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During the 20th century, many researchers studying heredity sought to apply their findings in the arena of mental health and human (mis)behavior. Many of these have examined, from the perspective of heredity and genetics, the desire to drink and its consequences. In this paper, the author examines hereditary explanations of alcoholism in two historical snapshots: the early decades of the 20th century and in the 1990s. Two things come to light. First is the persistence of an "entrepreneurial spirit," and second is a remarkable failure to take into account the many-leveled heterogeneity in causes of alcoholism. The author describes some sources of this heterogeneity and shows how epistemological problems that arise from it suggest the failure of genetic accounts to consider the social dimensions of disease classification, observation, and diagnosis that form the very basis of the designation "alcoholic."

Historical Preface

The historical roots of the notion that alcoholism is caused by some aspect of heredity are deep. Commonly referred to as *the drink question* or *inebriety* throughout much of the 19th century, researchers began using the term *alcoholism* after its coining in 1849 by Magnus Huss. Despite these terminological shifts, the intuition that there is something constitutionally unique about people who drink frequently and/or excessively has been circulating in Western thought since the writings of the ancient Greeks. But what was previously speculation enjoyed a confirmational boost in 1900 with the rediscovery of Gregor Mendel's statistical characterization of hereditary transmission of traits. With this conceptual tool for discerning meaningful patterns in the appearance of traits over many generations (Mendelian analysis), many research programs began analyzing the hereditary roots of a broad range of society's problems—alcoholism, criminality, feeble-mindedness, poverty, and prostitution, to name a few (see, e.g., Pearson, 1905; Punnett, 1905; Saleeby 1909; Whetham & Whetham, 1909).

Popular and scientific writings touting the hereditary origins of alcoholism in the first decades of the 20th century deployed a distinct set of arguments often aimed toward specific social policy agendas. Among the more conspicuous were those advanced by the temperance and prohibition movements. More subtle than these was the conceptual shift whereby addictive behaviors were increasingly seen as subjects for medical attention rather than as symptoms of moral weakness. As part of the trend of medicalization, alcoholism developed into a specific physiological disease with possible hereditary origins.[1]

The "gene myth" as a framework of explanation of human behavior holds that any trait, disorder, or disease is caused by a gene (Hubbard & Wald, 1997). This framework took hold in the 20th century through many interesting turns.

A recent search of publications during this period revealed only two English language book-length studies devoted to the question of the relationship between heredity and alcoholism. Just prior to the rediscovery of Mendel's work, G. Archdall Reid, a prominent medical researcher, published a book-length study of alcoholism as a hereditary phenomenon titled *Alcoholism: A Study in Heredity* (1901). In it, he used the purported hereditary origins of alcoholism to bolster an argument against the temperance movement's prohibitionist contingent. According to his reasoning, if alcoholism is the result of inborn metabolic or digestive errors, then limiting the sale of alcohol will do nothing, in principle, to remedy the source of the problem. Rather, such restrictions will deny choice and freedom to the much larger segment of the population that lacks

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these errors—those who can drink without fear of disease. Reid's book significantly enhanced his reputation as an authority on the medical investigation of human heredity, laying the groundwork for his widely used 1905 textbook for medical students on heredity and disease, the subsequent reprints of which (in 1906 and 1910) brought Reid a tidy profit (evidence of the entrepreneurial spirit, about which I say more below).

Only a few years before Reid's book on alcoholism, Leslie E. Keeley also published a book-length study on alcoholism titled *The Non-Heredity of Inebriety* (1896/1902). In it, the doctor argued against the hereditary origins of inebriety, claiming instead that the craving for drink is due to the conditioning effect of alcohol on the body over time, invoking a mechanism akin to a mild response to a toxin. According to Keeley,

> The disease of inebriety is a lesion of the tissue cells and nuclei caused by poison. This lesion is a variation of the molecular type of the cell; it is a re-adjustment or re-arrangement of the molecules of the cells, designed to give to tissues a resistance to the poison. This is an inevitable sequence of all poisoning which does not cause immediate death. (pp. 344-345)

Such a poison response could only bolster one's immunity to the poison over the course of many generations. Furthermore, because the craving for alcohol is a conditioned response to the poison, only those exposed to alcohol can develop the disorder. No one is born an alcoholic.

