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HUMAN SOCIOBIOLOGY AND GENETIC DETERMINISM

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INTRODUCTION

Recent efforts to forge a new discipline of sociobiology have engendered great controversy. While some of the disputes have been silly and needlessly bitter, many of them have touched on substantial issues of great general interest. This is particularly true of the attempts to evaluate the application of sociobiological reasoning to humans.[1]

Although the present paper will be severely critical of current sociobiology, it is not narrowly polemical in character. The argument is, at heart, methodological, drawing on biological as well as metabiological considerations to show that, as yet at least, attempts to apply sociobiological reasoning to humans are premature. The scheme of my methodological argument is of general interest in that it may be extended to work in related disciplines. Accordingly it will be useful to characterize my fundamental methodological concern briefly before turning to an examination of sociobiology.

Philosophers and historians of science have long recognized the importance of terminology and terminological reform in science. If one employs a descriptive apparatus ill-suited to the phenomena with which one is concerned it will be extremely difficult, perhaps impossible, to achieve a satisfactory explanation of the whole range of those phenomena without engaging in some form of terminological or conceptual reform. Perhaps the most-discussed example of this point concerns the chemical revolution of the late eighteenth Century. For example, it is not possible to give an adequate account of the chemical reaction in which mercuric oxide, then known as the red calx of mercury, when heated, gives off a gas if one

describes that gas (as Joseph Priestly did) as dephlogisticated air. In order to achieve an adequate explanation of this reaction, it was necessary first to revise the nomenclature and the descriptive categories of chemistry.

The central contention of this paper is that a situation similar to Priestly's obtains in contemporary sociobiology. I shall argue that there are strong (though perhaps not absolutely compelling) reasons for holding that, if one applies socially familiar descriptions of human social behavior ("altruistic," "aggressive," "friendly," "intelligent," "lazy"), the phenomena *as thus described* are not susceptible of sociobiological explanation.[2] I shall also argue that current genetic knowledge is not powerful enough to support typical sociobiological claims couched in terms of the familiar vocabulary by means of which we describe the socially significant behaviors and interactions of humans (and also of higher mammals).

These contentions, if correct, undermine the claim that sociobiology can establish the role of the genes in determining behavior as ordinarily described and thus provide us with a biological (indeed, a genetic) account of human nature. This failure, in turn, removes all support for claims to the effect that current sociobiology is of critical importance to the social sciences, to the design of social policy, or to philosophical anthropology. I shall not pursue these larger concerns here, but I shall amplify below on some of the methodological morals which can be drawn from this study.

WHAT IS SOCIOBIOLOGY?

Let us begin at the beginning. Sociobiology is a discipline (or would-be discipline) which has a founding document, E. O. Wilson's large and important book, *Sociobiology, the New Synthesis.*[3] For present purposes, I shall deal primarily with Wilson's brand of sociobiology although, as will be seen, my central points do not depend particularly on Wilson's formulations. Wilson defines sociobiology as "the systematic study of the biological basis of all social behavior" (p. 4). He believes that it completes the neo-Darwinian theory of evolution by reformulating the foundations of social science in such a way as to make "sociology and other sciences, as well as the humanities" into branches of evolutionary biology, in particular sociobiology (p. 4). It is of some importance that I am describing this extraordinary ambition correctly. To reinforce the point, consider the brief first paragraph of the concluding chapter of the book:

> Let us now consider man in the free spirit of natural history, as though we were zoologists from another planet completing a catalog of social

> species here on Earth. In this macroscopic view the humanities and social sciences shrink to specialized branches of biology; history, biography, and fiction are the research protocols of human ethology; and anthropology and sociology together constitute the sociobiology of a single primate species. (p. 547)

If Wilson's lead is followed, how is the aim of describing and explaining all social behavior to be realized? He claims it will be realized by means of an eclectic progression of argument from evolutionary studies (in which the "evolutionary prime movers" of socialization and social bonding are to be identified) through ecology (in which the environmental circumstances determining the course of selection for and against social systems and social behaviors will be identified) and population biology (in which the genetic variation which determines whether a given population of organisms *can* achieve a certain form of social behavior is studied) to sociobiology proper, which studies the selective advantage and genetic basis of social behaviors and behavioral tendencies.

Let me give a simplified illustration of sociobiological reasoning based loosely on the real and hypothetical examples of Wilson's second chapter. Consider a population of monkeys whose recent evolutionary forebears lived both in uniform, food-rich environments and in "patchy" environments – i.e., environments in which occasional very rich food sources are scattered among what are otherwise subsistence-level resources. On the basis of the evolutionary history of such a group (perhaps supplemented by population studies of their forebears and related groups) one would expect the monkeys to exhibit "graded" or "scaled" social behavior, adopting strategies which are optimally efficient in different ecological circumstances. Depending on the specifics of the case, one might expect, for example, that when other ecological factors (e.g., predation) do not intervene, a population which found itself in a uniformly rich environment would exhibit strong territoriality, with males excluding other males while enticing females to join them. Food not being a limiting resource, in populations of the right constitution this strategy maximizes the male's expectation of reproductive success. On the other hand, in a poor but patchy environment in which the likelihood of obtaining maximal benefit from the ephemeral and scattered rich sources of nourishment is increased by group search tactics, the monkeys would be expected to form cooperative bands or troops with fairly strong dominance hierarchies.

In spite of being drastically simplified, the example illustrates typical patterns of sociobiological reasoning: a determination of genetic capabilit-

ies by means of evolutionary theory, evolutionary history, and appropriate population studies yielding a determination of behavior or behavioral tendencies via scaled response to critical environmental variables.

To show the scope and extent of Wilson's ambitions for sociobiology, let me add a few quotations from his recent book, entitled *On Human Nature*[4] (hereafter *OHN*).

