

**HEREDITY AND THE HUMAN CONDITION:
A STUDY OF 20TH-CENTURY GENETIC ACCOUNTS OF ALCOHOLISM**

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Abstract

This dissertation takes as its starting point some curious historical parallels in research on the heritability of alcoholism from opposite ends of the 20th century, and the underlying continuity in assumptions implicated by these parallels. Rather than review mainstream historical narratives on the origins of genetic research and alcoholism studies, I examine evidence and developments as yet unexplored by scholars. First I examine the origins of recent research models and diagnostic criteria that provide evidence for the hereditary nature of alcoholism. Then I consider the assumption of genetic determinism and its relationship to strategies of propaganda employed by the Eugenics movement early in the century. Using these historical “snapshots” I draw out conceptual and philosophical problems with the genetic explanation of alcoholism that continue to confront researchers today. These limitations suggest two possible avenues of resolution: either we develop finer-grained strategies for distinguishing social deviance from physical disorders, or we develop an integrated understanding of the complex interplay of human biological and cultural systems by extending the approach known as Developmental Systems Theory. In the conclusion, I explore these options and their potential ramifications for our understanding of alcoholism in hereditary and human contexts.

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Introduction

Most of us know someone, perhaps in our family or circle of friends, who has seen some trouble stemming from alcohol. We may know people who have been arrested for DUI, or have had too much to drink and become the fool of the holiday party, or have chosen to drink at inappropriate times or places (e.g., lunches, classrooms, or cubicles). A smaller number of us know of someone whose trouble with alcohol has been extended, involving a number of incidents over significant stretches of time. These facets of the human experience of alcohol and attendant problems drive our concern over the issue of alcohol use, both personally and to some extent scientifically. Combined with this awareness of drinking problems, most of us are aware of our debt heredity, living as we do in a world rich with reminders and warnings about the traits, talents, and diseases purportedly caused by the genes we received from our parents. Such reminders come to us from a wide array of sources—from the finest literature to the tabloid reader, from the cinema to the television, and from doctors, scientists, pundits, and journalists worldwide. In this era we are told that the entire sequence of the human genome will be articulated and made available for scientific analysis and predictions. We are told that this breakthrough will usher in a golden age in medicine and health, as we develop gene-therapies and targetted pharmaceuticals for all of our current ailments, possibly even extending our lives. In light of this collection of information—alcohol problems, our own experience of heredity, and constant references to genetics in the media—one expectation might be that we will one day see the discovery of genes that cause alcoholism. What remains unknown to most of the American public is that

scientists have claimed to have made exactly this “discovery” on a number of occasions in the previous two decades, and the search for genetic causes of alcoholism is as old as the very discipline of genetics. In what follows, I trace some key attempts to attribute alcohol problems to hereditary factors. In so doing I lay the foundation for a critique of the enduring presumption that alcohol problems can be explained by genetic analysis.

The following dissertation is the result of a thread of interest in the application of genetics to human diseases and social problems that began during Richard Burian’s graduate seminar on the formation of genetics as a discipline. Exploring the parallel development of the eugenics movement and genetics at the beginning of the 20th century I grew sensitive to the tone of current genetic-determinist rhetoric in the media. Popular press presentations of the “gay gene” and “genes for alcoholism” led me to wonder what foundational assumptions about heredity and human nature might be propping up these stories. During a review of some textbooks on genetics from the early 20th century, I noticed the frequency with which alcoholism (or “intemperance,” or “inebriety”) were included in the lists of problems for which genetic science may one day provide remedies. In a subsequent search for contemporaneous studies of alcoholism it became clear that one of the textbook authors also happened to be a writer of some significance on the subject of alcoholism—Sir George Archdall Reid. This presented a clear picture of the complicity of eugenic aspirations in early 20th-century characterizations of the hereditary nature of alcohol problems, through the life and work of a single key figure. Since there are currently no published biographies on Reid, I go to some lengths to present his story in what follows.

This dissertation examines the following questions: In both popular and intellectual contexts, why has so much attention been paid to the notion that alcoholism is genetically determined? What are the problems attendant to this notion? Are there alternative ways of

accounting for the phenomena that have been overlooked? If so, what efforts have been made to pursue these alternatives? The aim of the dissertation remains philosophical: We can learn much about the complexities of the human condition and the limits of genetic determinism by examining in detail the singular case of human alcohol related problems.

We currently accept many ideas without much critical reflection. Many of these are aspects of the human experience that have been subjected to scientific scrutiny at one time or another. Examples are: the world is spherical in shape, the sun is the center of the solar system, there are hereditary components to intelligence, addiction is a progressive disease, and so on. What's interesting from a philosophical and historical standpoint about such ideas is that the origins have been closely scrutinized and critically examined for only few of these notions. One can easily locate hundreds of book-length treatments of the transition from the geocentric to the heliocentric model of the heavens, but little attention has been given to ideas that deeply influence "models" much closer to our daily existence—that is, pieces of our understanding of what it means to be a human being living and communicating with other human beings. To put a finer point on it, the phenomenology of human experience and self-knowledge is underrepresented in the extant body of knowledge generated by modern molecular, clinical and genetic science.

This gap in scholarship and intellectual thought is a vexing problem, since many of the challenges that lie ahead of us are no longer technical, but largely social and ethical in nature. We only need to look to the quandaries in medical ethics, where, for instance, the technological ability to keep an unconscious human body alive through intravenous feeding has been extended to years instead of days, but there has been no concomitant extension of our ethical or cultural consciousness in terms of the status such human bodies are to have during this period of extension. Are these unconscious human bodies still persons,

possessing the rights usually accorded you or I? While there is active debate on such issues, consensus and closure do not come readily or easily. What this and other cases illustrate is that even while we may be gaining steady ground with respect to some technical aspects of the human condition, there is still much work to be done in order to reconcile this progress with our cultural understanding of the human condition.¹ For me, this means that we may run the risk of applying our great technical prowess to “problems” that stem from inadequate concepts and models built up by reference to accumulated folk-knowledge. The danger posed by this weakness is that our attempts to intervene will have little purchase in our world.

One small step toward a reconciliation requires that we develop an understanding of the inherent limits of certain types of explanations of the human condition. For starters it is important to look carefully at the encroaching explanations of human behavioral and social problems from evolutionary psychology, darwinian medicine, and behavior genetics. As I will show, these explanations rely on bewilderingly complex accounts of fine-grained hereditary and evolutionary phenomena, but the very disorders they seek to explain are ramshackle constructions whose definitions and boundaries lack resolution. Worse still, there is a burden of providing a mechanism by which genetic material produces larger effects that these evolutionary stories fail to meet.

These areas of scholarship operate on the assumption that evolutionary theory offers viable explanations of virtually all facets of modern human problems—from fevers and infections to eating disorders and even emotional problems. I follow Hubbard and Wald in

¹ Putting this in the language of Wilfrid Sellars, we are still a far cry from a reconciliation of the Manifest and Scientific images of man. See “Philosophy and the Scientific Image of Man” in *Science, Perception, and Reality*, (London: Routledge and Kegan Paul), 1963.

calling this broad assumption the “gene-myth” (Hubbard and Wald 1997).² One commonly cited definition presents the idea like this:

Geneticization refers to an ongoing process by which differences between individuals are reduced to their DNA codes, with most disorders, behaviors and physiological variations defined, at least in part, as genetic in origin. It refers as well to the process by which interventions employing genetic technologies are adopted to manage problems of health. Through this process, human biology is incorrectly equated with human genetics, implying that the latter acts alone to make us each the organism he or she is (Abby Lippman, quoted in Hubbard and Wald 1997, p. 2).

All of this is part of a larger trend of medicalization--where problems formerly considered to be moral failings, or even crimes, are recast as having their origin not in the will of human beings, but rather in damaged biological tissues or processes. In some cases, the proposed biological mishap resides in the genes inherited at birth, a defect in the program that the individual will follow for the rest of their life and which may also be passed on to their offspring. These extreme cases of medicalization where the center of power is attributed to DNA are called “geneticization” or “genocentrism.” In what follows I will use “the gene

² One tempting alternative term is “genomania” (Hubbard and Wald 1997, p. 164). Throughout this dissertation I evaluate material that exhibits the prevalence and depth of genocentrism in thinking about alcohol problems. In so doing I use the terms “gene-myth,” “geneticization” and “genocentrism” interchangeably to refer to the prevalence and operation of genetic reductions of human traits. As an aside, Richard Lewontin offers an interesting hypothesis on the complicity of techno-science in this genocentric framing of human problems: “The invention of automatic DNA-sequencing machines was a response to a growing demand for sequence, but the availability of such machines and the great ease with which DNA can now be sequenced has meant that the problems on which geneticists work have become those that can be answered from DNA sequences” (Lewontin 2000, p. 128).

myth” as the phrase to capture the entrenched assumption that genes are the seat of power for determining *all* traits of the organism from conception to adulthood. These traits are not limited to physical attributes, like height and bone structure, but include more subtle characteristics such as intelligence, emotions, psychological states, and moral failings.

One way of countering this trend of “geneticizing” social problems is to present alternative accounts from—models, critiques, and explanations that may have merit but have not received much attention. A close review of the literature and theoretical work on alcohol problems can shed light on answers to basic questions such as:

- Where did our current attitudes on drinking behavior originate?
- How has the experience of disease and addiction figured into these attitudes?
- What elements of these norms have changed in direct response to social, technological, or medical innovations?
- What elements have remained the same despite change in other spheres of scholarship or innovation?
- To phrase the question in Kuhnian terms, how did the current dominant paradigm of genetic reductionism achieve hegemony, and how do the competing paradigms render both problem and solution?³

³ It would be an ambitious task indeed to document the provenance of the “paradigm” that fosters the scientific reduction of complex human situations to genetic determinants, and I will not attempt it. The genetic determinism that I attribute to the “gene-myth” arguably shares the form of the centuries-old mechanistic worldview. The clock-work model of the human body is consistent with the assumptions behind the gene-myth, that the genome determines all aspects of the organism, or to put it differently, that genotype determines phenotype. On this analysis, genes serve as the mainspring that completely drives the development of human biological and behavioral characteristics. This holdover from the mechanistic paradigm is what the following dissertation serves to critique.