Keeley's interests in the question of heredity and alcoholism were not entirely academic, so to speak. As the founder of a clinic for the treatment and rehabilitation of inebriates (using a method built around the toxin response theory), he stood to benefit considerably from positive reception of the theory that alcoholism had no basis in strict hereditary mechanisms. The more people he could convince of this, the more paying patients there would be for his clinic.

Thus, in the years immediately prior to the rediscovery of Mendel's work, debate over the role of heredity in alcoholism had already begun. An interesting and often-overlooked aspect of this debate is that the parties involved often stood to gain considerably from the success of their arguments, for or against a hereditary account of alcoholism. For the remainder of this article, I refer to such financial interests as the *entrepreneurial spirit*.

Skipping forward to our second historical snapshot, from the last decade of the 20th century to the present, it is interesting to note that although the debate is no longer polarized between hereditary and nonhereditary positions, many of the parties actively engaged in research have proprietary interests quite similar to their turn-of-the-century counterparts.[2] At this time, a common strategy for profiting from human genetic research involves patenting the methods for detecting whether an individual carries a particular gene or gene product.

Patenting the "Gene for Alcoholism"

In this section, I will review some arguments for the genetic basis of alcoholism that appear in patents granted in the United States since about 1988. I have two reasons for focusing on this literature. First, these documents are written in the same entrepreneurial spirit as that which infected the efforts of writers around the turn of the century. It would be quite unusual to endure the lengthy patent application process if one did not intend to gain financially from owning the patent rights to some technique, process, or product. These sources therefore offer a chance to examine the extent to which this entrepreneurial spirit affects the characterization of alcoholism in specific directions. Second, these patents, and the studies on which they are based, are the subject of considerable attention in the popular scientific press (e.g., *Time*, *American Scientist*, *Science News*, *Scientific American*, *Science*, and *Nature*).[3] Such wide exposure clearly serves to bolster the acceptance and legitimacy of these theories in the public consumption of science.[4] Therefore, by looking more closely at these "discoveries," it is possible to further the understanding of the proliferation of the gene myth.

A survey of the Lexis-Nexis™ patent database reveals a handful of patents granted since 1988 that deploy a hereditary account of alcoholism. These can be grouped into rough categories according to the mechanisms they specify by which genes influence drinking behavior. Some patents claim methods for testing for the presence of genes that code for specific neurotransmitters and receptors, whereas others claim genes associated with specific enzymes and metabolic products that play some role in the digestion of ethyl alcohol. Still others isolate genes that have a role in producing enzymes that break down toxic by-products of normal function in the brain on the supposition that

Table 1. Patenting the "Gene for Alcoholism"

Nutrient-metabolism genes (methods of testing for or delivering nutrients or metabolic products that are both "genetically determined" and "associated with alcoholism")

Tabakoff, U.S. Patent No. 4,770,996 (1988)—Identification of predisposition toward alcohol abuse. This patent is based on a test that compares the activity levels of two metabolic products found in the blood. These products are genetic products in the sense that all enzymes are genetic products, although the method of production and controlling genes are not specified. The two products are "platelet monoamine oxidase" and "adenylate cyclase." Two separate studies have shown that alcoholics have (a) greater activity levels of platelet monoamine oxidase and (b) lesser activity levels of adenylate cyclase than nonalcoholic controls. The patent is for the assays in which one can test for a ratio of these two activity levels.

Bradley, U.S. Patent No. 5,081,011 (1992)—Method and test kit for lymphocyte T-cell suppressor cells. Claims that a low level of a particular form of T-cell suppressor cell (CD8, to be exact) indicates "inherited substance abuse trait." Based on two previous studies that found correlations between other lymphocyte products and mental illnesses.

Dobbins, U.S. Patent No. 4,918,101 (1990)—Prevention and treatment of alcoholism with chromium. The claims here are (a) that alcoholism is consistently accompanied by hypoglycemia and diabetes; (b) that chromium is an essential and rare element for the regulation of insulin levels; and (c) just as control of insulin is a "magic bullet" for treatment and control of diabetes, dietary chromium (chromium picolinate) may be the crucial link in preventing and treating the body's metabolic debilitation in the face of long-term alcohol exposure. An additional patent was taken out in 1991 that added a "nutrient bar" as the delivery device for dietary chromium.

Neuro-receptor genes (methods of testing for the presence of a "gene for" alcoholism)

Blum, Noble, et al., U.S. Patent No. 5,210,016 (1993)—D2 dopamine receptor alleles. Claims that people inherit certain genes that build differing versions of dopamine receptors in the brain. D2 is a specific dopamine receptor that has been found to exist at greater frequency in persons diagnosed with "compulsive disorders." Alcoholism is a compulsive disorder. Patent is on methods for detecting the presence of the "genes" for the D2 receptor.