> What is truly new about sociobiology is the way it has extracted the most important facts about social organization from their traditional matrix of ethology and psychology and reassembled them on a foundation of ecology and genetics studied at the population level in order to show how social groups adapt to the environment by evolution.... We have begun to construct and test the first general principles of genetic social evolution. It is now within our reach to apply this broad knowledge to the study of human beings. (pp. 16-17)

As examples of the distinctive traits of humans calling for sociobiological explanation, Wilson borrows a list from the anthropologist George Peter Murdock:

> Age-grading, athletic sports, bodily adornment, calendar, cleanliness training, community organization, cooking, cooperative labor, cosmology, courship, dancing, decorative art, divination, division of labor, dream interpretation, education, eschatology, ethics, ethnobotany, etiquette, faith healing, family feasting, fire making, folklore, food taboos, funeral rites, games, gestures, gift giving, government, greetings, hair styles, hospitality, housing hygiene, incest taboos, inheritance rules, joking, kin groups, kinship nomenclature, language, law, luck superstitions, magic, marriage, mealtimes, medicine obstetrics, penal sanctions, personal names, population policy, postnatal care, pregnancy usages, property rights, propitiation of supernatural beings, puberty customs, religious ritual, residence rules, sexual restrictions, soul concepts, status differentiation, surgery, tool making, trade, visiting, weaving, and weather control. (p. 22)

With regard to such social (or, better, *cultural*) traits as these, Wilson says that his theory, to be successful,

> must not only account for many of the known facts in a more convincing manner than traditional explanations, but must also identify the need for new kinds of information previously unimagined by the social sciences. The behavior thus explained should be the most general and least rational of the human repertoire, the part furthest removed from the influence of day-to-day reflection and the distracting vicissitudes of culture. (p. 35)

In the remainder of that book, Wilson offers schematic explanations of the following major human traits, among others: aggression, sex roles (including homosexuality and differential status and roles for males and females in the workplace), altruism, and the social role of religious institutions and the tendency toward religious belief. As we shall see below, acceptance of such lists of traits as the proper *explananda* for sociobiology raises major difficulties.

I should add that, although Wilson is responsible for the major synthetic text in sociobiology and for popularizing the ambitions of his discipline, those who call themselves sociobiologists fall roughly in two camps – a pro-Wilson and an anti-Wilson camp.

The anti-Wilson camp tends to resist sweeping synthetic generalizations about such supposed social traits as aggression, division of labor, mating systems, etc., in favor of specific study of specific social behaviors in specific groups of animals. They deplore Wilson's willingness to allow some sociobiological explanations to employ *group* selection, albeit in relatively limited ways. Their account of their discipline is roughly the following: sociobiology is the attempt, by now well-founded, to explain social behaviors – especially those which *prima facie* benefit the group rather than the individual – in terms of *individual* selection. There are a number of behaviors – e.g., self-sacrifice, foregoing reproduction – which seem not to be in the genetic interest of the individual. (How can an animal increase the proportion of the genes it received from its parents in the next generation if it leaves *no* offspring?) The explanation of these behaviors has long been a problem of considerable importance to evolutionary biology.

The outstanding accomplishment of sociobiology, according to these thinkers, is that it has explained a great many such behaviors on the basis of *individual* selection. In the powerful quantitative theory of kin selection (which has been especially successful in its application to the very extraordinary genetic systems of social insects), sociobiologists *have* offered a powerful solution for many such difficulties. If by foregoing reproduction, an animal can sufficiently increase the number of close relatives produced in the next generation, more of its genes (technically, more genes identical by descent with the genes it received from its parents) may survive in that

generation than if it had reproduced without the support of a socially organized community. Thus in specifiable circumstances, which seem to be met quite cleanly in certain cases in the ants, bees, and termites, an animal is more likely to increase the representation of its genes in the next generation if it is neuter than if it is capable of reproduction. Explanations of this sort, based on individual selection, are at the heart of many accounts of specific behaviors in sociobiology.

GENETIC DETERMINISM IN SOCIOBIOLOGY

Returning to the main argument, let us inquire after the role which (Wilsonian) human sociobiology assigns to the genes in the determination of social and cultural traits. There is an immediate problem: at times Wilson writes as if certain traits and tendencies, such as might appear on a corrected version of Murdock's list, are genetically fixed in human nature, and at times he speaks as if it is *differences* in traits — like being blue-eyed versus being browned-eyed — which are genetically determined. I think that only the second way of talking is legitimate. *All* expressed traits are the result of epigenesis. That is, they are produced by interaction of the genetic system with the environment. On the other hand, it is at least sometimes true that *differences* in expressed traits are due to differences in genes and not differences in the environment. Until I indicate otherwise, I will interpret the claim that a trait is genetically determined as the claim that its difference from some alternative is caused by a genetic difference.[5]

At times, Wilson formulates genetic determinism quite weakly. He allows, for example, that ethological, ecological, and genetic factors all have some effect on social traits, and says that a trait counts as genetically determined if it "differs from other traits at least in part as a result of the presence of one or more distinctive genes" (*OHN*, p. 19). But since this definition says nothing about the *extent* of genetic influence on the trait in question, nearly all relevant traits are likely to fit this description.[6] In any case, this weak version of genetic determinism makes genetic determinism compatible with environmental and cultural determinism: on the present interpretation, "the genes cause (some particular) social behavior" is compatible with "the environment (or culture or whatever) causes (that particular) social behavior." For this reason, a sociobiology which limited itself to this weak version of genetic determinism would be innocuous and uninteresting.

What supposedly gives sociobiology its bite is a strengthened form of genetic determinism according to which, given the environmental factors which the relevant organisms (here, humans) invariably encounter, certain behaviors, or the tendencies to manifest those behaviors, are fixed by specific genetric factors. According to this form of genetic determinism, the behaviors in question (or, in the case of tendencies, the proportion of the population which manifests them in given circumstances) can be wholly accounted for by the neo-Darwinian theory of genetic selection, employing appropriate ecological, ethological, and natural-historical input. As I understand Wilson, he hopes to employ just such an account of the origins of our behaviors and of the strengths of our tendencies to manifest them as the principal tool in calculating social policy with respect to aggression, altruism, criminality of various kinds, sexual roles, and so on.[7] Thus Wilsonian genetic determinism claims *not* that the genes determine social behavior *by themselves,* but rather that they set limits on behavior (and thus on human nature) and establish *costs* (in terms of the degree, difficulty, and disruptiveness of the required environmental intervention) for altering that behavior.