Science and Technology Studies (STS) provides at least one strategy for developing answers to these questions. In the field of Science Studies many scholars focus on the social and personal ramifications of scientific and technological developments and the policies that affect them. Applying these concerns to the genetic focus in alcoholism research would generate dissertation-length studies highlighting both (a) the personal experiences of the many sufferers of alcohol problems and the place of hereditary knowledge within this “local knowledge” framework, and also (b) the role of social and political policy in shaping the cultural and scientific understanding of alcohol problems. While these are perfectly worthwhile uses of scholarly energy, they will not be addressed in this dissertation. My goals in this dissertation follow from my intuition that the gene-myth associated with alcohol problems is so deeply entrenched in many segments of our culture that to focus on individual experiences of “alcoholics,” or the course of social policy aimed at treating them, will do little to unseat the gene-myth. Those that study alcohol problems generate their own media focus with each new “discovery” of a genetic marker for alcohol-related behavior or physiology. It therefore follows that in order to challenge the gene-myth our critiques must employ the same language as those who adhere to it, and alter the emphasis on genetic factors from within the bio-medical framework. Challenges that originate in personal narratives of alcohol-affected persons probably will not be taken as relevant and will not alter the status of the gene-myth. Consequently, in what follows I focus on some aspects of the “internal history” of alcohol studies and hereditary science, thereby providing a critique of the hegemony of bio-medical and genetic explanatory reductions of alcohol problems from within the bio-medical framework. Without a critical examination of the currents that power this intellectual drift toward a genetic-foundational view of disease, we risk endorsing the current hegemony of the gene myth, even though there may be many non-genetic factors

that provide greater purchase on disease—factors that may provide more useful hand-holds for treatment or management.

As my research on the application of genetic principles to human social problems progressed, I was struck once again by the lack of progress that has taken place regarding the study of hereditary patterns of alcohol-related problems. When comparing the state of alcohol studies in early 20th century with that at present, another curious continuity emerges. The assumption that the study of heredity and genetics will lead to the alleviation of problems associated with heavy alcohol use seemed to have remained intact, while other items on the eugenicists' list such as “feeble-mindedness” and “consumption” have succumbed to the analytical engine of science and scholarship.

In this respect the attempt to explain alcohol problems by reference to genetic factors is unique among the list of problems originally targeted by eugenicists. Feeble-mindedness has been divided into several distinct disorders and conditions, and the very word tossed out as a result of the growing disciplines of psychiatry, psychology, and neuroscience. Many of its former referents are now considered distinct medical disorders attributed to identified physiological or developmental problems. This catch-all term is no longer in use, and for good reason. One aim of the following study is to shed some light on why exactly alcohol problems are characterized today in such similar fashion to that of the early 20th century. I show that at several points in the 20th century little has been done to challenge the assumption of hereditary influences on alcohol problems.

Readers will note that the focus of this dissertation is limited to the 20th century. While this is important for reasons of scope and space, it does present a necessarily incomplete picture of the sources from which our modern assumptions about alcohol and heredity have arisen. The fundamental reconfigurations of natural philosophy, science and

that took place during the 18th and 19th centuries set the stage for the casting of alcohol problems in a medical and genetic framework during the 20th.

It would be folly to attempt a review of the social and conceptual landscape that makes up the temperance movement in the 19th century. To be sure, there is a considerable literature that addresses exactly these concerns. However, it must be recognized that the framing of alcohol-related problems and the context in which theories about their origins in heredity formed were established by trends that began in the 19th century. The framework in which alcohol problems and heredity were considered in the 20th century is the result of the possibilities outlined during the 19th century in political, moral and scientific movements that touched on heredity and/or temperance issues. Even the term “alcoholism” was introduced during the 19th century in a book on the subject by Magnus Huss, a Swedish physician, titled *Alcoholismus Chronicus* (1849). Originally conjured to describe chronic alcohol use (that is, being a “drunkard” rather than being merely drunk), the term has been subsequently imbued with varying degrees of moral baggage. During the previous two centuries a transformation swept through characterizations of alcohol problems from being considered sins, crimes, diseases of the will, psychological abnormalities, and medical disorders (Conrad and Schneider 1980; Kunitz and Levy 1995; Valverde 1998). This is true for both the United States and Britain, with an alarming degree of congruence. Since the relevant factors behind each of these designations range from national political trends, models of deviance and conformity, and the public awareness and reception of biological theory, I submit that modern considerations of the problem can benefit considerably from historical perspective on what has been deemed relevant to the issue in the past.⁴ For

⁴ See Nye (1984, esp. Chapter 1) for a compelling account of the relevance of broad-scope history for modern medical, psychological, and criminal designations. The aim of Nye’s historical investigation of deviance designations is to show that “deviance is a process in

centuries the use and misuse of alcohol has taken place within cultural frameworks that include an awareness of human heredity. The ancient sources of this awareness are not trivial for enlightenment era visions or modern characterizations of heredity and drinking. For example, the utopian plan outlined in Plato's *Republic* is a classic thought experiment on the ideal structure of society, and it employs an understanding of heredity in its plan. One interesting feature of this ideal society often goes unremarked: the program for breeding the guardians of the society.⁵ A further example of these issues appearing in classic texts is

which societies confer a social meaning on certain acts they consider deviant. This process is far from being an arbitrary one; it reflects the values, the anxieties, and the cultural norms of any society. What is more, the context of values and anxieties out of which standards of normal and pathological are shaped changes over time, sometimes radically so" (p. 15). However, for medical and legal specialists living "in the moment" these designations appear to be well-grounded naturalistic categories. The brief review of some historical snapshots that follows is meant to counter this epistemological positivism regarding the designation "alcoholic." The perspective I flesh out in this dissertation holds that designations like "inebriate" and "alcoholic" can best be understood by examining the boundaries of societies values regarding normal and deviant behavior over time, rather than the development of scientific work mapping the hereditary manifestation of certain types of this deviance.

⁵ Couched within the social architecture for the governance and protection of the *Republic* is a scheme for the selection and maintenance of a militia of sorts, called the "guardians," whose sole task is the defense of the state against aggressors. It thus serves a crucial role in the long-term viability of the state, as otherwise the state may suffer disintegration at the hands of a foreign army, even while functioning in perfect political harmony internally. Most interesting for our purposes is the care with which Plato devises his scheme for the selection and pairing of mates among the guardians, in order to generate desirable offspring. Plato writes "It follows from our former admissions, I said, that the best men must cohabit with the best women in as many cases as possible and the worst with the worst in the fewest, and that the offspring of the one must be reared and that of the other not, if the flock is to be as perfect as possible. And the way in which all this is brought to pass must be unknown to any but the rulers, if, again, the herd of guardians is to be as free as possible from dissention" (Plato 1997, 459e). A deceptive lottery system of mate-pairings was also feature of the proposed breeding scheme, in which the rulers secretly and systematically arranged for the "best" pairings, while the guardians themselves were encouraged to believe the outcomes resulted from pure chance (see *Republic* 459e-460d).

It is clear that Plato's program for systematic human procreation shares a great deal with the goals of eugenics promoters in the 19th and early 20th century. The example of Plato's *Republic* did not escape the founders of the eugenics movement in the first decades of the twentieth century. William E. Castle (1916), for example, speaks highly of the breeding program of the *Republic*, going so far as to attribute the decline of Ancient Greece and the

Aristotle's writings. In the *Nicomachean Ethics*, Aristotle frequently uses examples of drinking to excess.⁶ The fact that Aristotle employs cases of drunkenness specifically as examples worthy of moral consideration underscores the point that, even for the ancients, humanity's relationship with alcohol was never a simple equation. Rather, the moral, religious, and cultural significance of alcohol use prompted rich consideration and debate.⁷

Given the recent explosion of the number of studies seeking genetic determinants of human disease, it seems the time is ripe for an examination of the definition of diseases such as addiction. A crucial step in this examination is to articulate how genetic information relates to disease states. The question of what constitutes health, and conversely disease, has received attention for centuries, albeit without a great deal of continuity. Many of the same

Roman Empire to poor performance in breeding the better "stock" of society. This remnant from Plato's *Republic* provides only a glimpse of the historical roots for ideas concerning hereditary determination and manipulation of human traits.

⁶ It is interesting to note the way in which alcohol use appears. In book III, Aristotle invokes examples of drunkenness and drunken behavior to illustrate his arguments concerning obligation, responsibility, and self-control. First, in the context of a discussion regarding responsibility, he writes "Indeed, we punish a man for his very ignorance, if he is thought responsible for the ignorance, as when penalties are doubled in the case of drunkenness; for the moving principle is in the man himself, since he had the power of not getting drunk and his getting drunk was the cause of his ignorance." He continues, "So it is, too, with respect to weakness and infirmity; no one would reproach a man blind from birth or by disease or from a blow, but rather pity him, while every one would blame a man who was blind from drunkenness or some other form of self-indulgence. Of vices of the body, then, those in our own power are blamed, those not in our power are not. And if this be so, in the other cases also the vices that are blamed must be in our own power" (Book III, 5, 13/14).

Aristotle clearly saw cases of inebriation as exemplary of loss of control, nonetheless allowing retention of responsibility on the part of the individual. But these are merely details of what Aristotle has to say about responsibility—what is relevant for our purposes is the fact that at nine separate occasions throughout the text of the *Nicomachean Ethics* Aristotle employs drunkenness as an example.

⁷ One further data point reflecting the cultural heritage of alcohol-related behavior is the Bible. As reported in Sournia (1990), the following passages of scripture contain references to drinking of many sorts: Genesis 9:20-25; Genesis 19: 30-38; Jeremiah 35: 5-10; Proverbs

questions have been raised and answered differently by independent authors in various times and places. For example, consider the French notion of “degeneration” in the 18th century—an idea tied to nationalist politics, as well as a medicalized perspective of race and heredity in that culture. The notion of degeneration had a remarkable effect on the shape of hereditary science and its place in the criminal justice system in France, leaving a legacy that remains even at the time of this writing.⁸ A brief look at the alcohol problems promises to flesh out our understanding of the role of political, professional, sociological, and technological elements in the modern construction of complex disease states.

In this dissertation I argue that the current fascination with genetic explanations of alcoholism rests on shaky ground. Attempts to reduce alcoholism to genetic aberrations display two fundamental problems. First, the definition of alcohol is in need of conceptual clarification and refinement. As I show, even current diagnostic criteria for alcohol dependence can be met by a great number of distinct behaviors, attitudes and incidents—all of which are conflated and obscured by the application of the gene-myth. This definitional heterogeneity, coupled with the fact that human cultural uses of alcohol differ widely, renders social-scientific measurement and prediction of alcohol related outcomes deeply problematic. Second, there are epistemological problems that belie the popular presentation

31: 6-7; Proverbs 23: 29-35; Psalms 104: 15; Samuel 1: 12-15. An analysis of the moral and cultural status of these remarks would be an interesting exercise.