Neuro-function genes (methods of testing for the presence of a "gene for" alcoholism)

Manowitz, Poretz, et al., U.S. Patent No. 5,736,325 (1998)—Arylsulfatase-A alleles. Arylsulfatase-A (ASA) is an enzyme involved in first steps of biochemical breakdown of sulfatides in many human tissues (especially the brain). Sulfatides can disrupt the function of some neurotransmitters. Many different molecular-structure variants of ASA have been found, each of which differs slightly in biochemical function. Some of these variants have been found at greater frequency in people with "metachromatic leukodystrophy"—a disorder that causes slight retardation and general mental and behavioral messiness. Some ASA variants, 12 to be exact, were found in a sample of 56 hospitalized alcoholics (Hulyalkar, Nora, et al., 1984). This patent claims rights to several methods for the isolation, amplification, and detection of ASA variant "genes" using various tissue samples as well as for any kit for such detection based on any of these methods.

Note: Left out of this group of patents is a patent taken out by researchers at the University of North Carolina at Chapel Hill (Swift, Kupper, et al., U.S. Patent No. 5,464,742, 1995) for the design of studies aimed at statistical methods for investigating the association between any given set of genes and any particular disease. This is essentially a description of empirical and statistical methods for constructing *samples*, then comparing them using some statistical tools to determine the association between the gene alleles and the disease. This differs from Mendel's work because the methods attempt to account for diseases that are *polygenic*, that is, that have their sources in very many genes. It has been left out of the table because it does not propose any specific *mechanism* for an association of genes and alcoholism, proposing instead only a means for *description* of such association.

a lack of these enzymes can lead to toxic buildup and brain dysfunction, eventually causing behavioral disorders such as alcoholism. Focusing on the genetic pathway that leads to alcoholism, these patent claims can be referred to as implicating (a) nutrient-metabolism genes, (b) neuro-receptor genes, or (c) neural function genes.

In Table 1, the claims of each of these patents have been arranged according to these groupings, with detailed descriptions of the claimed causal pathway between gene and alcoholism in each case.

Notice that each group of patents in Table 1 implicates a markedly different causal pathway for the etiology of alcoholism. Alcoholism is purportedly linked with, and perhaps caused by, a wide range of entities such as immune system byproducts (Bradley, U.S. Patent No. 5,081,011, 1992; Tabakoff, U.S. Patent No. 4,770,996, 1988); neurotransmitters (Blum, Noble, et al., U.S. Patent No. 5,210,016, 1993), enzyme deficiencies (Manowitz, Poretz, et al., U.S. Patent No. 5,736,325, 1998), and even blood sugar levels (Dobbins, U.S. Patent No. 4,918,101, 1990). Such heterogeneity in the mechanisms thought to underlie alcoholism is representative of studies on alcoholism and heredity in recent years.

The glaring problem one notices when reading these patent documents is the vagueness of the causal connection between the genetic markers and alcoholic behavior. But the vagueness points to a deeper epistemological problem: The direction of the causal

arrow is in each case left open to question. That is, the studies on which the patents are based do not perform longitudinal analysis to determine which comes first: the physiological marker or the alcoholic behavior. For instance, if one looks at the patent by Manowitz, Poretz, et al., U.S. Patent No. 5,736,325, (1998), the studies finding correlations between ASA enzyme variants and alcoholism used samples of *hospitalized* alcoholics. Because severe alcoholic exposure over time undoubtedly alters the structure and function of human physiology, finding a higher level of any particular enzyme in a sample of people who already are alcoholics fails to addresses the question of whether the alcohol contributed to the measured effect or the measured difference contributed to the alcohol use.

On this "chicken and egg" point, there is much philosophical and technical work being done at present (Beurton, Falk, et al., 2000; Jablonka & Lamb, 1995). However, lacking a theoretical unification of developmental biological systems that describes a particular role for genes in interaction with environmental complexities, the issue is not likely to be resolved for some time.