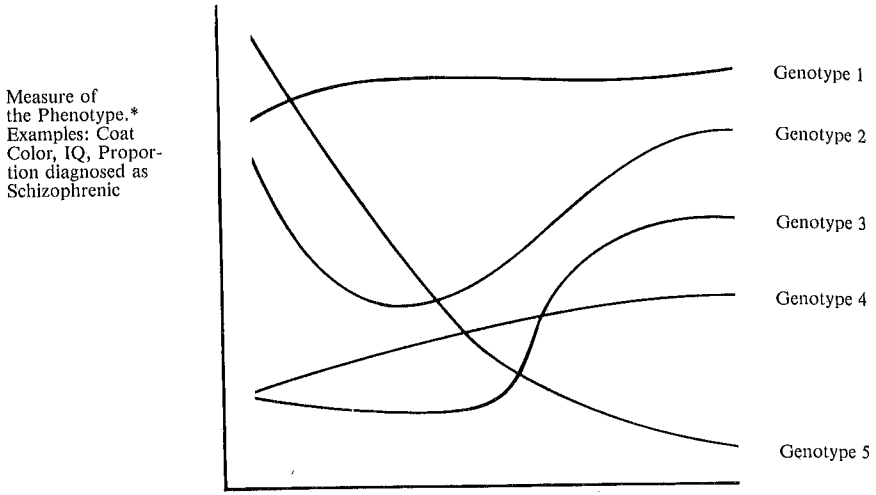
DIFFICULTIES WITH GENETIC DETERMINISM

Let us examine more closely the precise degree of genetic determinism which is claimed in typical sociobiological arguments. As I have just indicated, it is *not* typically claimed that our genes flatly determine altruism, criminality, intelligence, sex role, social role, or various specific components of these social characteristics. But a person's genetic constitution, so the argument runs, introduces strong dispositions which can, with varying degrees of difficulty, be altered by environmental intervention. In straightforward cases, each gene or gene complex has a so-called norm of reaction.[8] The degree to which a trait is genetically determined is a relative matter, ascertainable only by a complex series of comparisons. Frequently, the correlation between possession of different genotypes within a population and possession (or degree of expression) of an affected trait is taken to be a measure of the degree to which the trait is genetically determined. I shall now show that such correlations are an inadequate measure of the degree to which a trait is genetically determined, and that they do not yield real knowledge of whether, or to what degree, the behaviors one is interested in are, in fact, genetically determined.

To start with, covariance of genotype and phenotype does *not* establish

FIGURE ONE

NORMS OF REACTION



Examples: Highest diurnal temperature during coat growth, degree of "educational enrichment," scaled measure of "family cohesiveness."

*This measure is a *population* measure – e.g., % of organisms in the population exhibiting the trait or the average magnitude of the trait in the population (with the specified genotype).

the degree to which a trait is determined by the genes *except* with respect to the genetic and environmental backgrounds in which the trait was measured.

As an example, consider height in humans, a fairly heritable trait. Heritability, written "h^2," is the standard measure of the correlation between genotype and phenotype. Suppose (as I believe is approximately correct) that the heritability of height in America and in Japan is 0.8. (It is often said that this means that 8/10 of the variance in height between individuals is accounted for by genetic factors. But, as I shall show, this has clear meaning only *within* a population and within a specified environment.) Does this mean that 80% of our height is determined genetically? *NO.*

Transplant fifty families from America to Japan and fifty families from Japan to America and provide them with the local dietary regimens and there is a major difference – somewhere around three to six inches! – in the heights obtained in the next generation as compared with the heights expected if transplantation had not taken place. Heritability measures the contribution of the genes which have been singled out only *within* a population with the genetic and environmental backgrounds held reasonably constant.[9] Because of this hidden dependence on background conditions whose precise role in achieving the traits in question is unknown, it is not clear whether a clear meaning can be given to the claim that the genes, or some complex of genes, determines – or determines n% of – a given trait.

To put the problem more generally: the gap between genotype and phenotype is immense. In humans, each parent contributes a few thousandths of a gram of DNA, strung together in twenty three molecules whose total unfolded length is about a yard. To have an effect on the adult phenotype, a gene must interact with other genes, with the chromosomal structure of the cell, with various components of the nucleus, with the chemical make-up of the cytoplasm, with the physical conditions affecting the cell, with the timing of differentiation and development, with the tissue structure of the developing organism, and with the alterations which the external environment effects on all of these. Thus *all* development is epigenetic – i.e., involves an inextricable mixture of genetic and non-genetic factors.

Again, a special class of interactions illustrates the indirectness with which a gene typically affects phenotypic traits – to wit, the multiplicity of interactions among genes. The genetic complexities here include epistasis, pleiotropy, modifier genes, switch genes, variable penetrance, insertion sequences, genomic rearrangements, and so forth. No gene has its characteristic effect without the cooperatioin of a multitude of other genes.

The dependence on multiple interactions and multiple pathways reinforces the point that if one changes the genetic or the environmental background, a given gene or gene complex may yield startlingly different effects. This substantiates the major point which I set out to establish, namely that *the within-population correlation between genotypic and phenotypic characters does not measure the degree to which the genes or genotypes in question cause the trait or traits in question.*[10]

At first glance this claim may seem to be a narrow one about genetics. But it will later become clear that it is a special instance of a general methodological point: causal ascriptions in complex systems are extremely sensitive

to the treatment of background conditions, to the way in which the boundary between the system and its environment is drawn, and to the way in which the system is analyzed into parts.

DESCRIPTION DEPENDENCE

Before considering how one might defend genetic determinism against this criticism it will help to face a second, closely allied difficulty mentioned in the introduction of this paper – to wit, description dependence. *The adequacy of a causal model depends on the way in which the supposed causes and effects are described.* Let us look a bit more closely at the standard example of phlogistic chemistry.[11]

When heating the red calx of mercury in a sealed vessel, thereby transforming it into mercury, Priestley noticed that the air contained in the vessel was altered. After performing appropriate tests (and a couple of false starts), he determined that it had been transformed into "dephlogisticated air," i.e., air from which "phlogiston" had been removed. Now phlogiston was supposed to be both a metallic principle and the substance of heat, so its removal meant (a) that the resultant gas would have a high affinity for phlogiston (that is, would support combustion extraordinarily well), and (b) that the absorption of the phlogiston would make the calx metallic – i.e., convert it into mercury.

It turned out, over the long haul, that this plausible story could not be converted into a satisfactory explanation of oxidation, combustion, and the common chemical properties of metals *because of a mismatch between Priestley's descriptive categories and the constituents of the actual reactions he wished to explain.* A satisfactory account of the common properties of metals was not available for a long time – but Lavoisier's remarkable accomplishment was to provide new descriptive categories and terminology which proved capable, over time, of allowing satisfactory (if imperfect) explanations of oxidation, combustion, and many further features of chemical reactions.

Situations like this are more common than has been generally recognized. It is the sort of thing which worries Richard Lewontin in a major theoretical work on the limitations of current genetic theory.