⁸ Burian, Gayon and Zallen (1988) articulate the development French genetics research programs in the unique context of degeneracy theory and rival approaches from embryological research. Nye (1984) develops a detailed account of the connection between ideas of national decline and hereditary degeneracy theory in 19th century France. The interplay of political and scientific currents in the formation of criminology and establishment of precedents in the justice system in France underscores the need for careful reflection on the role of hereditary science in the U.S. See also Carlson (1985) for a concise review of the origins of medical degeneration theory and its effect on psychology in the U.S. and Britain.

of oversimplified models of “genes for” alcoholism (termed by some as “alcogenes”). These problems stem from the sheer complexity of gene-to-product relationships that anyone in molecular genetics or developmental biology understand as basic features of biological systems. Without addressing complexity and non-linear, non-additive causal pathways *within* the organism, studies that seek genetic “markers” of alcoholism by analysis of covariation between genetic material and alcohol-related outcomes are destined to fall short of understanding the mechanisms (biological or other) that produce these outcomes. Finally, I will describe a new corner of scholarship called Developmental Systems Theory (DST), that offers promise as a framework for developing models of such complex biological and cultural interactions.

A caveat: There is one large area of study that I will not address in this dissertation. The amount of attention given to alcohol problems among Native Americans throughout the history of this nation is astounding. The assumption of genetic contributions to alcohol problems among this population prevails despite a vocal minority of opinion to the contrary. One result of this rift is a contest for providing the explanation of the human condition as experienced by Natives. In this respect these humans and their collective lives comprise a contested epistemic landscape. Among the more compelling social and cultural accounts that avoid genetic reductionist assumptions is a book length study titled *Drunken Comportment* (MacAndrew & Edgerton 1969). Using cross-cultural evidence gathered from accounts of explorers, trappers, missionaries, and others, the authors deploy evidence for a theory that pins alcohol problems among Native Americans to drinking behavior learned from their first contacts with westerners and their own use of alcohol. This hypothesis gains support from accounts of the drunken comportment of westerners who first exposed native Americans to drinking behaviors. They claim that most natives at the time of this introduction had either

no prior exposure to alcohol, or exposure to alcohol or other substances only in the context of tribal ritual and rites of passage. The timidity with which many of these unexposed Native Americans reportedly conducted themselves before learning to drink like westerners suggests there is something to a social learning model of alcohol-related behavior. If these behaviors are learned and developed into entrenched customs, then the problems that arise from these behaviors and their persistence through subsequent generations does not require an explanation on the basis of genetics. There is considerable potential for an empirical test of this hypothesis, although to my knowledge none have been conducted. The subject of genetic accounts of Native American drinking behaviors and problems is rich enough for a dissertation in its own right. In order to develop a perspective on the continuity of genetic accounts of alcoholism and lay the ground for their critique, I do not delve into the detailed literature concerning the Native American aspect of the issue.

In the next chapter I examine the heterogeneity that has existed historically (vestiges of which remain today), in the concept and meaning of “alcoholism.” I review the methods employed by researchers in their studies of heredity and alcoholism. Here we will see that despite what might appear to be a unified consensus on alcoholism, there is a diversity of opinion on both the proper description of the behaviors associated with the condition as well as how their causes can be isolated.

In the second chapter I focus on one social and political movement whose role in shaping the subsequent study of alcohol problems is under-appreciated: eugenics. The genetic determinism and racist reproductive policies projected by eugenicists in the United States and Britain early in the 20th century frequently portrayed alcohol problems as a principal example of the sort of social problem that selective human breeding could eradicate. The depth of the assumption that problems with alcohol were the result of

hereditary or genetic factors in this context is stunning. In order to articulate the connection between eugenics and the gene-myth of alcoholism, I begin the chapter with an examination of a figure whose work effected growth on both of these fronts. The involvement of Sir George Archdall Reid in the context of eugenics and his subsequent work on the hereditary aspect of alcoholism highlights the connection between eugenic thinking and the ensuing quest for genetic causes of alcoholism. After reviewing the extent of Reid's impact on the gene-myth for alcoholism, I turn to a review of the entrenchment of eugenic ideas and examples of alcoholism as a eugenic target in the context of college-level textbooks on the new science of genetics. I show that during the first two decades of the 20th century, when the discipline of genetics was first forming, there was a considerable amount of application of breeding principles and genetic analysis to human social problems by authors of these texts. This review serves to highlight the growth of an ideology within genetics according to which many of society's problems not only can be reduced to genetic factors, but may be controlled or treated using genetic science. A brief sample comparison of genetics texts published in the 1990s shows that this ideology continues to be represented even recently.

Chapter three begins with a consideration of what phrases like "the gene for" could possibly mean. As I show, the sheer non-linear complexity of the cellular machinery in which nuclear DNA functions poses serious difficulties for the casual use of such phrases. Next I show that particularly in the context of human disease, there exists a similar non-linear complexity in the social realm. With these considerations in mind, I move to an examination of one of the latest attempts to account for alcoholism from the perspective of genetics and evolutionary theorizing. I argue that this attempt, the "frugivory hypothesis," betrays the ignorance of those working in behavior genetics and "darwinian medicine" concerning longstanding problematics in studying the biology sociology of alcohol problems.

There are alternative strategies for explaining alcohol problems in longitudinal perspective, even from a strictly biological perspective, and I offer some examples in this chapter.

The fourth and final chapter sketches a modest proposal for dealing with these thorny issues, particularly how we can benefit from an appreciation of the limits of heredity in explanations of behaviors and social problems such as alcoholism. I explore on the one hand a reassessment of the distinction between disease states and social deviance, drawing on the idea of “drinking careers” from alcohol studies, and on the other hand the ramifications for alcoholism research of employing some insights from Developmental Systems Theory (DST). I find that DST may provide a useful framework in which finer-grained explanations of the complex causal situation in alcoholism may one day be modeled.

Finally, a note on terminology: In presenting the heterogeneous nature of alcoholism I have been careful to use a number of different terms for alcohol-related problems. By continually shifting among terms like “problem drinking,” “alcoholism,” “alcohol problems,” or “alcohol-dependence,” I employ a not-so-subtle rhetorical device meant to emphasize the heterogeneity of phenomena and circumstances that are often crudely and clumsily filed under the rubric of “alcoholism.” In the vast literature on the subject there is frequent confusion whether alcoholism is a behavior, condition, disorder, or disease. While many of those that study addiction and alcohol problems closely will be quick to acknowledge the subdivision of this term through empirical research in recent years, this fact has not been realized by those focused on human behavior genetics, or the popular-science journalism that broadcasts their work.

Chapter 1: Alcoholism(s) and the geneticization of alcohol problems

In this chapter I will isolate some conceptual and empirical problems brought up by genetic reductionist accounts of alcoholism. The survey begins with the variety of methods that produce evidence suggesting hereditary or genetic contributions to alcoholism, proceeds to problems of defining and measuring alcohol-related behaviors, and finally examines the most widely used diagnostic criteria for alcohol abuse/dependence.

1. Gathering Evidence for Claims that Alcoholism is Hereditary

What sort of evidence supports the notion that alcoholism is hereditary? The evidence comes from a mixture of sources, distinguishable by their underlying methodologies.:

- (1) family studies
- (2) twin studies
- (3) adoption studies
- (4) biological & genetic marker studies
- (5) animal studies
- (6) evolutionary reconstructions

In order to render apparent the multiple and shared limitations of these approaches, and to provide a perspective from which alternative factors can be compared, we must look closely at the strategies for gathering evidence, generating and testing hypotheses in each of these types of studies, how these methods arose, and then examine the limitations attendant to such strategies.

Family Studies

Evidence for the hereditary basis of alcoholism has been gathered by looking for statistical patterns in large family pedigrees, or alternatively, by comparing relatives of alcoholics against relatives of non-alcoholics. In both cases these methods are called “family studies” as the unit of analysis does not delve deeper than the level of family associations. For example, one much quoted finding states “Alcoholics were 6 times more likely than samples of the general population and 2 times more likely than psychiatric populations to have one or both parents who were alcoholic” (Cotton 1979, p.89). This from Nancy Cotton’s review of “The Familial Incidence of Alcoholism,” which focuses on thirty-nine studies of alcohol-related problems in families and offers a meta-analysis of their results. These studies employed methods ranging from mail-based surveys to interviews with trained psychiatrists. Cotton chose studies that had gathered data concerning the drinking problems or habits of family members (most often parents) and also linked this data to individual subjects who were alcoholics⁹ themselves. This study concluded that “In every study of the families of alcoholics and non-alcoholics, the incidence of alcoholism was higher in the families of alcoholics” (Cotton 1979, p. 111). The family study strategy thus employs observations, measurements and comparisons between groups of families, supporting the common adage “it runs in the family.”

The problems attendant to this study of alcoholism are those shared by many strategies for patient-focused social scientific research. First, the studies rely heavily on the

⁹ This is a slippery point in the chain of reasoning, for among the studies there is little overlap concerning the definitions of alcoholism and mental illness. In some cases researchers defined mental illness on the basis of whether a subject ever consulted a psychiatrist (Cotton 1979, p. 103).

patients themselves for information about the drinking patterns of their relatives. It is possible that subjects are consistently over- or under-estimating the diagnosable frequency of alcoholism among their family members. This questionable reliability for reporting on the status of their own family members only further problematizes any extrapolations from these samples to the general population. Also, the samples gathered are made up almost entirely of hospitalized patients or inpatients of psychiatric or substance abuse clinics. This limitation of the samples clearly calls for qualifying the conclusions that “the incidence of alcoholism in families of *alcoholics seeking help* is higher than in non-alcoholics or *alcoholics who do not seek help*.”

But there is another limitation to this type of evidence. Family study findings are statistical in nature, limited to the greater frequency of alcoholism among relatives of alcoholics compared against either the general population or abstainers. Evidence gathered in this way points to an hereditary link, but does so without discrimination between cultural heredity and biological mechanisms. In other words, finding that there are more alcohol problems among families of alcoholics than non-alcoholics points to the result of either socialization and learning, or some unspecified inherited biological factor. Absent a means for distinguishing among these sources of contribution (or even more challenging, an understanding of how they co-mingle), family studies provide only *suggestions* of a biological hereditary basis for alcoholism. No explanation of an hereditary aspect of alcoholism can be generated solely by family studies.

Twin Studies

When today’s journalists, pundits and scholarly writers mention hereditary aspects of alcoholism, they often refer to so-called “twin studies” that have been published since the mid 1960s. These employ a strategy of comparing the incidence of alcoholism and other

social and psychological disorders between identical and fraternal twins. The logic goes as follows: identical twins have the same genes, so any differences they exhibit are not a function of the genes they possess, but rather of social or culturally mediated forces. Conversely, the similarity of identical twins inhabiting very different social and cultural environs is thought to indicate the influence of purely genetic factors. Particularly in cases of identical twins that have been separated, if there is a higher incidence of a disorder among those whose biological parents also had the disorder compared with those twins whose biological parents did not have the disorder, it is concluded that the increased incidence betrays the power of the genes.