From a different perspective, it is striking to note that one element these patents share is the attempt to place the source of alcoholism somewhere within the human constitution (in most cases the genome), further entrenching the medicalized view of the problem as an individual affliction instead of a social problem (Conrad & Schneider, 1980). The link between such characterization and the entrepreneurial spirit that underlies patent-seeking is worth further investigation. Only a large-scale comparison of profit-based versus nonprofit research programs could point to an answer (a task I will not attempt here). However, given the funding situation in which human behavior genetics exists, it seems unlikely that a nonprofit body of work can be found.

Etiological Heterogeneity

Putting aside epistemological suspicions arising from association of the entrepreneurial spirit and genetic-determinist accounts of alcoholism, there are still further epistemological difficulties stemming from the etiological heterogeneity of alcoholism. A few researchers have attempted to compile comprehensive reviews of different descriptions of alcoholism. Most prominent among them is E. M. Jellinek's (1960) appraisal of theories of the origins of alcoholism in the first half of the 20th century. In this meta-analysis, he displayed no less than 111 distinct theories grouped into four broad subsets (see Table 2).

Table 2. Etiological Speculation on Alcoholism, 1938-1959

Etiological Category	Number of Distinct Theories
As a psychological illness	33
As a symptom of psychological illness	13
As physiopathology	
Allergy	6
Biochemical-physiological disease	13
Brain pathology	9
Nutrition	7
Endocrinology	6
As indicative of pharmacological response	24
Total	111

Source: Adapted from Jellinek (1960).

Such a large number of different theories on the underlying causes of alcoholism serves to highlight the heterogeneity of alcoholism at (at least) two levels. First, it is possible that these studies of alcoholism employ different definitions of alcoholism and thereby proliferate and confuse a number of different diagnostic criteria, lumping them all under one heading. This problem was rampant at the time of Jellinek's (1960) work but is arguably less of a problem today, as researchers have converged on the diagnostic criteria provided in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*). Although these criteria are problematic on their own terms,[5] they perform a positive function. They offer some way of trimming down the heterogeneity of phenomena on which researchers can base their claims to have found common physiological (or other) states in the alcoholics plucked out by the criteria. Second, the large number of theories of alcoholism points to the deep-seated need for conceptual clarification.[6] For even if research were to show (statistically?) meaningful clusters of phenomena of drinking behavior and physiology, progress on the question of hereditary causes can only take place if there is feedback between research results and diagnostic criteria. However, even assuming we have stable definitions of "alcoholisms" and unique diagnostic criteria for each, drawing the link back to any genetic underpinnings will be complicated by heterogeneity at a far more basic level.

The Norm of Reaction

Now, putting aside for the moment the diversity and heterogeneity of causal pathways presumed responsible for all these alcoholisms and focusing simply on one causal pathway—the action of genes—there is another, somewhat more technical difficulty. It was noted in about 1909 in a study of purebred, self-fertilizing lines of crustaceans that different genotypes would develop different phenotypes (in this case, shape of head) in reaction to any one environmental variable (e.g., ambient temperature, nutrient levels, etc.).

The reactions were neither uniform nor linear—that is to say, in the presence of the same environmental situation, each genotype would generate a different growth pattern over time in a very consistent manner. When represented graphically, these growth patterns appear as curves (see Figure 1). Originally termed *Reaktionsnorm* by Richard Woltereck (1909), these complex, nonuniform relationships between genotype-phenotype-environment are now known as *norms of reaction* (Sarkar 1999).

The epistemological difficulty posed by such phenomena, especially in the description of such complex systems of heredity and development as underlie human behavior, is that for any one phenotypic outcome (alcoholism), there can be many possible underlying norms of reaction, each generated by a different genotype in response to divergent environmental exposures.[7] So the attempt to trace the causal chain backwards (so to speak) from a phenotype to an underlying genotype without strict and precise control of the temporal sequence of environmental exposures is folly. This intermixing of levels of feedback systems lies at the foundation of the philosophy of biology.[8] Absent a complete solution to this vexing intermingling of causes in developmental systems, claims to have found "the gene for" a human behavior must remain deeply suspect.

What Is a "Gene"?

Now it is possible to look at an even finer level of phenomena implicated in claims to have found "genes for" traits. In this case, assume that answers have been found to the problems of level interrelation noted above. Focusing on the molecular level alone, how can an answer be constructed for the question, "What is the gene for *x*?"

