disease in terms of symptoms and signs, while not independent of subjective factors, would permit the development of such alternative paradigms of disease as behavioral and biological ones that selectively emphasize complementary aspects of the disease process. But to conceive and define a disease in terms of one causal aspect is to impose *a priori* the appearance of implausibility and confusion on other approaches.

Within the context of our own cultural institutions and practices in dealing with disease, some more specific negative consequences of the typology "genetic disease" can be seen. But Sheldon (14) and Engel (16) have emphasized the effect of the nomenclature employed in identifying a disease on therapeutic and preventive measures elected by the physician. In this way, identifying a disease with one range of necessary conditions (say, genetic) focuses attention on one range of treatment and/or prevention. This in turn results in preconceptions of false or inappropriate dichotomies. For example, either one aborts a fetus identified through amniocentesis and caryotyping as having a genetic disease, or one produces a child with the disease. This dichotomy is false in cases where there exists an alternative way of avoiding the symptoms of the disease. For example, it is possible to diagnose glucose-6-phosphate dehydrogenase deficiency prenatally. Avoidance of foods and drugs that precipitate it is an alternative to both abortion and production of a child with the disease symptoms, but this alternative may be obscured by identifying the disease with the genetic antecedent. With genetic diseases for which prenatal diagnosis is not yet possible, such as phenylketonuria and galactosemia, avoidance of production of an individual afflicted with the disease would be possible only if reproduction were prevented altogether. But again this is a false dichotomy; in the case of PKU strict observance of a phenylalanine-free diet for six years avoids or greatly reduces the associated mental retardation (17), and a similar result is achieved with a galactose-free diet in cases of galactosemia. Even in writers who appear not to fall prey to such false dichotomies, we find evidence of a conceptual distinction between a disease and a set of symptoms as its manifestation that indicates that the disease has been identified with the cause. For example, in discussing the use of phenylalanine- and galactose-free diets to avoid the mental retardation associated with phenylketonuria and galactosemia, Murray writes: "The dream of the physician is, after all, the prevention of *the manifestation of disease* rather than its cure after it has been found" (18) (italics mine). So, according to Murray, it is the manifestation of the disease and not the disease itself that is prevented in the case of genetic diseases with manipulable components; and possession of the genetic component is a sufficient condition for possession of the disease, regardless of whether it is

manifest. In this sort of conception, the genetic disease can be prevented only by preventing creation of individuals who possess the genetic component.

The foregoing point is closely related to another one, namely, the stigmatization of persons who have genetic diseases. Not only have we come to use the term "carrier" for ones whose offspring bay be at risk for some genetic disease, thereby invoking folklore figures like Typhoid Mary. We have long had practices based on the principle that those who are genetically diseased ought not to reproduce. By extension of the "carrier" metaphor, sterilization becomes a form of quarantine. We thus slide into social policies and attitudes that ought at the least to be squarely faced, independently debated, and adopted, if at all, both with the justification of a solid cost-benefit analysis and with full knowledge of the price paid in the currency of individual liberty and self-determination.

The identification of a disease with its genetic component, since it displaces the patient's complaint as the primary determinant of the occurrence of the disease, has other potential political and social consequences that raise grave concerns. One effect of separating disease from the diseased one's complaints is that the finding of disease can be made on the complaints, actual and anticipated, of others. The controversy surrounding the "XXY syndrome" serves as a case in Point. An unproven, low statistical association of criminality and the occurrence of an extra Y chromosome in males has been suggested. Walzer and Gerald of Harvard Medical School initiated a long-term study of newborn XYY males, in part to determine the degree of genetic (i.e., chromosomal) contribution to behavior problems. But they have been quoted as holding that the XYY chromosome pattern " is a 'disease,' . . . and . . . children who have it are entitled to medical treatment just as they would be for any other disease" — this, despite their admission that while "some XYY children are 'hard to handle,' others are 'perfectly fine.'" (19) The associated syndrome seems to have at best only an indirect relation to the complaints of XYY individuals; rather it is the complaints of parents, teachers, and any who have been victimized by XYY individuals who are criminals that constitute a large part of the syndrome. And, XYY individuals are all classified as diseased, irrespective of whether "hard to handle" or "perfectly fine," because they possess the genetic basis for the disease. It appears evident that there is an enormous environmental component in the determinants of the antisocial behavior of those who are "hard to handle"; how dies calling the XYY individual diseased on the basis of his chromosomal pattern enhance our understanding of why the behavioral syndrome occurs? Given that the incidence of the correlation is low, it looks as though the identification of the disease with the genetic component only serves to direct attention to a relatively unimportant component of the total cause. And, so directed, we are led to the utterly useless stigmatization of XYY individuals as all incurably diseased. (20)