The idea for using identical twins as research objects is usually attributed to Francis Galton and an article he wrote for *Fraser's Magazine* in 1875. However, there is good reason to conclude that Galton did not envision the twin study method as we know it today. Rather, he saw twins as valuable objects of research for uncovering the power of *social* forces on the character of adults (Rende et al. 1990). He was interested in the extent to which social assimilation could render two *different* people *similar* (and vice-versa) from childhood through maturity. He wrote:

We might begin by enquiring about twins who were closely alike in boyhood and youth, and who were educated together for many years, and learn whether they subsequently grew unlike....We can enquire into the history of twins who were exceedingly unlike in childhood, and learn how far they became assimilated under the influence of their identical natures (Galton 1876, p. 566).

Galton's "twin method" was constructed to gauge the strength of social, not genetic or hereditary forces. Moreover, he did not propose anything resembling the method we are

familiar with today, of comparing fraternal and identical twins to determine the power of genetic forces.¹⁰

Despite the historical problem of determining to whom to attribute the “discovery” of the modern twin-study method, researchers have homed the technique since the early 1970s and applied it to disorders ranging from anti-social personality to addictions to food, sex, and even gambling.¹¹

In textbook accounts of the twin-study evidence for a genetic basis for alcoholism, the most commonly cited sources are European studies of twin cohorts (Goodwin et al. 1973; Goodwin 1979; Bohman 1987; Cadoret and Gath 1978; Schuckit et al. 1972). More difficult to find are acknowledgments in the literature of accounts of problems with twin-study methods written as early as the 1960s (Scarr 1968; Heath et al. 1989; Gurling et al. 1981; Murray et al. 1983).

Sandra Scarr’s compelling critique of the twin study method focuses on the chance for confusing environmental and genetic factors in certain twin study designs. In many of these twin studies, researchers employ measures of twin zygosity gathered through either statements from their parents or from medical records, without comparing the two for matches. The clever strategy for assessing bias in favor of genetic factors goes as follows: first look at the subset of twins in the study whose parents either did not know, or were *wrong* about their zygosity (i.e., answered incorrectly, claiming their twins were identical when tests and records indicated that they were fraternal--and vice versa).¹² This subset gives us a

¹⁰ See Rende et al. (1990) for an assessment of the roots of this confusion.

¹¹ On gambling see Winters and Rich (1998); Eisen, Lin, et al. (1998). On eating disorders see e.g., Klump, Kaye et al. (2001). For an application of this method to the study of sexual orientation see Kirk, Bailey, et al. (2000). The method has also been applied to the study of loneliness, see e.g., McGuire & Clifford (2000).

¹² This produces an admittedly small sample of only 11 twin sets.

chance to test the relative power of the family's expectancy of "identity" against true genetic "identity." Do those twins mistakenly thought to be identical behave as similarly as identical twins? Is parental confusion more powerful than genetic factors in shaping twin behavior? Looking at a rather small American sample of twins whose parents were mistaken about the zygosity of their twins, Scarr found that genetic differences are more important than parents' perceptions of the genetic differences, therefore supporting the idea that environmental bias in twin studies is not a serious difficulty (when taken on its own terms). There is environmental bias, it is just not *stronger* than genetic identity. And in cases where twins share the same genes and yet are believed to be fraternal twins, their difference, measured both by themselves and by their activities in the social world, is remarkably close to difference of fraternal twins who were mistakenly thought to be identical (Scarr 1968, p. 226). This finding underwrites a degree of skepticism concerning the immunity of twins to the social forces exerted by their parental environment or social contacts. Galton may have been right all along about social influence and identical twins: twins provide a convenient gauge not only of the power of heredity, but of the confounding influence of social forces operating in the guise of heredity. For the study of alcoholism, twin studies offer a glimpse into the competition of social and genetic forces, but these are limited by the shared family context in which they often take place. Consequently, arguments built on them must be considered with a reasonable skepticism.

Adoption Studies

Applying a methodological twist to family and twin study strategies, adoption study methods involve the use of subjects (sometimes twins) who have been adopted away from their biological parents (Bohman et al. 1981; Cloninger et al. 1988; Goodwin et al. 1973).

Using a technique described as "cross-fostering" Cloninger et al. parsed their sample at first

into several groups for comparison: children of alcoholic biological parents, children of non-alcoholic biological parents, adoptee alcoholic parents, adoptee non-alcoholic parents. In Table 1.1 these groups have been arranged in a matrix, generating four possible combinations.

Table 1.1 — Matrix for Adoption Studies of Alcoholism.

	Alcoholic Adopting Parent	Non-Alcoholic Adopting Parent
Child of Alcoholic Parent	A	B
Child of Non-Alcoholic Parent	C	D

This matrix is treated as a tool for disentangling genetic from social contributions to alcoholism by parents (i.e., biological versus social heredity). If a large proportion of children of non-alcoholic parents become alcoholics themselves when adopted by alcoholic parents (C), this would support a socialization or learning model of alcohol-related behavior.¹³ On the other hand, if a large proportion of adopted away children of alcoholic biological parents become alcoholics themselves when adopted by non-alcoholic parents (B), this would seem to support the notion that the hereditary mechanism behind alcoholism is largely genetic. Furthermore, by comparing these two groups against each other there is a possibility of assessing the *relative strength* of these two forms of heredity (social v. biological) for the sample of subjects under study. So if a *greater proportion* of those in cell (B) than in

¹³ An elegant learning model is offered in O'brien, Childress et al. (1992).

cell (C) become alcoholic, we may be warranted in saying that the biological forces of heredity exert greater influence than the social forces.¹⁴

This type of matrix allows for other interesting comparisons, involving subdivisions of each cell—by race, sex, socio-economic status, or any number of social or biological variables. The possibilities multiply further if alcoholic status can be determined for both mothers and fathers (biological and adoptee), and relative influence can be determined for each of these subgroups.¹⁵

There is an element missing in this research design that is crucial to the status of claims regarding the hereditary nature of alcoholism: to the extent that results suggest biological rather than social means of hereditary transmission of drinking behavior the methodology fails to address the issue of the underlying biological mechanism. While there may be a biological basis for the hereditary patterns, it may be premature to assume that this basis is wholly genetic (as a counterexample I present an alternative biological, non-genetic mechanism in Chapter 3).

Biological and Genetic Marker Studies

Another research method used in the quest for the genetic factors with which to explain alcoholism takes a more direct biological approach. Beginning in the 1940s and

¹⁴ Such claims require a number of caveats—such as the limitations of certain characteristics of the sample (at what age do the adoptions take place? Does the adoption agency match race or economic status between adoptee and biological families?), and also the extent to which the sample is generalizable to the larger population, and so on.

¹⁵ This is precisely where Cloninger et al (1981) and Bohman et al. (1981) focused their attention. Cloninger et al. found that certain types of drinking behavior seemed to appear only in males, and most prominently among those whose biological parents exhibited the same behaviors, whereas Bohman et al. found that there were parallel but weaker patterns for women.

gaining considerable momentum in the 1970s, biochemists and clinically minded researchers sought to uncover individual metabolic products, enzymes, or genes that appear with statistically significant frequency in groups of alcoholics but remain relatively scarce in the general population. In these cases, researchers dispense with pedigrees and large family trees and focus instead on special populations gathered from alcohol and drug treatment centers, psychiatric clinics, prison populations, or other managed settings. I parse these studies into two stages: exploratory and confirmatory. In the “exploratory stage” researchers mill through tissue samples, blood, or other biological materials gathered from confirmed alcoholics and look for salient frequency differences from the general population (these explorations, particularly in neurological contexts, may employ cadavers).

In the “confirmatory stage” researchers test these salient factors in different populations than the original study (often in a different country), or collect additional data on possible confounding variables. There are a vast number of elements that have gathered attention through this method, including blood constituents,¹⁶ a variety of neuroreceptors and the genes that code for their production,¹⁷ metabolic enzymes,¹⁸ and even enzymes that act within the brain.¹⁹ After finding a statistical association between some molecule and rates of alcoholism in a population, researchers deduce which genes are involved in the

¹⁶ One proposed candidate is platelet monoamine oxidase B (MOA). See e.g., Anthenelli et al. (1998).

¹⁷ For work on the dopamine D2 receptor and associated genes, see Blum, Noble et al. (1993).

¹⁸ Aldehyde Dehydrogenase or ALDH, see Ferguson and Goldberg (1997).

¹⁹ Arylsulfatase A. See Park, Poretz et al. (1996).

production of these molecules, and perform further studies to determine the degree of association between these genes and alcohol-related diagnoses.²⁰

The significant limitation to this approach stems again from the vagueness concerning the connection of the underlying mechanism for alcoholism. When findings show a stereochemical variant of the sulfate-reducing enzyme Arylsulfatase A (ASA) occurs with lower frequency in alcoholics than in non-alcoholics, researchers are still faced with the question whether the frequency difference is *responsible for* or a *result of* long term exposure to alcohol, a question they frequently beg (Hulyalkar et al. 1984; Manowitz, Poretz et al. 1998; I discuss individual examples of this at the end of this chapter). So while marker studies manage to get closer to possible genetic underpinnings of some alcohol problems, they do so in a way that leaves open the question of the underlying mechanism, or the specific biochemical pathway, possibly introducing spurious associations as a result. As I discuss further in chapter 3, even the action of genes and their products is mediated by a host of environmental factors, so gene-condition correlations without controls for a number of environmental variables are not particularly informative. This limitation appears in the frequent failure of “confirmatory” studies, where in many cases the findings of candidate markers cannot be replicated using a different sample of subjects (Conrad and Weinberg 1996; Holden 1994; Sander et al. 1999).

A recent search of a database devoted exclusively to alcohol studies reveals over 1200 published articles touting or testing biological and genetic markers for alcohol-related

²⁰ These searches can even take place entirely in isolation from information about the biological function of these genes—probing only for *associations* between chromosome loci or entire genomes and alcohol-related diagnoses in a sample. For one review of such approaches see Reich et al. “Genome-wide search for genes affecting the risk for alcohol dependence” (1998).

behaviors, problems, or disorders in the previous two decades.²¹ Despite the lack of known mechanisms that could bridge the gap between genetic material and human alcoholism, this cottage industry devoted to finding markers for alcoholism continues unabated.