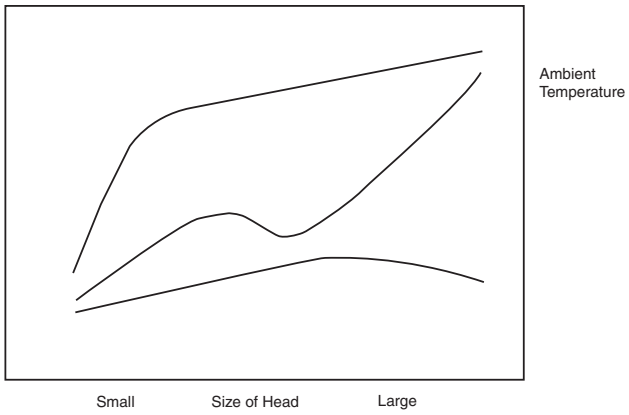

Figure 1. The Norm of Reaction
Note: The *x*-axis represents measurements of a phenotypic variable (head size), and the *y*-axis represents an environmental variable (temperature). Each line corresponds to the behavior of a unique genotype. The curving lines illustrate the nonlinearity of genotype-phenotype-environment interactions in complex organisms.

Take as an example a single enzyme and the gene(s) that produces it. At the outset, it is important to keep in mind that there is much more involved in the production of the enzyme than simply a sequence of base pairs[9] on a molecule of DNA. The relevant strand of DNA must be *readable,* that is, have an *open reading frame (ORF)* where the surface of the molecule is accessible by other cellular machinery. This cellular machinery consists partly of messenger and Trenter RNAs that perform translations of the base pair sequence into a sequence of amino acids. In addition, the sequence of amino acids so produced will collect together and "fold" to form a polypeptide, a molecule that can eventually serve a biological function (such as an enzyme). In the folding process, it often discards some of its parts while also picking up molecules produced by similar methods elsewhere on the DNA sequence. Finally, the molecule must be made available for interacting with other molecules, functioning essentially as an enzyme (Beurton, Falk, et al., 2000; Jablonka & Lamb, 1995).[10] And this is only an abstract of the process! In claiming to have discovered "the gene for enzyme *x*," the discoverers must articulate what the relevant cellular machinery are in addition to DNA the sequence(s) that produces the enzyme. Furthermore, because there is considerable variance in which set of cellular machinery is relevant to any set of products, there is a complex heterogeneity of causes

within the level of genes themselves, entirely aside from the causal heterogeneity of the complex biological systems in which the genes function.

The Looping Effect of "Human Kinds"

Now I will move from epistemological problems that arise from the molecular levels of phenomena back out to similar problems arising at the macrosociological level. Here, I describe another vexing feedback system that I maintain must be taken into account if a full understanding of the role played by heredity in complex human behaviors such as alcoholism is ever to be gained. Because the genetic accounts discussed above make no mention of interrelationships at this societal level, the goal of a full account of alcoholism and heredity is clearly far from realization.

Ian Hacking (1995) recently called attention to an epistemological difficulty that affects only humans, a problem he called the "looping effect of human kinds." He introduced *human kinds* as a particular type of identifiable and meaningful classification. They differ from their philosophical counterpart, natural kinds, only in that they are subject to this looping effect. He defined *human kinds* according to the following four criteria:

> (i) kinds that are relevant to some of us
> (ii) kinds that primarily sort people, their actions, and behavior, and
> (iii) kinds that are studied in the human and social sciences, i.e., kinds about which we hope to have knowledge, and also
> (iv) that kinds of people are paramount; [they] include kinds of human behavior, action, tendency, etc. only when they are projected to form the idea of a kind of person. (p. 354)

In short, human kinds are historically contingent yet socially real. They constitute the classes into which we place other human beings and so have real consequences. But taken more broadly and historically, they are fundamentally arbitrary: They shift over time, differ according to where they are applied on the planet, and seldom map onto underlying features of the world that philosophers would call *natural kinds*.[11]

Most interesting for our purposes is that human kinds, such as the kind-designation *alcoholic*, affect the entities to which the *kind* label is applied. Trees do not alter their behavior if we call them by a different name, whereas people often alter their behavior when they have been labeled as alcoholic, learning disabled, depressed, and so on. But Hacking's (1995) insight goes beyond mere labeling theory in arguing that the kinds act on aggregates at more than an individual level, generating effects at institutional levels and thereby complicating our scientific schemes of classification and the empirical studies reliant on them. That alcoholism exists as a category to which one can be assigned (even if only in the future) can significantly influence not only the behavior of individuals with respect to alcohol intake but also the anticipatory actions of social and scientific institutions. The legitimacy of the category as a social and personal endpoint ebbs and flows over time and across geographic location.[12] But most important, the kind classification can underwrite its own expansion, legitimacy, and objectivity. This is the looping effect.