> The problem of theory building is a constant interaction between constructing laws and finding an appropriate set of descriptive state variables such that laws can be constructed. We cannot go out and describe the world in any old way we please and then sit back and demand that an explanatory and predictive theory be built on that description. The description may be dynamically insufficient.[12]

It is only when we have described the "units" of behavior, if any, which correlate nicely with the ultimate effect of the genes (or of major units within the genotype) that the observed correlations can be used to achieve a deep understanding of the evolutionary significance of the behavior and of the degree to which it is controlled by the genes. Without this knowledge, we do not have the basis for estimating the *genetic* obstacles to particular social policies regarding the alteration of behavior any more than Priestley had a way of estimating the *weights* of the products of a chemical reaction. I know of no good grounds to suppose that the behaviors, if any, which correlate well with genetic constitution are those which are of social interest. I very much doubt that they are as generalized as "aggressiveness," "age grading," "courtship," "tendency to religious belief," "sexual division of labor," and so on.

In short, if one means by a "trait" a property which is genetically significant, one must establish that the properties one wishes to explain are, in fact, traits. In the case of human traits of interest to social scientists, sociobiologists have failed to do this.

OUR KNOWLEDGE OF WHAT THE GENES DETERMINE

I have been arguing that when one starts with an arbitrary description of a phenotypic property, it is not safe to assume that there is a univocal and clearcut genetic contribution to the development of that property and that, in the specific instance of human behavioral phenotypes, even when this assumption is correct it is unusual to be able to disentangle genetic from environmental contributions in any useful way. Unless such a trait behaves in simple Mendelian fashion (and how many socially significant behavioral traits do that?), the best that one can normally expect from present phenotype-down genetics, properly pursued, is the identification of an inextricable morass of contributing factors of a great variety of types, factors whose interactions depend on various unknown and unspecified boundary conditions.

It is appropriate to ask if the situation is any better when one starts from the DNA and works one's way "up" to the phenotype. Unfortunately, at present the situation is just about as bad for behavioral phenotypes when

one starts from the DNA as it is when one starts from behavior.[13] With few exceptions, what we have learned from the study of DNA in higher organisms has to do with which proteins they make when transcribed and translated. Note that the production of protein is thoroughly epigenetic in that a structural gene cannot make protein by itself, but only provides the information which will be used if the gene is "switched on." Nor will the proper result be achieved unless the cellular environment falls within certain limits.

Occasionally we know a fair amount about complicated biosynthetic pathways and the function of certain proteins in metabolism or in specific tissue systems. We know something about gross behavioral and morphological disturbances caused by the lack or by excess of various compounds. But, although we know a little bit about the signals which turn genes on and off and a good deal about the mechanisms by means of which the information on structural genes is converted into protein, we do not have anything resembling satisfactory understanding of the regulatory system by means of which development is controlled.

We do know that the regulatory system has epigenetic components, but we do not know how the quantities, types, and locations of proteins are controlled, nor how developmental processes are kept in the proper sequence so that, within a broad range of environmental conditions, the programmed morphology is achieved. We do know that some factors serve as developmental triggers. For example, the proper combination and sequence of juveile hormone and ecdysone trigger metamorphosis in certain insect tissues. But this does not tell us how, once the maturation of adult tissues has begun, the "right" morphology is achieved. We do not know how the sequence of events within a cell and within a mass of cells fated to become, say, an insect wing is controlled in the face of considerable environmental variation to yield a tissue of a certain shape, with boundary characteristics of a certain kind, cellular geometries of appropriate sorts, appropriate concentrations of proteins, lipids, organelles, and so on, and appropriate sensitivities to chemical and electrical signals, etc.

We do know that in humans only about 1/2% to 5% (some new estimates are higher) of the genome codes for structural proteins, i.e., for those proteins employed in building cells and tissues. It is a good guess that a fair portion of the remaining genes play some role in regulating the processes involved in achieving the correct sequence and degree of gene expression.[14] But we have virtually no idea of the large-scale regulatory controls involved nor of the respective roles of genetic and epigenetic factors in achieving regulation.

Our ignorance of the pathways from genes to morphology is great. Our

ignorance of the pathways from genes to behavior, pathways which surely vary with differences in the achieved morphology, is even greater.

A USEFUL EXAMPLE

I would like to sharpen and develop my claims by working with an example. If ever there was a clean case of genetic determination in humans, it is found in the so-called genetic disease or genetic deficiency known as phenylketonuria, or PKU for short.[15] Discovered in the 1930's by the Norwegian physician and biochemist, Ashbørn Følling, PKU was initially described as a behavioral disease brought on by an inborn error of metabolism, a specific kind of biochemically induced severe idiocy, often associated with varying degrees of autistic behavior and marked by the reaction of urine with $FeCl_3$ to produce a characteristic green color. Følling was able to show that the color of the reaction was produced by phenylpyruvic acid, an unusual metabolite, not normally excreted in the urine.

By the 1950's, PKU was fairly well understood. Victims of the disease are normal or nearly normal at birth. However, they are unable to metabolize phenylalanine, one of the essential amino acids. This inability is due to the absence of a specific enzyme, found primarily in the liver of normal humans, phenylalanine hydroxylase. The absence of the enzyme is, in turn, correlated with the presence, in double dose, of a non-sex-linked recessive gene which either fails to produce any enzyme or produces a modified enzyme totally unable to catalyze the digestion of phenylalanine. The normal enzyme, in the normal biochemical context, converts phenylalanine to tyrosine, another essential amino acid. So the victims of PKU are flooded with phenylalanine and starved for tyrosine. This affects their metabolism in a variety of ill-understood ways and somehow affects brain cells. The specific effects are not well understood, but they include severely abnormal formation of myelin, the protective sheath of nerve cells which plays a prominent role in brain physiology. Since tyrosine (the amino acid whose concentration is abnormally low) is required for pigment formation, most victims of the disease are blond and blue-eyed. They are also ill-nourished in a variety of ways and tend to have reduced growth: most untreated phenylketonurics fall in the lowest 10% in stature.

The inheritance of PKU followed a classic Mendelian pattern. In fair-skinned populations (e.g., Scandinavians), about one birth in 20,000 or 25,000 exhibited the symptoms of the disease. About one out of four matings between known carriers of the gene yielded symptomatic offspring. The statistics in the 1950's looked very Mendelian. At this point, enough was

known about the disease to attempt to locate children at risk by mass screening and to attempt to treat them. The results are mixed and rather complicated, but fascinating. Let me sketch some of them before I draw some morals from my study of this condition.

1. When one defines PKU by current biochemical criteria, very few phenyleketonurics, probably fewer than 5%, escape autism, related behavioral disorders, and severe retardation. These biochemical criteria are, however, quite stringent, requiring that the blood serum concentration of phenylalanine be above ten times the normal level. A high proportion of people with relevant genetic abnormalities manifest serum levels of from two or ten times normal – and these are at much lower risk for exhibiting behavioral disturbance or severe intellectual impairment.[16]

2. The greatest damage occurs in the period from birth to age six months (or perhaps one year) and is largely irreversible.