Animal Studies

Another means of isolating biological or genetic factors related to alcohol use and abuse employs other organisms—usually rats, mice, rhesus monkeys, and common fruit flies (*Drosophila melanogaster*).²² This research takes place in two distinguishable steps. Often researchers attempt to replicate a specific aspect of human alcoholism in animal models before probing for physiological differences between these animals versus a control group. For example, a researcher might induce some symptoms of physiological withdrawal in rats by feeding a specific mixture of alcohol and water over a period of time. Rats experiencing the most severe withdrawal symptoms (seizures and death) after alcohol is removed from their diet can be treated as a sample with a specific biological relationship with alcohol that can be teased out by careful comparison against rats with no significant withdrawal symptoms. The comparison might point to a type of brain cell, a digestive enzyme, or dietary supplement as the salient difference. Just as in cases of marker studies on humans, researchers then isolate the gene or set of genes that produce the biological marker. This becomes the basis for claims to have found genes for alcoholism. Taken on its face,

²¹ National Institute on Alcohol Abuse and Alcoholism's "Alcohol and Alcohol Problems Science Database" ETOH (<http://etoh.niaaa.nih.gov>; accessed 6/10/02).

²² The history of attempts to model alcohol-related behavior in animals would be a lengthy and fascinating undertaking. Raymond Pearl conducted experiments using chickens (Pearl 1917). Other early-twentieth century inquiries involved guinea pigs, rabbits or dogs. In one attempt to work with guinea pigs researchers discovered they could not compel the animals to drink any fluids containing alcohol. They resorted to placing alcohol-soaked cotton balls in with the animals in air-tight containers, until they appeared intoxicated (Stockard 1912).

however, such claims are best understood as discovery of genes for symptoms of alcohol use in *rodents*.²³

The criticism most frequently levied against this approach centers on the degree to which human alcoholism can be modeled by these animals. The measures of alcoholism that animal studies attempt to model are often specific features of long-term alcohol-*abuse* rather than aspects we would normally associate with human alcoholism (e.g., such as withdrawal seizures, liver damage, nervous system effects, etc.; see Norton 2001).²⁴ In other words, this research offers a window into the biology of the *effects* of prolonged alcohol use, but fails to shed any light on the very human condition of the desire to drink or to the social and personal problems that might lead to or result from frequent drinking to excess (Erickson 1996). Alcoholism in humans is not *defined* in biological terms alone—even the Diagnostic and Statistical Manual of the American Psychological Association defines alcohol dependence in terms of a subtle mixture of social and physical criteria (see below).

Evolutionary Reconstructions

Recent endeavors to support the genetic reductionist account of alcoholism take evolutionary theory and paleontology as their starting points. Practitioners of “Darwinian Medicine” and “Evolutionary Psychology” claim that the alcoholic problem we face today is an evolutionary predicament. Natural selection has produced genes that predispose certain

²³ See for example Deitrich, Bludeau et al (2000); Homanics and Hiller-Sturmhofel (1997); Ng, O’Dowd et al. (1994).

²⁴ Distinguishing between biological effects of long-term alcohol exposure and the criteria by which alcoholism is currently diagnosed is crucial here. While effects like those listed figure in diagnostic criteria, they account for only a narrow slice of the phenomena that lead clinicians and treatment specialists to diagnose “alcohol abuse or dependence.” These criteria are the subject of close scrutiny and critique in section 3 of this chapter.

of us to addictive behaviors, alcoholism among them. These arguments take a variety of forms, from claims that the brain is comprised of problem-specific “modules” designed to deal with situations from our evolutionary past, to zoological studies that establish the nutritional value of alcohol for a variety of species ancestral to mammals.²⁵ Speculations like these parallel many 19th century writings, when evolutionary arguments for addictive behaviors were focussed on racial and moral differences. But with the modern development of sophisticated methods for constructing evolutionary scenarios from limited evidence, these speculations have taken on the air of legitimate scientific research.

Since the methods involved in current evolutionary reconstructions are so varied, and many have received wide criticism, I turn my attention in chapter 3 to the latest argument for the evolutionary origins of human alcoholism—the “frugivory hypothesis.”²⁶ The critique of this argument serves as a model from which weaknesses in other evolutionary explanations can be pinpointed.

2. Alcoholism and the Problem of Heterogeneity

One of alcoholism’s chief oddities that becomes apparent when seen in historical context is a lack of conceptual clarity in both what alcoholism is (definitional heterogeneity) and how alcoholism occurs (etiological heterogeneity). These distinct concepts are commonly

²⁵ The literature on this front is too vast to provide a comprehensive list, but for a quick look at some of the more influential publications, see Berrigan (1999); Berlim and Abeche (2001); Daly and Wilson (1999); Glander (1998); Hill (1995); LeGrand (2001); Logan and Qirko (1996); Nesse (1984; 1990; 1994; 1999a; 1999b; 2001), Nesse and Berridge (1997); Nesse and Williams (1994; 1997; 1999). Chapter 3 includes a detailed review and critique of the hypothesis advanced in Dudley (2000).

²⁶ Frugivory is a term that denotes feeding on fruit.

confused. Any given definition of alcoholism we examine may lump together phenomena that have completely distinct underlying mechanisms; and any proposed etiological description of alcoholism (how the condition develops and progresses), can be applied too liberally and conflate meaningful subcategories of the experience of alcoholism. But this is only the background setting for a much larger problem: there are a great number of both definitions and etiological accounts of alcoholism, and the interrelations among them are staggeringly numerous and complex.

Take for example the 19th century proposals that alcohol problems result from the lack of a certain vitamin in the diet.²⁷ This can be treated as both an etiological and definitional hypothesis. If research can uncover a specific biochemical pathway by which a lack of a vitamin produces a craving for some nutrient-rich source (on analogy with salt-cravings, for instance), then the etiological hypothesis can be fleshed out and put to a critical test. However, at the same time this etiological hypothesis can influence the definition of alcoholism. We could construct definitional criteria for alcoholism and include among them “lack of vitamin X.” Such a definition will have an impact on future research on the problem, because samples of “alcoholic” patients will be selected on the basis of this criterion for testing against non-alcoholic samples. The subsequent findings of research performed on these samples run the risk of exaggerating the importance of the vitamin deficiency, and more problematic, runs a risk of *masking* other significant features of the phenomenon. This is only one example of the manner in which disease etiology and disease definition are tightly intertwined.²⁸

²⁷ Lesley Keeley (1892) believed this to be true, and even developed a network of treatment clinics and dietary supplements based on the idea.

²⁸ I am sympathetic with writers that cast this “definition/observation” cycle as a fundamental feature of reasoned systems applied to humans. Similar patterns appear in areas

While it is important to distinguish answers to these “how” and “what” questions, in historical context they are often confused or conflated. Since the etiological story of alcoholism is foundational to any definition of alcoholism, I initiate this investigation with a brief look at the etiological heterogeneity in accounts of the disorder.

A handful of scholars have attempted to compile comprehensive reviews of different descriptions of “alcoholisms.” Most prominent among them is E.M. Jellinek’s review of theories on the origins of alcoholism in the first half of the twentieth century (Jellinek 1960).²⁹ In his classic text *The Disease Model of Alcoholism* he proposed the most widely studied and accepted subdivision of disease definitions of alcoholism based on a review of research literature at the time (Jellinek 1960). Increasing in severity through the scale, he labeled these five subtypes (he called them “species”) “alpha” through “gamma” alcoholism. Alpha and beta alcoholism were defined by increasing psychological dependence alone, without physical withdrawal or craving. Epsilon alcoholics were plagued by uncontrolled drinking occurring in periodic episodes. The gamma and delta alcoholics, however, showed signs of physiological adaptation to alcohol, including withdrawal symptoms, severe craving, and “loss of control” over drinking. The delta alcoholics exhibited withdrawal symptoms with the least exposure to alcohol, and consequently drink almost continuously, with the greatest physical consequences from attempts at abstinence. Jellinek proposed this scale of alcohol problems was inherently progressive—individual patterns could potentially be described in terms of transitions from one stage to another. However, Jellinek’s main

like criminal law (where the definition of a crime feed-forward to influence both behavior of individuals and measurements of their behavior). Ian Hacking (1995) calls this the “looping effect of human kinds.” See chapter 4 for more discussion of his approach and how patterns such as this may shape or current understanding of alcohol problems.

²⁹ See Roebuck and Kessler (1972) for another compelling overview of the constitutional, psychological, and sociological accounts of alcohol problems and the tensions among them.

concern in developing his subclassification of alcohol problems was to define the limits of the disease concept of alcoholism. In this respect he argued that *only* delta and gamma alcoholism truly belonged within the disease category; subtypes alpha, beta and epsilon were at bottom psychological problems and therefore not amenable to physiological, genetic, or clinical study.

In Jellinek's model, only the gamma and delta could be called "alcoholism" according to the disease model. This is a point seemingly lost in much of the rhetoric that surrounds the quest for genetic components of alcoholism in the recent popular science literature, where a wide net is cast for "alcoholism" according to diagnostic criteria like the DSM or the ICD, and the hunt continues for genetic correlations. If there is anything to Jellinek's disease classification model, it underscores the need for finer-tuned definitions that can establish first and foremost which classes of alcohol-related phenomena are amenable to hereditary analysis, rather than deploying the genetic hypothesis to all aspects of alcohol-related behavior.

Table 1.2 — Jellinek's Study of Etiological Speculation on Alcoholism - 1938-59 (adapted from Jellinek 1960)

<u>Etiological Category</u>	<u>Number of Distinct Theories</u>
Psychological Illness	33
Symptom of Psychological Illness	13
Physiopathology	
<i>Allergy</i>	6
<i>Biochemical-Physiological Disease</i>	13
<i>Brain Pathology</i>	9
<i>Nutrition</i>	7
<i>Endocrinology</i>	6
Indicative of Pharmacological Response	24
(TOTAL)	(111)

Jellinek's meta-analysis of the alcohol literature circa 1960 compiled no less than 111 distinct theories, which fall into four broad subsets (of his own naming; See Table 1.2).³⁰ Such a large number of theories concerning the underlying cause of alcoholism serves to highlight the heterogeneity of alcoholism on (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 work, but is arguably less of a problem today, as researchers have converged on the diagnostic criteria provided in sources like the American Psychological Association's Diagnostic and Statistical Manual (DSM IV) and the diagnostic guidelines published by the World Health Organization (WHO; these diverge very little from the DSM criteria). Although these criteria are problematic on their own terms they perform a positive function. Namely, 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 that the criteria isolate. Nonetheless, the DSM criteria have been criticized widely for their conflation of social dysfunction with a disease state (see Hess 1995 on this point, especially chapter seven; see also Conrad & Schneider 1980; Peele 1985). This points to a deep underlying problem: there

³⁰ Interestingly, there are obvious parallels between these decades-old etiological speculations and those explored in research and treatment literature today. For instance, in table 2.2, the reference to alcoholism as an "allergy" can be seen today in the literature of Alcoholics Anonymous where the disease is described as a specific allergy to alcohol held by the individual. This allergy separates the members from the rest of humanity because unlike them, their relationship with alcohol is beyond the reach of their will or power to control. As I will discuss further, there are also recent attempts to link alcoholism with nutritional deficiencies, specific pathologies of the brain, even as a specific response to the inherent biochemical properties of alcohol and its effects on human tissue (i.e., pharmacological response). That so many different explanations of alcoholism have been pursued throughout the twentieth century without an emergent consensus on what defines the condition indicates that some fundamental questions still need to be addressed.

is no purely physiological or medical description of alcoholism, beyond physiological dependence on or severe toxic effects from alcohol exposure. Absent such a model, all researchers can do is to find people *who have been diagnosed* for largely social reasons, and then work backward by trying to find common physiological states among those so labeled. This approach dominates in genetic marker studies and ultimately begs the question of the biological-genetic foundation of alcoholism. See the next section for a closer analysis of the DSM criteria.