As a kind subject to such looping effects, alcoholism can inform, revise, and bolster its meaningfulness for human beings of the future. This feature of alcoholism is crucial for any study of heredity and drinking behavior, for it constitutes a phenomenon that can corrupt or influence any patterns observed among populations of humans so designated—even genetic studies. Although it is perhaps difficult to see at first, I maintain that awareness of such dynamics at all levels of phenomena is crucial to a proper understanding of the history and current status of alcoholism, especially with respect to its purported origins in heredity.

Concluding Remarks

It may seem that I have outlined an entirely negative project, criticizing accepted ideas and the links between them while avoiding positive claims. This is not my intent. Of course, if my criticism has done anything to unseat the gene myth in relation to alcoholism and, by implication, other complex human behaviors, I will be pleased. But I have in mind a positive project also. From a philosophical perspective, the current historical overview and reflections may supply a useful insight: Despite technical progress in answering the questions posed by society's problems over the past century, the preference for posing only certain questions needs an explanation. Why, after all, are people increasingly fascinated with medicalized models of society's problems? Philosophical reflection on the tendency among Western intellects to privilege for so long a time certain questions (and answers) is needed. My intuitions at present lead me to believe that there is

something deeper in the history of the enlightenment scientific project that privileges simplicity as both an epistemic and ontological virtue.[13] Good theories are elegant, reductive, and account for a wide range of phenomena with the fewest or simplest theoretical entities. Thus, the myth of simplicity in our explanations could have steered the course of research in the human sciences to the position it now occupies. The above reflections on associations between alcoholism and heredity indicate that an examination of this subterranean current is overdue.

Notes

1. As Dobbins, U.S. Patent No. 4,918,101, (1990) put it, the physiological description of the disorder eclipses the moral aspects: "Craving for alcohol is not due to moral weakness. It is a sickness—the continued response of the body to a pathological lack in the diet of the essential trace element chromium" (p. 2).

2. Because a full history of the development and refinement of research on genetic causes of alcoholism is a task beyond the scope of this article, I restrict my focus for heuristic purposes to limited historical snapshots of the field. However, I maintain that the similarities between early- and late-20th-century appraisals of alcoholism differ very little, despite whatever technical progress can be claimed between the two periods. This is not to say that the theoretical understanding of the disease was static during the interim, for, of course, there were many subtle and interesting debates throughout. Determining why the present looks so eerily like the past is part of my motivation for writing this article.

3. For a detailed examination of discovery rhetoric regarding the "gene for alcoholism" in popular press sources, see Conrad and Weinberg (1996).

4. I use the term *consumption* because it captures the exchange between mass media and lay public better than the commonly used alternatives (e.g., *public understanding of science*, *public reception of science*, etc.). To the extent that the public can be construed as consumers of entertainment and mass media, consumption of news about discoveries and progress in science is the better characterization.

5. The *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*) criteria have been criticized widely for their conflation of social dysfunction with a disease state (see Hess, 1995, on this point, especially chap. 7). This points to a deep underlying problem: There is no purely physiological or medical description of alcoholism beyond physiological dependence on or severe toxic effects from alcohol exposure. Absent such a model, it is all researchers can do to find people who have been diagnosed for social reasons and then work backward by trying to find common physiological states among those so labeled.

6. See Roebuck and Kessler (1972) for a compelling overview of the constitutional, psychological, and sociological accounts of alcohol problems and the tensions between them.

7. See Hogben (1933) for a poignant illustration of these epistemological problems.

8. See Grene (1967) for the first detailed appraisal of the problem. More recent articulation of the nature of the problem can be found in Lewontin (2000).

9. The bases that enter into these pairs are adenine, thymine, guanine, and cytosine.

10. See also Burian (1997) for aspects of the problems determining the relationship of genes to the development of organisms.

11. Hess (1995) briefly discussed the historical shifts and intercultural variation in some disease designations, noting that disease classifications at the level of psychological disorders (embodied in the *DSM*, for instance) are peculiarly dynamic.

12. Alcoholism is diagnosed less in Finland than in the United States, perhaps because drunkenness is an acceptable role for people to play.

13. Some initial steps in philosophical examination of the notion of simplicity in science can be found in Rescher (1990).

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