3. Most of the damage can be controlled by severe and carefully controlled restrictions of phenylalanine intake. Thus the way the condition is described is important. If it is identified by its behavioral effects and the physiological damage involved, an environmental cure is possible. If, instead, PKU is identified, as is now standard, by its metabolic characteristics or in terms of the genetic constitution of the individuals involved, it is possible to render most victims of the disease asymptomatic by environmental intervention.

4. Dietary control which keeps blood serum levels of phenylalanine within the range normal for people who do not suffer from the disease results in a fair proportion of cases in which the patient suffers from phenylalanine or generalized protein starvation. Such starvation has very serious effects – sometimes including death.

5. Dietary control which keeps phenylalanine serum levels at two or three times the normal seems to be most effective in alleviating symptoms and reducing side effects.

6. There is extreme variability from subject to subject in the intake of phenylalanine per kilogram of body weight which achieves the desired serum level and considerable variability in the level of serum phenylalanine at which behavioral disturbances, intellectual impairment, or abnormal myelin formation may occur.

7. This variability seems to be due to the different degrees to which different subjects mobilize secondary pathways for transforming phenylalanine into other substances, the particular concentrations of such substances which result, and the particular tolerance for such abnormal concentrations which different individuals have.

8. There are a large number of conditions which either mimic PKU to a high degree, or are aberrant forms of the disease. About one-third of the cases found on initial screening do not meet the requirements now set for "classic" PKU. Some of these involve transitory developmental episodes in which phenylalanine metabolism is temporarily shut down (often without detectable damage) or chronic, but incomplete blockage of the pathway from phenylalanine to tyrosine. Others are chronic. PKU with abnormal forms of expression is sometimes hard to distinguish from these related metabolic disorders.

9. Even siblings who are affected with the disease exhibit quite different metabolic patterns, quite different tolerances for the abnormal metabolites involved, and quite different effects from particular serum levels of phenylalanine and tyrosine.

THREE MORALS AND A THOUGHT EXPERIMENT

This brief review of current knowledge of PKU illustrates the two methodological claims for which I have been arguing. One was that the success of the causal analysis of a condition or an occurrence (here, specifically, a genetic analysis) depends on the way we describe the supposed effects. Although there is unquestionably a Mendelian autosomal recessive gene involved in PKU, unless the effect is described narrowly as blockage of the metabolic pathway from phenylalanine to tyrosine by failure to produce an active form of pheylalanine hydroxylase, the disorder does not behave in Mendelian fashion (and perhaps not even then). Thus if one defines the condition by the conjunction of its behavioral and morphological symptoms with the colorful reaction of urine with $FeCl_3$, it is debatable whether the condition *as thus characterized* should count as genetically caused. It is certainly clear that when PKU is delimited in this way, the recessive gene, in double dose, *need not bring about* PKU, for environmental *prevention* is possible. Given the genotype, the symptoms will occur only if a variety of ill-understood (and mutually interacting) environmental and genetic factors are present during early development. To be sure, some combination of these factors is, in fact, *normally* present. Even though we don't know what they are, we *assume* their presence when we label PKU as a genetic deficiency or as an inborn *error* of metabolism.

This last sentence points to the second moral mentioned above, namely the sensitivity of causal ascriptions to the delimitation of the system under investigation and of the (normal) environment of that system. An analysis of genetic determination requires a judgment (perhaps tacit and unthinking)

regarding what is to count as the normal environment of the relevant population and what are to count as the boundaries of the genetic system and the way in which it will be analyzed into parts. Only when these matters have been settled can one properly determine which effects to ascribe to a gene or a genotype.

To drive this point home, consider a simple thought experiment. Suppose that most people – say 95% – are phenyleketonurics but that most human diets include appropriately low levels of phenylalanine and high levels of tyrosine. Then penylalanine and substances like meat with high phenylalanine content will be seen as environmental poisons and those lucky few who have the now-normal gene coding for phenylalanine hydroxylase will be seen as highly tolerant, for partly genetic reasons, of those poisons, as some of us are highly tolerant of arsenic.

Here we see clearly the need to delimit the normal state of the population and the normal environment (or range of environments). Mere genetic analysis of an individual who lacks a gene coding for phenylalanine hydroxylase does not show that he has a genetic deficiency or an inborn error of metabolism. *By itself* such a deficiency is no more startling than the lack of a gene to metabolize arsenic harmlessly. What makes the lack count as causally significant with respect to a functional characterization of the phenotype is the ecological judgment that foodstuffs high in phenylalanine belong to the normal environment, cannot be avoided, and must be digested, while foodstuffs rich in arsenic do not belong to the normal environment or are avoidable. Thus the ecological reference system – that is, our delimitation of what belongs to the normal environment and what counts as the normal state of the population – is presupposed or built into the ascription of genetic causation. Whether a claim that a certain gene or genotype causes a certain effect is correct or not depends on the population considered and on the conditions which are held constant. We see, therefore, that the success of causal ascription in genetics depends on the reference system employed as well as the way in which the supposed effect is described.

This discussion also points to a third moral as well, one which deserves full discussion but which can only be adumbrated briefly here. It is that *as our theoretical knowledge changes, the phenomena which we investigate are transformed.* We have seen that the drawing of system boundaries, the division of the system under investigation into parts, the characterization of the normal range of environments, and the descriptive categories employed interact with one another and affect the validity of causal ascriptions. It is inevitable that such changes will occur with increasing knowledge of the interactions of ill-understood complex systems with their environments. As

they do so, the classification and delimitation of the phenomena under investigation will also change. This transformation of the phenomena, exemplified by the shift from dephlogistication to release of oxygen, is an essential ingredient in the "constant interaction between constructing laws and finding appropriate descriptive state variables" to which Lewontin referred. It can be expected to be of the greatest importance where the gap between the "theoretical" system and the phenomena which that system is to explain is the largest. As should be clear, this describes precisely the situation which obtains in human behavior genetics.

A SECOND FORM OF GENETIC DETERMINISM

Before summarizing the upshot of this discussion for behavior genetics, however, there remains a loose thread to take care of. I have been working with what I think is Wilson's official account of the genetic determination of a trait, according to which a trait is genetically determined if and only if its differences from other traits are caused by genetic rather than environmental differences among the organisms carrying the trait in question (model: blue eyes versus brown eyes). I have argued that, by using everyday and social-scientific descriptions of social "traits," current sociobiology is incapable of separating the contributions of the genes from the contributions of culture and the environment.