The large number of accounts of alcoholism also points to the deep-seated need for conceptual clarification among possible variants of the phenomenon, a situation describable by the term “alcoholisms.” 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. While there is some work in this direction (testing the validity of the DSM criteria, for instance) most genetic studies do not question the origins of the criteria for which they seek genetic associations. The heterogeneity of alcoholism does not stop with diagnostic criteria and definitions. In addition to these sources of fracture within alcoholism, I would add the complexity of providing an account of the function of genetic material.

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 this far more basic level. As I will discuss in detail at the end of this chapter, the lack of a one-to-one correlation between genetic material and protein products betrays the importance of taking non-genetic factors into account in any reductionist program. The situation facing those trying to link molecular genetic phenomena with complex human disease is daunting. Even the presence of a single enzyme within a cell

cannot be explained by the presence of a single gene—there are other factors that participate in the complicated chain of events that “read” genetic material and produce molecular products like enzymes, amino-acids, or proteins. And in cases where many genes are involved in the causal pathway an accounting of the multiple sets of these gene-reading factors is required. With such a complicated picture at the molecular level alone, it is surely folly to rush to conclusions about genetic causes of complex human behaviors, conditions, or disorders.

There are at least two models for developing descriptions, definitions, and diagnostic criteria for psychological disorders. One centers on the interplay of statistical investigations with clinical observations, the other stems from less technical typological constructions like those used by physicians throughout the 19th century.³¹ The statistical approach uses the technique called analysis of variance, often called ANOVA. An example of this approach can be seen in the development of categories of personality traits like those found in Cattell’s 16 personality factors (Cattell 1970; see Table 1.3). In order to develop these categories, psychologists administered a vast number of test questions to a large sample. Each individual’s answers were compiled and coded, and the entire data set was evaluated using a technique that quantifies the degree to which each of any two particular answers vary in exactly the same way across the entire sample. So if everyone in the sample answered “yes” to two different questions, those questions would be called co-variant (and the same would be said if the two questions are inversely related, that is, if every “yes” to a certain question

³¹ Although American medical and clinical practice during the 19th century was quite an unprofessional affair (Starr 1982), there was a proliferation of complex categorical and explanatory schemes during this period built on the basis of both humoral theory and germ theory.

occurs with a “no” to the second question). These two questions would receive the highest possible score of covariance.

Now, imagine we could take these questions and divide them up into clusters based on the “factor” we hoped they were measuring. We could name each of these factors and then run the ANOVA test within the set of questions and test the degree to which our predictions of their covariance match the actual data. Questions that do not meet a certain cut-off of co-variance are removed from the “factor” clusters, and in this way we separate the more meaningful questions from clutter. Then we test these “factor” clusters against each other to see if they co-vary, and in doing so determine whether two of the factors should be combined into a single factor, and so on. Using this method of analyzing variance, developing clusters or factors, and testing factors against each other and our theoretical predictions, we can develop a sense for what general categories are most salient for personality in a sample. Cattell’s 16 personality factor instrument is just such an extension of statistical analysis and definitional feedback using the ANOVA technique, among others. The factors developed this way go by names like “submissiveness,” “parmia,” “radicalism,” and so on. Using this ANOVA method to determine the statistically meaningful clusters of variables in a data set has been found to be a remarkably useful tool throughout the human sciences, and a large number of researchers consider it a fundamental tool for scientific inquiry into many aspects of the human condition.

Of course the power of this approach is limited by the type of questions we ask, and the answers respondents choose to give. To the extent that diagnoses of alcoholism rely on physicians questions and subjects’ answers, the patterns we find in large collections of clinical records will reflect this self-reporting error. Even with statistically sophisticated analytical tools, the initial data in such situations is inherently limited by the fact that humans

possess the ability to recognize, reflect on, and interpret their own behaviors—and these reflections do not exist in a historical or cultural vacuum.

Table 1.3 — Cattell's 16 Personality Factors (1970)

Sizothymia Reserved, detached, critical	A	Affectothymia Warm-hearted, outgoing, easygoing
Low intelligence	B	High intelligence
Ego weakness Affected by feelings, emotionally less stable	C	High Ego Strength Stable, mature, faces reality, calm
Submissiveness Obedient, mild, easily led, docile	E	Dominance or ascendance Aggressive, competitive, stubborn
Desurgency Sober, taciturn, serious	F	Surgency Enthusiastic, heedless, happy-go-lucky
Low superego strength Disregards rules, expedient	G	Superego strength, character Conscientious, persistent, moralistic, staid
Threctia Shy, timid, restrained	H	Parmia Adventurous, "thick-skinned", socially bold
Harria Tough-minded, rejects illusions	I	Premisia Tender-minded, sensitive
Alaxia Trusting, accepting conditions, tolerant	L	Protension Suspecting, jealous, dogmatic
Praxernia Practical, has 'down-to-earth' concerns	M	Autia Imaginative, bohemian
Naivete Forthright, unpretentious	N	Shrewdness Astute, worldly, polished
Untroubled adequacy Self-assured, placed, secured	O	Guilt proneness Apprehensive, self-reproaching, insecure
Conservatism of temperament Conservative, respecting	Q1	Radicalism Experimenting, liberal, analytical
Group dependency Socially group dependent, 'joiner'	Q2	Self-sufficiency Self-sufficient, resourceful, prefers own decisions
Low self-sentiment integration Uncontrolled, lax, follows own urges	Q3	High strength of self-sentiment Controlled, exacting, will power, socially precise
Low ergic tension Relaxed, tranquil, torpid	Q4	High ergic tension Tense, frustrated, driven, overwrought

The second model for the construction and evaluation of diagnostic categories is more antiquated. Simple observations of individual cases throughout the 19th century provided material for developing categories of ailments whose effects were more subtle than death. Prior to Francis Galton's introduction of regression analysis and Ronald A. Fisher's correlation coefficient (1930), physicians interested in mental phenomena would mine case histories for similarities in the patient's physical presentation, course, duration, and outcome. By grouping according to symptoms and disease course physicians developed categories of ailments whose relationship with natural phenomena were inherently colored by social, racial, and economic factors (Szasz 1974). Some writers have argued that the first studies of alcohol-related behavior betray these corrupting influences, and the categories so developed suffer from wide cultural variability as a result (Alasuutari 1992; Conrad and Schneider 1980; Levine 1981; MacAndrew and Edgerton 1969; Valverde 1998). Since modern alcoholism research focuses on the construction of disease definitions using statistical methods, I turn next to a discussion of the DSM criteria and their inherent limitations.

3. "Alcoholism(s)" and Diagnostic Criteria from the DSM IV-R

The American Psychological Association (APA) publishes a manual designed to aid in diagnosis and treatment of psychological conditions called the *Diagnostic and Statistical Manual* (DSM). The DSM provides categories, definitions, and diagnostic criteria for a wide variety of conditions, disorders, and syndromes of interest to medical and psychological professionals. The complex task of refining diagnostic criteria for alcohol-related problems began as early as 1940, when E.M. Jellinek identified 39 different diagnostic systems in use by current practitioners (NIAAA 1995; Schuckit 1994). Since this time, the DSM criteria have become the principal tool for classification and diagnosis of alcoholism in the United

States.³² Alcohol-related disorders and diseases first entered into the DSM criteria as a subset of the category of disorders labeled “personality disorders, homosexuality, and neuroses” (NIAAA 1995). On the third revision of the DSM in 1980, the classification of alcoholism was shifted from its place as a “personality disorder” to a “substance abuse disorder,” and it was split into two distinct categories labeled “alcohol abuse” and “alcohol dependence.” These categories are divided roughly according to the relevance of physiological consequences (such as withdrawal effects) compared with psychological features (behaviors and attitudes surrounding alcohol use). Of these two divisions, alcohol *dependence* is used most frequently in psychological studies, although it is sometimes conjoined with features of alcohol abuse (Schuckit 1994). This subtype classification persists today, in the DSM fourth revision, under the heading “Substance dependence” (see Table 1.4).

³² The World Health Organization (WHO) also maintains a classification system in which alcohol-related problems are defined. This document, the International Classification of Diseases tenth revision (ICD-10), was created for the purpose of compiling worldwide statistics on illness and death, and consequently includes standard measures of alcohol-related diseases such as cirrhosis of the liver. Its use as a diagnostic instrument is therefore quite limited (NIAAA 1995; Schuckit 1994).

Table 1.4.—DSM-IV criteria for substance dependence. From the *Diagnostic and Statistical Manual of Mental Disorders, fourth Edition*. Copyright 1994, American Psychiatric Association.