Finally, let me cite the case of some research in progress into the genetic components of cancer, and point to two possible ways of handling the outcome.

It has long been known that susceptibility to bronchial carcinoma in cigarette smokers is varied; the occurrence of lung cancer in individuals who have a similar history of rate and duration of smoking is markedly dissimilar (a fact that has bolstered the claims of cigarette manufacturers that it has not been proven that smoking causes cancer). In recent years, the National Institutes of Health have supported research into the basis for such variability. Recent reports indicate that the variability centers about low, intermediate, and high inducible aryl hydrocarbon hydroxylase activities, with susceptibility to bronchogenic carcinoma being associated with the higher levels of activity; these levels of activity are in turn thought "to result from two alleles at a single locus with the three [levels of activity] representing homozygous low and high alleles and the intermediate heterozygote." (21) This and other research has prompted one researcher to observe: "The studies in the journal by Kellerman and his associates suggest that the measurement of BP- [benzypyrene-]metabolizing enzymes in human lymphocytes may allow us to detect members of the population who are uniquely sensitive, or who are resistant to carcinogens in cigarette smoke, and further studies along these lines are to be encouraged." (22)

As I view it, the question is not whether such research ought to be continued, but what is to be done with its results. Will it, for example, prompt public concern for those individuals carcinogenically sensitive to cigarette smoke, and reinforce legislation to protect them from unintentional exposure to tobacco smoke by restricting the sale and private use of tobacco cigarettes to individuals known to be resistant? Or, will the results of these researches be used to identify bronchial cancer as a manifestation of a genetic disease, thereby rendering the individual responsible for avoiding, as best he/she can, the noxious substance? Depending on how the knowledge resulting from the research is characterized, what many regard as a matter calling for regulation in the public interest may become a matter involving only prudential considerations for individuals having a genetic disease — despite the fact that bronchial carcinoma is a condition resulting from the interaction of genetic and environmental factors. In light of the valuational components of the criteria listed earlier for the use of "genetic disease," the socialization of the anticipated results of this and similar research should be highly instructive.

Conclusion

To sum up, I have been talking about shifts in the development and application of the notion of disease that have a strongly anti-humanistic, anti-individualistic implication. From its original denotation of a dis-ease of some individual, the concept has progressively been sharpened and shaped to include, first, causally related congeries of symptoms and signs, and then, inclusion of specific causal mechanisms of their productions as individuators of diseases. I believe that each of the shifts involves a selection among alternatives, and I have argued that the

selection among the set of possible causal components is a choice that reflects a decision (perhaps unwitting) to locate responsibility for disease within the individual rather than in environment. This can be seen as directing not only research and treatment toward the individual but also as directing our conception of causal responsibility away from environmental factors that can be controlled only through social resolve, and locating causal responsibility within individuals. As it becomes so located, the *individual* becomes laden with the responsibility to avoid the manifestation and spread of the disease. The policies which Carwin and Cavalli-Svorza and Bodnar have in mind are ones that aim at controlling the spread of disease conceived as genetic by restricting individual freedom — either prudentially through counseling or at the policy level through limitation of reproductive freedom. Linus Pauling has proposed that we each be tattooed or otherwise obviously labeled, so that we can recognize and avoid those with whom we would be at risk for defective offspring before becoming romantically involved. This would presumably be enforced legally and culturally with the kinds of taboos we have against incest, and it would be much more efficient than the unreliable, arbitrarily applied, and easily concealed device of children taking family names.