I am grateful to Professor Robert Richardson of the University of Cincinnati for finding a lacuna in my argument. There is a second form of genetic determinism employed by Wilson and many other sociobiologists. I must show that it does no better than the first.

The point of the list of universal (human) socio-cultural traits which Wilson borrowed from Murdock was not to establish genetic correlations with *variations* in human social practices, but to exemplify *in*variants in the human species – and to argue that these are the highly canalized consequences of genetic (rather than, say, cultural) factors. This suggests an alternative doctrine of genetic determinism, one which has been articulated on occasion by Ernst Mayr and others, according to which a trait which is invariant in a population or species is genetically determined if it is the normal phenotypic expression of an underlying genotype whose effects are highly canalized.[17] Obviously there are phenotypic universals which it is natural to treat as genotypically determined in this sense. Examples in humans include the ability to learn language and bipedal locomotion.

Equally obviously, in humans at least, there are phenotypically universal tendencies which it is implausible to treat as genetically determined.

Examples include the tendency to get *4* as the result of adding *2* and *2* and the tendency to locate one's domicile within an hour's commuting time of one's workplace. I suspect that the difference is our intuitive (and quite possibly mistaken) belief that these latter tendencies could be readily altered by environmental intervention. But for the aggregated traits which interest social scientists and social policy makers, our intuitions are of little use. We do not, after all, have direct genetic knowledge of the relevant genotypes, if there are any. That is, we cannot characterize the supposed genes or gene complexes underlying tendencies to, say, "aggression" or "xenophobia" independently of those tendencies and we have, at present at least, no satisfactory way of telling whether such tendencies rest on any sort of unitary genetic foundation.

Accordingly, we must first find out how to use our knowledge of the evolutionary history of man and of the genetic system to separate those tendencies which are culturally determined, which are learned responses to the environment, which are environmentally plastic, or which stem from generalized abilities to imitate and figure out from those tendencies which are the unavoidably canalized phenotypic expression of genotypes which have been driven to fixation by natural selection.

But to solve *this* problem we must solve the problem of finding the right descriptive categories (Lewontin's "appropriate set of descriptive state variables") to employ in describing behaviors. *That is, we must match our descriptions of behavior to the phenotypic units which have, in fact, been fixed by natural selection.* It should be clear by now that, when we start by employing descriptions of social behavior from everyday life, social science, or social policy, it is unlikely that our descriptions are well-matched to genetic mechanisms. The problem of finding genetically relevant descriptions is virtually untouched; we have not yet conquered the gap between the genotype and behavioral phenotype, at least, not for humans.[18]

THE PROSPECTS FOR HUMAN BEHAVIOR GENETICS

It seems only fair, having come this far, to step out on a limb and bring these considerations to bear on the prospects for human behavior genetics. I do so with some trepidation, for the matter cannot be settled *a priori,* but only in light of appropriate knowledge of the field — and I do not know enough about ongoing work to have a clear and reliable overview of its accomplishments, its strengths, and its weaknesses. Nonetheless, applying my argument to such behavior genetics as I have read suggests the following considerations.

1. It is unlikely that there is a simple genotypic basis, like that for PKU, for such complex, composite social categories as "aggressiveness," "altruism," "sex-role differentiation," and so on. If we wish to delve into the degree to which normal behaviors are genetically determined, it would be wiser to start by looking for model behaviors which behave in a Mendelian manner (as, in first approximation, PKU does), but which are not connected to gross distortions of behavior or gross genetic abnormalities (like, alas, the feeblemindedness and autism of PKU). So far as possible, investigations of human behavior from the phenotype should begin with traits meeting these constraints. From what I gather, few such behaviors are known in humans and higher mammals, and their connection with socially interesting behaviors is *terra incognita.* This means that most traits of social interest are not currently susceptible to a reliable or robust genetic analysis. There may be some exceptions: for example, there is some chance that we are near to having the basis for a genetic analysis of stuttering.[19] But, to repeat Lewontin's earlier warning, in general, we cannot describe the world in any old way we please and then sit back and demand that an explanatory and predictive theory be built on *that* description.

The most important task for phenotype-down behavioral genetics in higher mammals is to arrive at units of behavior to which the genotype is maximally relevant. It remains wholly unclear whether any of these fit the currently available frameworks for describing socially significant actions,[20] what they are, and how relevant they are to the behaviors which sociobiologists would like to explain.

2. To carry out the requisite genetic analysis, it is necessary to fix the genetic and environmental backgrounds against which alternative genetic conditions are examined. But in the case of human social behaviors, we cannot and ought not carry out experiments which might reveal the relevant norms of reaction — namely, experiments in which many individuals with (nearly) identical genotypes are exposed in a controlled way to specifically different environments. Furthermore, we cannot and should not control the environments in the relevant ways: we cannot make the training of individuals truly uniform, we cannot fix the social institutions they encounter, we cannot fully regiment the roles played by the agents they encounter, and so on. Nor can, or should, we prevent people from choosing more congenial environments if the ones which are experimentally useful prove distasteful to them.

Still further, we do not have any clear knowledge of which factors belonging to the "normal" environment are most significant in shaping the development of social behavior. It is obvious that various aspects of culture

– nurturing, language-learning, schooling, exposure to various forms of social interaction – are relevant to the determination of behavioral normality and must be included in the reference system as parts of the "normal" environment. But which aspects of which cultures should we treat as normal? Which aspects should we vary to establish norms of reaction? How do we isolate the various cultural factors relevant to, say, "aggressiveness" and how do we divide the genotypes which we wish to test when we have neither an adequate classification of the relevant cultural factors nor any independent means of picking out the relevant genotypes? Yet, as I have shown, until we can identify the *relevant* factors which are held constant for the population, we will be unable to separate the contribution of the genes to the formation or manifestation of a behavior pattern from the contribution of environmental factors.

3. In animal studies, where one can fix the genetic and environmental backgrounds, it will be possible to assign definite heritability values to certain behaviors and to localize the genes which, *against the fixed background,* influence the traits in question. But until one knows the specific pathway by which the tendency to behave in the specified way is brought about, it will be very difficult to extrapolate such findings reliably. If we wish to learn whether the particular genes in question retain their effects against different genetic backgrounds or in different environmental circumstances, we must evaluate the sensitivity of the genetic control of the behavior involved to specific kinds or ranges of variation. In somewhat more technical language, we must learn how well the genetic control of a particular behavior is canalized, i.e., how well it is buffered against variations in the environment and in other parts of the genotype. Even when control of a behavior is highly canalized, there may be highly specific environmental factors which will alter the behavior in specific ways or release it from rigid genetic control – recall the role of reduction of phenylalanine intake in control of PKU.