<p>DSM-IV CRITERIA FOR SUBSTANCE DEPENDENCE</p> <p>A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:</p> <p>(1) <u>tolerance</u>, as defined by either of the following:</p> <p style="padding-left: 40px;">(a) a need for markedly increased amounts of the substance to achieve <u>Intoxication</u> or desired effect</p> <p style="padding-left: 40px;">(b) markedly diminished effect with continued use of the same amount of the substance</p> <p>(2) <u>Withdrawal</u>, as manifested by either of the following:</p> <p style="padding-left: 40px;">(a) the characteristic <u>withdrawal syndrome</u> for the substance (refer to Criteria A and B of the criteria sets for Withdrawal from the specific substances)</p> <p style="padding-left: 40px;">(b) the same (or a closely related) substance is taken to relieve or avoid withdrawal <u>symptoms</u></p> <p>(3) the substance is often taken in larger amounts or over a longer period than was intended</p> <p>(4) there is a persistent desire or unsuccessful efforts to cut down or control substance use</p> <p>(5) a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects</p> <p>(6) important social, occupational, or recreational activities are given up or reduced because of substance use</p> <p>(7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced <u>depression</u>, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption)</p> <p>Specify if:</p> <p style="padding-left: 40px;">With Physiological Dependence: evidence of tolerance or withdrawal (i.e., either Item 1 or 2 is present)</p> <p style="padding-left: 40px;">Without Physiological Dependence: no evidence of tolerance or withdrawal (i.e., neither Item 1 nor 2 is present)</p>

These criteria betray at least three fundamental problems: (1) the loose heterogeneity of alcoholism concepts, (2) a degree of relativity introduced by reliance on social and cultural consequences of behavior for defining a clinical-physiological disease state, and (3) the imperfection of animal models of human conditions that have obvious social and cultural dimensions.

In Table 1.4 we can see that the criteria for substance abuse (including alcoholism) are very flexible, requiring that an individual meet “three (or more) of the following [criteria], occurring at any time in the same 12-month period.” At first glance, this might seem a perfectly manageable set of criteria, but with a bit more scrutiny it becomes clear that it clusters a remarkably broad and diverse set of phenomena under the rubric of a single disorder. The point of looking closely at this diversity of phenomena is to determine whether or not we are over-stretching the concept of “substance abuse,” perhaps overlooking meaningful subsets of these criteria that could stand perfectly well on their own. In short, we can use these criteria in part to help answer our continuing question: “how many ‘alcoholisms’ are there?”

Meeting at least three of the seven criteria allows for a considerable number of different combinations. We can find the exact number using this equation:

$$\frac{(7!)}{(3!)(4!)} = \frac{(7 \bullet 6 \bullet 5 \bullet 4 \bullet 3 \bullet 2 \bullet 1)}{(3 \bullet 2 \bullet 1)(4 \bullet 3 \bullet 2 \bullet 1)} = \frac{5040}{144} = 35$$

If we limit the number of combinations to those that meet three of the seven criteria, there are *at least 35 possible arrangements*. In other words, there are 35 *different* criteria-meeting scenarios in which a particular individual can be diagnosed with substance abuse disorder.

And if we include all the possible combinations of meeting *more* than three criteria, the number of scenarios increases still. This loose heterogeneity spreads the phenomenon of alcoholism disturbingly thin.

The second troubling aspect of these criteria stems from the elements that invoke social and cultural context for assessing the presumed clinical-physiological state. One criterion holds that “(5) a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects.” While it might seem trivial at first glance, the amount of *time* and *resources* an individual spends in substance-related activities is influenced by the amount of time and resources an individual has relative to other individuals in a population. Furthermore, given a certain level of resources in a population (determined by income level, or socio-economic status indicators, for example) the differences between individuals may not reflect differences in physiological disposition towards substance abuse at all, but rather idiosyncratic and subtle situational differences among individuals. The use of time, energy, or resources expended in acquiring a substance opens a door for considerable social and cultural variance, which, if it is not treated as such, can very easily be mis-read by social scientists and clinicians as indicative of physiological (and at worst, genetic) difference.³³

³³ This point is best illustrated with a brief example. Consider for a moment the difference between alcohol use in an urban center such as downtown Baltimore, Maryland, where liquor stores dot the landscape with greater frequency than the Starbucks coffee shop. In such a setting, the time, energy, and resources required to obtain a suitably stiff drink is considerably less than in the setting of the suburbs of nearby northern Virginia. Criteria for substance abuse that gloss over the differences inherent in these settings introduce the potential for considerable mis-representation by statistics, if not outright error on their behalf.

Of these twelve criteria there are two more that introduce potentially troublesome culturally relativistic elements. The sixth criterion attributes a portion of the substance abuse definition to “(6) important social, occupational, or recreational activities are given up or reduced because of substance use.” Depending on which terms in this phrase we place our emphasis, it is clear that we can generate a number of different meanings. First, what count as “important” activities obviously vary with social and cultural context, as well as age, gender, and any number of other variables. Second, it would be difficult to generate a universal list of activities that persons in different situations and from different cultural backgrounds consider to be recreational, versus, say, social. And so on. While it may be important that an individual changes behavior in the presence of increasing use of a substance, the way we conceive of and *measure* such change must be carefully developed. For it is even conceivable that use of certain substances, in certain contexts, results in *increased* activity in some important aspect of an individual’s life (e.g., by providing a ticket into a social or occupational subcultures that one otherwise lacked).³⁴

The last of these problematic substance-abuse or dependence criteria is the seventh: “(7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption).” Here the door to culturally relative and variable phenomena is opened by bringing in the individual’s *knowledge* of there being a problem. Introductory students of

³⁴ This feature of alcohol use finds considerable support in Valliant’s longitudinal analysis of a cohort of Harvard students and their subsequent careers (Valliant 1995). There is a clear cohort of subjects for whom heavy alcohol use is tightly correlated with occupational and personal achievement.

sociology can recognize that there is a firm connection between socio-economic status and individual health that is more a function of the degree of health-education individuals receive than the quality of care available to them. Educated, upper-middle class citizens are more likely to know when there is a “problem,” no matter what they do after noticing it. This variance in socio-economic terms alone poses a problem for interpreting data filtered through this criterion.

I have argued that at least three of the seven DSM criteria for alcohol abuse and dependence introduce vague and relative cultural and social elements. Criteria 5, 6, and 7, outline behaviors, beliefs or situations whose interpretation ultimately involves features both of the cultural milieu that the individual and researcher inhabit, as well as larger cultural norms concerning appropriate or expected behavior.

If the aim is to articulate a detailed map of the cultural status of alcohol-related behaviors on a nation-wide or culture-wide scale, then these criteria, if accounted for fully and separately in diagnoses of alcohol abuse and dependence, would be a good start. In short, we could take the 35 possible criteria-sets for alcohol dependence, give them different names, and tease them out of the data gathered from any cultural or geographically textured site (using ANOVA methods) for an idea of what alcohol-related problems adhere in which situations, for which populations, and how severely.

Unfortunately this is not the strategy that is reflected in the diagnostic guidelines, criteria, and the research on genetic determinants of alcohol use that take these criteria as the starting point. It is clear from the turn toward large-scale statistical analyses of differences between populations that meet criteria versus those that do not, that the potential differences *within* the population of DSM criteria-satisfiers is not what gets attention. Meanwhile there is no dearth of work on the intra-population genetic differences among

these criteria-satisfiers. This imbalance only begins to suggest the depth at which the gene-myth may be entrenched in psychological research and alcohol studies.

It is worth noting here that the DSM criteria are predominantly employed in *research* conducted by clinicians, psychologists and social scientists; rarely are these criteria used to construct a diagnosis of an individual in the absence of a researcher attempting to generate of sample of “alcoholics.” For example, David Robinson noted that “the process by which a person is deemed an alcoholic is poorly understood....General practitioners and other helping professionals seldom diagnose alcoholism: they simply validate the judgments of the family, the drinker or whoever is presenting ‘the problem’” (Robinson 1976).

My final reflection on issues raised by the DSM criteria for alcohol abuse centers on the issue of transferring knowledge gained through studies of animal models to human contexts, and vice versa. As it stands, it is difficult if not impossible to reproduce in animals the features of alcoholism that we take to be important in its human manifestation. Looking to the DSM criteria again, it is clear that we could use animal models to study the biochemistry underlying *physical* dependence; it is not clear, however, that we could gauge in any meaningful sense the *social* or *occupational* impact of alcohol use on, say, a rodent. There is an interesting situation in the field of alcohol studies, where researchers working with rodent models frequently claim to find biological or genetic markers for alcoholism.³⁵ Meanwhile, the very definition of alcoholism for humans involves such a complex and variable mixture of social, cultural, psychological and biological factors that it can only have meaning for animals after throwing out nearly half the criteria. And even then, all that is left are biological indicators. It should come as no surprise then, that there is at present a

³⁵ See for example Deitrich, Blundeu et al (2000); Homanics, Le et al. (1998); Ng, O’Dowd et al. (1994).

dominant focus on the biological factors associated with alcoholism at the expense of an understanding of the cultural conditions that surround its manifestation, development and meaning. This preponderance of feedback from biological and genetic-determinant studies only serves to further entrench the gene-myth with respect to alcoholism.

It would paint an incomplete picture of genetic-determinist studies of alcoholism to review only the methods by which they have gathered data. The use of data gathered by genetic studies will also steer the formulation of future problems in alcoholism research. In the following section I review one of the uses to which the genetic-reductionist approach has been put, in the form of proprietary interests in gene-isolation technologies aimed at finding the “gene for” alcoholism.

4. Patenting the “Gene for Alcoholism”

One aspect of recent research into the genetic model of alcoholism that has escaped attention in the press is the increasing number of patents filed in the United States for methods generated by the quest for “the alcoholism gene.” In many of the cases where authors received attention in the popular press for discoveries of associations between biological markers and alcoholism, they have sought patents on methods of applying their findings in a treatment context. As I will show in this section, this aspect of the modern genetic reductionist program for alcoholism mirrors events from the early decades of the 20th century.

A recent search of (English language) publications in the late 19th and early 20th centuries revealed only two book-length studies devoted to the role of heredity in alcoholism. One of these is a book by G. Archdall Reid, titled *Alcoholism: A Study in Heredity* (Reid 1901). I will return to Reid’s writings in the next chapter, but for the moment I will

state that Reid was committed to a view of alcohol problems that highlighted the role of heredity and natural selection in its manifestation (evidence for this claim is provided below). Only a few years before Reid's book on alcoholism, physician Leslie E. Keeley also published a book-length study on alcoholism titled *The Non-Hereditary of Inebriety* (1902 [1896]). In it, he argues 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. 344-5).

According to this model, a poison-response bolsters immunity to the poison over the course of many generations. Furthermore, since the craving for alcohol is an individual 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 chain of private clinics for the treatment and rehabilitation of "inebriates" (using a method built around the toxin-response theory) he stood to benefit considerably from a 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 he might have for his clinic (one advertisement for “The Keeley Cure” lists 28 clinic locations across the United States).³⁶

As both Reid and Keeley show, in the years immediately prior to the rediscovery of Mendel’s work, the issue of the role of heredity in alcoholism held interest. One aspect of this focus is that the parties involved often stood to gain personally from the success of their arguments, *for* or *against* a hereditary account of alcoholism. One way to describe such financial interests is as the “entrepreneurial spirit.”