As against these shifts, I want to urge a recognition of the value-ladenness of such concepts of those design professions, genetics and eugenics, as "genetic disease." Having recognized that value-laden character, I want to urge the reexamination of those influences that shaped our step-wise profession to the notion of genetic disease. Those influences are complex, and I cannot pretend to have adequately identified or structured them. But surely they will include such factors as the facts that our medicine has been focused on individuals, has been curative rather than preventive, and has been pursued under not only cost-benefit models of rational decision-making but also under an increasing pressure from technological and epistemological imperatives.

As this picture emerges in outline and begins to be filled in in detail, we see that in important and fundamental ways the individual and his or her rights have been displaced as that for the sake of which we design; steadily the individual is becoming the last and greatest technological challenge, that which we will manipulate, that which is to be designed.

Immanuel Kant wrote of the nature of ethics that central to behaving ethically is acting always so as to treat others as ends in themselves, not merely as means. I think if Kant were alive today, he would propose a "Copernican revolution" in genetics, turning it around so that it is made to exist for the sake of individual humans, bu showing that without such a presumption, genetics cannot justify its own existence. For the species is but an aggregate of individuals, stretching through time; to treat its health, progression, continuation, and efficiently managed evolution as the end of genetics, is thereby to subordinate the individual and his or her interests and welfare as relatively unimportant. But it is an individual (or group of individuals) that does that elevation of the species and subordination of individuals and their interests. This means that to will the ascendancy of the importance of the species over the individual is to will that individuals' interests be accorded less than primary importance. But this implies, since it is an individual's or group's interest that leads to the elevation of species over individuals, that interest is also to be subordinated. If so, the geneticist cannot will *his* vision of the end of genetics without implicitly willing that it, as the vision of an individual, *not* be made the end of genetics.

To recognize all of this and its implications is, as Marc Lappé of the Hastings Institute writes,

> . . . to envision a society in which eugenic concepts and policies play an insignificant role — where individual differences . . . [are] *not* subject to economic or genetic capitalization. Genetic differences [t]here would be seen as just as real, but would be denied the moral relevance afforded to other principles, such as liberty and equality of opportunity (and fairness).
> Such a society would likely be less "efficient," both economically and genetically, than would an eugenic society [where] the aim is rapid improvement of stocks of individuals with desirable qualities, so that the gains made each generation can be reinvested through gametic or mate selection into the next generation. [O]pting for "efficiency" in such circumstances is itself a moral decision which carries social costs. For example, under the guise of efficiency it might be expedient to reduce programs whish would aid the mentally or physically handicapped. Under some circumstances, a less efficient society might e a more moral one. (23)

Somewhat more fleshed out, the proposal such as Lappé and I make for the design professions of human eugenics and genetics is that, first, they recognize the moral limitations on what they may properly manipulate, recognizing that the manipulation of individuals for the sake of groups at a level that invades that which is most distinctively individual — one's genetic constitution — transgresses Kant's moral maxim. Second, we propose that the alternatives to locating the responsibility for transmitting genetic disease and for preventing its

manifestation be examined and made the priorities for future research in these fields. Third, we propose that human euthenics be accorded a higher social and funding priority than human genetics, to reflect the commitment to hold fast to the individual and his or her rights and welfare as that for the sake of which our elaborate institutions of science and medicine exist. Only at the extremes of our understanding are genetic manipulative techniques to be recommended as preferred responses tot he prospects of disease.

In closing, let me say that I view the issues with genetic engineering to be typical of other design professions. As a humanist and as a human affected by those professions, I feel constrained to call for the re-introduction of the Kantian ideal into those professions. They should design for humans, not design humans for other's purposes. To David Roy's query with which I started, the answer given by philosophy insofar as I am able to speak for it, is: each humans' welfare is the end of your efforts; do not come to treat humanity's members only as means.

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