SUMMARY AND CONCLUSION

It is time to take stock of my argument. I shall summarize a few central points and then comment very briefly on the strength of my results. First, I have argued that if sociobiology is to be of any general interest, it must maintain that the behaviors it studies are rather tightly controlled by the genetic constitution of the organisms under investigation. Although it allows considerable importance to environmental circumstances and

triggers, in the end, it must treat environmental variation as sufficiently circumscribed for the genes to retain fairly tight control of behavioral responses in different environments. Otherwise the role of genetic selection, and hence of the genes, in the determination of behavior is too diffuse to be of theoretical significance in the social sciences or of practical importance in matters of social policy.

Second, I have argued that we have very little knowledge of the pathways by means of which genes influence behavior, at least in higher mammals and humans. Nor do we know which particular social behaviors, if any, are under the relatively tight sort of genetic control required to make applied sociobiology worth paying attention to. I have shown that such measures as heritability are inadequate indicators of the "tightness" of the genetic determination of traits. I have suggested that the more we "lump" behavioral traits under generic descriptions like "aggressiveness," "homosexuality," "intelligence," "differentiation in sex-role," and so on, the less likely it is that the behavior thus described is under fine-grained genetic control.

Third, I have argued that there are cases in which the *way* we delimit the system of concern will substantially affect the validity of our ascription of causal efficacy to genetic and environmental factors. If particular environmental circumstances or interventions can alter the effects of the genes (as is the case in PKU), then by delimiting the system of concern widely enough to include the relevant environmental variation we alter what counts as the effects of the genes. Given our immense ignorance of pathways leading from the genes to social behaviors, this consideration is sufficient, by itself, to undermine available arguments for both of Wilson's (strong) versions of genetic determinism for social behavior: the "traits" in question need not be, and typically are not, universal among humans and it has not been shown that the differences in the traits manifested by different individuals are due to genetic rather than environmental differences.

Fourth, among the difficulties which applied sociobiology must overcome, the following loom large: the immensity of the gap between genotype and phenotype, the extreme complexity of the pathways from the genes to behavior, the uncertainty about the units of behavior, if any, which are under moderately straightforward genetic control, the probable mismatch between socially significant descriptions of behavior and the genetically relevant units of behavior, the uncertainty about which environmental and genetic factors should be counted as part of the "normal" background in assessing the effects of particular genes, and the moral insupportability of (and physical constraints governing) experimentation directed at determining the genetic and environmental backgrounds relevant to human

behavior. In the near future we shall *very* seldom be in a position to separate the contribution of genetic factors from that of non-genetic factors in behavior of social importance.

How powerful are these results? Well, they certainly do not show that a science of sociobiology is impossible. At most, they show that we have little reason to expect sound, large-scale applications of sociobiological reasoning to higher mammals or to humans in the near future. Nor do they show to what extent our behavior is – or is not – genetically determined. Rather, they show that we do not at present have adequate means of finding out which social "traits" are genetically determined and that our evaluations of genetic determination are often conceptually confused. My arguments have also shown that our present knowledge does not support any strong claims, one way or another, about the precise genetic limits placed on human social behavior. Accordingly, we cannot soon expect to realize Wilson's dream of estimating the genetic feasibility – or the genetically imposed cost – of specific social reforms in the service of social policies aimed at altering sex roles, class differences, criminal behavior and so on.

Stepping back a little further from the argument, I believe that I have undermined the claim that current sociobiology can utilize the genetical theory of natural selection to shed critically new light on human nature. I have done so not by arguing *a priori* or by starting from grand philosophical premises, but by a methodological argument. Thus I have shown the importance and value of strictly methodological evaluation of scientific works which purport to have deep social and philosophical ramifications. It seems to me that there ought to be much greater attention paid to such methodological arguments than is now common since, as we have seen, they can undermine the pretentions of science which has overreached its knowledge base, while directing research into more fruitful channels.[21]

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FOOTNOTES

1 Cf., for example, the six major anthologies on this topic:
Arthur Caplan, ed., *The Sociobiology Debate* (New York: Harper and Row, 1978); Michael S. Gregory, Anita Silvers, and Diane Sutch, eds., *Sociobiology and Human Nature* (San Francisco: Jossey-Bass, 1978); Gunther S. Stent, ed., *Morality as a Biological Phenomenon* (Berkeley and Los Angeles: University of California Press, 1978); Napoleon Chagnon and William Irons, eds., *Evolutionary Biology and Human Social Behavior* (North Scituate, MA: Duxbury, 1979); George W. Barlow and James Silverberg, eds., *Sociobiology: Beyond Nature/Nurture?* (AAAS Selected Symposium, 35) (Washington, D.C.: American Association

for the Advancement of Science, 1980); and Ashley Montagu, ed., *Sociobiology Examined* (Oxford: Oxford University Press, 1980).

2 There is an additional twist which I shall not pursue in any depth. Since our self-characterizations play a significant role in social phenomena, acceptance of a radically restructured descriptive vocabulary will probably alter those phenomena. We may not be free, as Lavoisier was, to alter our descriptive apparatus without altering the phenomena under investigation.

3 E.O. Wilson, *Sociobiology, the New Synthesis* (Cambridge, Mass.: Harvard University Press, 1975).

4 E.O. Wilson, *On Human Nature* (Cambridge, Mass.: Harvard University Press, 1978).

5 After completing the final revision of this paper, I found a closely parallel passage on p.28 of John Maynard Smith, "The Concepts of Sociobiology," in Stent, *Biology as a Moral Phenomenon,* pp. 21-30. See also below, n. 17.

6 Anthony Leeds has pointed out to me that there are many cultural traits for which this claim is not true – e.g., the use of recurved as opposed to unrecurved bows. He rightly suggests that a finer-grained analysis should distinguish between social traits in Wilson's sense (e.g., presence versus absence of an incest taboo) and cultural traits as often intended in anthropology (e.g., the various types of incest taboo). I add that this is grist for the mill of my main argument: there is no way of clearly separating "social" and "cultural" components of traits of interest to social scientists – and hence no way of isolating in pure form a set of socially significant traits (phenotypes) which are determined (primarily) by the genotype.

7 Cf., e.g., *OHN*, pp. 147-48, where Wilson sets forth the target of learning enough about "our innate predispositions" to enter into certain sexual roles to enable us to adjust "the instruments of education and law... concerning sexual discrimination, the standards of sexual behavior, and the reinforcement of the family" in such a way as to (a) know the social cost of whichever policy objectives we might adopt and (b) minimize the human unhappiness which the adopted policy brings with it. "Human nature [presumably as determined by the genes] is stubborn and cannot be forced without cost."

8 A standard introductory discussion of norms of reaction may be found in Theodosius Dobzhansky, *Genetics of the Evolutionary Process* (New York: Columbia University Press, 1970), pp. 36-41 and 291-296. For an important account of norms of reaction and the caution required in employing them, cf. R. C. Lewontin, "The Analysis of Variance and The Analysis of Causes," *American Journal of Human Genetics, 26* (1974), 400-411.

9 For example, individuals with two PKU alleles, discussed below, are of abnormally small stature, but the normal genes which these alleles replace are usually part of the "genetic background" in measurements of the heritability of height.

10 There are two ways to resolve this difficulty of causal ascription, both of which I shall explore more fully later. The first is to determine the *pathways* by which the genes achieve their effects; the second is to explore the degree of canalization of the phenotypes in question.

11 A useful philosophical treatment of this case, including references to appropriate primary and secondary sources on the case history, is Philip Kitcher, "Theories, Theorists and Theoretical Change," *Philosophical Review, 87* (1978), 519-547, especially 529 ff. Cf. also Stephen Toulmin, "Crucial Experiments: Priestley and Lavoisier," in P.P. Wiener and A. Nolan, eds., *Roots of Scientific Thought* (New York: Basic Books, 1957), pp. 481-496.

12 Richard Lewontin, *The Genetic Basis of Evolutionary Change* (New York: Columbia University Press, 1974), p. 8.

13 A basic introduction to behavior genetics is provided by G. E. McClearn and J. C. DeFries, *Introduction to Behavior Genetics* (San Francisco: W. H. Freeman, 1974). A more comprehen-

sive text is John L. Fuller and W. R. Thompson, *Foundations of Behavior Genetics* (St. Louis: C. V. Mosby, 1978).

14 Recent work on "introns" (inserted sequences of DNA at various places within a structural gene) supports this guess. One role which inserted sequences play is to regulate the transport of the RNA transcript of the structural gene through the nuclear membrane and into the cytoplasm, where the revised transcript, with its introns deleted, is "translated" into protein. Cf. Francis Crick, "Split Genes and RNA Splicing," *Science,* 204 (1979), 264-271 or Pierre Chambon, "Split Genes," *Scientific American,* 244 (1981), 60-71.

15 Among the many works on PKU which I consulted, the most useful are Davis S. Kleinman, "Phenyleketonuria: A Review of Some Deficits in our Information," *Pediatrics,* 33 (1964), 123-134; Eugene Knox, "Phenyleketonuria," in J. B. Stanbury, J. B. Wyngaarden, and D. S. Fredrickson, eds., *The Metabolic Basis of Inherited Disease,* 3rd ed. (New York: McGraw Hill, 1972), pp. 266-295; and Committee for the Study of Inborn Errors of Metabolism, *Genetic Screening: Programs, Principles and Research* (Washington, D.C.: National Academy of Sciences, 1975).

16 Cf. "Historical Experience of Screening for PKU" in *Genetic Screening,* pp. 23-43.

17 Maynard Smith in *Biology as a Moral Phenomenon,* p. 26 n.5., makes the parallel point, characterizing the resultant form of genetic determinism as follows: "There are universal characteristics of human societies which exist because of universal features of human nature that evolved by natural selection." Maynard Smith considers this to be Wilson's official account of genetic determinism.

18 Maynard Smith's way with the present form of genetic determinism is short, and, for many traits, decisive. "There are characteristics that are 'genetically determined' in the sense that individuals of a given genotype will develop the characteristic in almost all environments compatible with life.... But the characteristics treated by Wilson as human universals are not of this kind. There are environments in which some human genotypes do commit incest, or are reluctant to participate in ritual acts, or adopt roles atypical for their sex and society ...[T]he postulation of innate, socially relevant characteristics of human behaviors has been taken to imply that almost all human societies will acquire those characteristics, regardless of initial conditions or conscious intentions. I do not know how this implication can be justified, but it certainly cannot be justified by evolution theory." *Ibid.,* pp. 28-9.

19 Cf. Kenneth Kidd, "A Genetic Perspective on Stuttering," *Journal of Fluency Disorders,* 2 (1977), 259-269; and Kenneth Kidd, Judith Kidd and Mary Ann Records, "The Possible Causes of Sex Ratio in Stuttering and its Implications," *Journal of Fluency Disorders* 3 (1978), 13-23.

20 In a personal communication (11/4/80), Michael Ghiselin parodies the excesses of behavior geneticists in this connection trenchantly: "We are told that one can select for 'emotionality,' but it turns out to be the rate of defecation in an experimental setup." Although the particular research which Ghiselin had in mind concerned the behavior of mice, the point is quite general.

21 The present version of this paper owes much to the helpful criticisms and advice of B. Blustein, L. Darden, M. Ghiselin, F. Gifford, M. Grene, A. Leeds, A. Lugg, R. Richardson, A. Rosenberg, and seminar discussions at the University of California, Davis, the University of Pittsburgh, and the University of Western Ontario. I am grateful to all concerned.

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A METHODOLOGICAL ANALYSIS OF SOCIOBIOLOGY

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I

Since the publication of Professor E. O. Wilson's book *Sociobiology: A New Synthesis*[1] in 1975, the field of sociobiology has captured the imagination of much of the general public as well as of the academic world. At the time of its publication the book was proclaimed by *The New York Times* as marking the birth of a new science.[2] Subsequently, magazine articles, interviews with Wilson, articles in biological journals, articles in journals in the social sciences, symposia conducted by various academic disciplines, a high school curriculum entitled "Exploring Human Nature," and even a movie featuring interviews with Wilson, another prominent sociobiologist Robert Trivers, and the physical anthropologist, Irven DeVore, have all been devoted to descriptions, discussions, and sometimes debates on sociobiology.[3] This enormous interest no doubt derives from the ambitious goals of the subject: sociobiology claims to be able to explain the social behavior of animals including humans in terms of underlying evolutionary biological principles. In Wilson's words, "The ultimate goal is a stoichiometry of social evolution" (p. 63).

In his vision of the future of the biological and behavioral sciences, Wilson surmises that in the next hundred years or so cultural anthropology, social psychology, and economics will be incorporated into sociobiology. An understanding of emotions and ethical judgments will be based on a full neuronal explanation of the human brain at the level of the cell. "Cognition will be translated into circuitry" (p. 575). "Having cannibalized psychology, the new neurobiology will yield an enduring set of first principles for soci-