Moving forward to the last decades of the twentieth century, it is interesting to note that although the debate is no longer polarized between hereditary and non-hereditary positions, researchers have proprietary interests quite similar to their turn-of-the-century counterparts. For example, in the 1990s a common strategy for profiting from human genetic research involved patenting the methods for detecting whether an individual carries a particular gene, or gene product.

I now turn to those arguments for the genetic basis of alcoholism that appear in patents granted in the United States since about 1988. These sources offer a chance to examine the extent to which this entrepreneurial spirit affects the characterization of alcoholism in specific directions. I have two reasons for focusing on this literature. First, these documents obviously fall within the same “entrepreneurial spirit” that influences 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. Second, these patents, and the studies

³⁶ Keeley clinics arguably posed a direct challenge to both the hereditary interpretation of alcohol related problems, as well as the model on which inebriate asylums were constructed that treated the patients as though they were insane. Hence, the Keeley cure offered a more attractive alternative for persons with alcohol problems wishing to maintain a semblance of control over their lives than contemporary competitors (Valverde 1998, p. 72).

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*).³⁷ Such wide exposure bolsters the acceptance and legitimacy of these theories by the consuming public.³⁸ A closer look at these “discoveries” can help us flesh out an 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 an hereditary account of alcoholism. We can group these 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, while 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 somewhat toxic byproducts of otherwise normal biochemistry in the brain, on the supposition that a lack of these enzymes can lead to toxic build-up and brain dysfunction, eventually causing behavioral disorders, possibly even alcoholism. By subtly altering some biochemical processes of the brain, these enzyme variants are thought to play a role in altering how the mind works, generally speaking. Focussing on the genetic pathway that leads to alcoholism,

³⁷ For a detailed examination of discovery rhetoric regarding the “gene for alcoholism” in popular press sources, see Conrad and Weinberg’s (1996) article titled “Has the Gene for Alcoholism Been Discovered Three Times Since 1980?”

³⁸ I use the term “consuming” here 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.). While other ways of capturing the dynamic may be more valuable for historical purposes, at the end of the 20th century this term seems fitting. 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.

I refer to these patent claims as implicating (i) nutrient-metabolism genes, (ii) neural function genes, or (iii) neuro-receptor genes.

In the table below the claims of each of these patents are arranged according to these groupings, with detailed descriptions of the claimed causal pathway between gene and alcoholism in each case.³⁹

³⁹ Left out of this group is a patent taken out by researchers at UNC Chapel Hill (Swift et al. 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. It has been left out of the table above 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.

Table 1.5. 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 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 (i) greater activity levels of platelet monoamine oxidase, and (ii) lesser activity levels of adenylate cyclase than non-alcoholic controls. The patent is for the assays in which one can test for a ratio of these two activity levels (Tabakoff 1988).

Bradley 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) indicates “inherited substance abuse trait.” Based on two previous studies which found correlations between other lymphocyte products and mental illnesses (Bradley 1992).

Dobbins 1990 – Prevention & Treatment of Alcoholism with Chromium. The claims here are (i) alcoholism is consistently accompanied by hypoglycemia and diabetes, (ii) chromium is an essential and rare element for the regulation of insulin levels, and (iii) 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 (Dobbins 1990). No precise mechanism for the connection between alcoholism and dietary Chromium is articulated.

Neuro-Receptor Genes (Methods of testing for the presence of a “gene for” alcoholism)

Blum et al. 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 (Blum, Noble et al. 1993).

Neuro-Function Genes (Methods of testing for the presence of a “gene for” alcoholism)

Manowitz, Poretz et al. 1998 – Arylsulfatase-A alleles. Arylsulfatase-A (ASA) is an enzyme involved in the first steps of biochemical breakdown of sulfatides in many human tissues (esp. the brain). Sulfatides can disrupt the function of some neurotransmitters. Many molecular-structure variants of ASA have been found, each of which differ 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 were found in a sample of 56 *hospitalized* “alcoholics,” 12 of them to be exact (Hulyalkar et al. 1984; new sample studied in Park, Poretz, et al. 1996). This patent claims rights to several methods for the isolation, amplification, and detection of ASA variant “genes” using various tissue samples, and any kit for such detection based on any of these methods (Manowitz, Poretz et al. 1998).

Notice that each group of patents 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 (Tabakoff 1988; Bradley 1992); neurotransmitters (Blum, Noble et al. 1993),⁴⁰ enzyme deficiencies (Manowitz, Poretz et al. 1998), and even blood sugar levels (Dobbins 1990). Such heterogeneity in the mechanisms proposed as causes alcoholism is representative of studies on alcoholism that pursue profitable validation of the gene-myth.

The glaring problem one notices when reading these patent documents is the *vagueness* of the causal connection between the genetic markers and alcoholic behavior. While researchers may show a statistical association between the presence of an enzyme and different outcomes relative to diagnoses of alcohol abuse, the specific links in the causal chain from enzymes to behavior are at best only sketched roughly.

Moreover, 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 in order to determine which comes first: the physiological marker, or the alcoholic behavior. For instance, looking at the patent by Manowitz, Poretz et al. (1998) it is interesting to note that the studies finding correlations between ASA enzyme variants and alcoholism used samples of *hospitalized* alcoholics (Park, Poretz, et al. 1996; Hulyalkar et al., 1984). Since 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 already hospitalized for problems stemming

⁴⁰ Not only have these neurotransmitter gene alleles been called the “alcoholism gene”—Kenneth Blum (2001) has even attempted to label the an allele of the D2 dopamine receptor gene “alcogene.” It will be interesting to see whether this newly-coined term will

from their drinking fails to address the question whether the alcohol contributed to the measured effect, or if the measured difference contributed to the alcohol use.

On this “chicken and egg” issue, there is much philosophical and technical work presently underway (See e.g., the multidisciplinary approaches being undertaken by Beurton, Falk et al. 2000; Jablonka and 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. As I show in chapter 4, there is a promising approach, but it may require a wholesale reconfiguration of the concept of alcoholism and other addictions, in addition to imposing proper limits on the zeal for the genetic reductionist program.

5. Conclusion

This chapter has presented and isolated deep conceptual and empirical problems that threaten the tenability of the genetic reduction of alcoholism. By surveying the methods by which evidence for hereditary influences are gathered, the feebleness of statistical associations between measures of alcohol problems and genes is evident. From family study methods through biological marker studies and evolutionary reconstructions, findings meant to bolster the gene-myth of alcoholism are constructed. What these studies fail to address adequately is the problem of defining alcoholism. The very definition of the disorder is based on social and cultural criteria so vague that biologists would shun their use in their own technical work. Nevertheless, genetic investigations searching for markers, predictors, predispositions and determinants of alcoholism persist. The conceptual and empirical

find its way into future genetic reductionist accounts of alcohol related behavior, perhaps further entrenching the gene-myth.

heterogeneity of alcohol-related behavior and disease over the course of the 20th century rarely incites mention in these contexts. Even recent revisions to the DSM diagnostic criteria for alcoholism exhibit 35 possible sub-classifications with differing mixtures of social and physiological components. The problem of heterogeneity of alcoholism must be overcome but current research on the genetics of alcoholism does not offer a clear method for doing so.

In odd juxtaposition to the multiplicity of alcoholism definitions in the late 20th century, genetic reductionist models of alcoholism continue to move forward as far as the patent stage. These attempts to profit from the genetic reduction model are built on shaky ground. Without conceptual clarification at the level of the fundamental definition of alcohol problems, genetic reductionist accounts will continue to lack competition. The difficult task of reconfiguring the assumptions that simplistically connect hereditary knowledge and the human condition must be undertaken. Before I turn to address possibilities for such a reconfiguration, I believe one promising approach to this undertaking involves deploying concepts from developmental biology and the emerging field of developmental systems theory. To this issue I will return in chapter 4.

Chapter 2: Continuity: The gene-myth of alcoholism across the 20th century

“Having once discovered your drunkard by every means, moral or otherwise, endeavour to keep him sober, but use your knowledge to prevent him perpetuating his kind; prevent him marrying just as you would a person of weak intellect, or else render his marriage unfruitful.”
—Sir George Archdall Reid (1896, p. 765)

In the previous chapter I reviewed methods by which hereditary patterns in alcohol problems have been defined. I also noted the problem posed for such research by the heterogeneity of alcoholism as both a defined concept and a diagnostic category. Despite these shortcomings there are continuing efforts to secure patent rights to tests based on candidate “genes for alcoholism.” Here I turn to answer the questions “How did we get here? What are the sources of our current set of theories and problems?” In order to address these concerns, I focus first on the theoretical work of Sir George Archdall Reid and the foundation he laid for the future conceptualization of alcoholism as an hereditary disorder. Next I turn to the parallel representation of eugenic social policies and the characterization of alcoholism as a genetic problem in genetics textbooks during the first decades of the 20th century. It is during this time that the eugenics movement was living up to its aims, successfully passing sterilization legislation in much of the United States. Also during these years the science of genetics established itself as a discipline in its own right. By examining the presentation of eugenics within ostensibly scientific textbooks my aim is to show how deeply the undercurrent of the gene-myth runs in this era. The connection between our current fascination with genetic explanations of alcoholism can be traced to the subtle persistence of rhetoric and ideology established by these eugenic arguments early in the century that sought to establish biological explanations for all of society’s problems. Readers will note the historical leap this implies. In the interest of presenting new research and fresh

arguments (which I take to be the *point* of a dissertation) I will not rehearse the entirety of historical scholarship documenting the Temperance movement, its endorsement of calls for national Prohibition, the phenomenon of Native American drinking, or the development of the Alcoholics Anonymous organization and its consequences for modern treatment models. Instead of presenting these stories again, I will limit my focus to as yet untold aspects of the development of modern attitudes and scholarship on alcoholism. My aim is to document the entrenchment of the gene-myth with respect to alcoholism, and for this purpose the use of limited historical “snapshots” will suffice.

1. Popularizing Eugenics: Sir George Archdall Reid and the Medicalization of Social Problems 1890-1910.

In this case one source of the connection is an overlooked figure whose work found audiences in intellectual and scientific circles in Britain and the United States, writing texts on medical topics, heredity, and also the first booklength study of alcoholism as an exemplary case for the furtherance of 20th century hereditary theory. I argue here that Sir George Archdall Reid played a crucial bridging role between the eugenics and medical communities,⁴¹ and most importantly, did so using the topic of the hereditary nature of alcoholism. His work on alcoholism represents a significant step in the entrenchment of the gene-myth with respect to alcoholism that persists to this day.

This section will be organized around three main points. First, I discuss the life and work of this overlooked figure. Second, I explain why I think this writer was of more

⁴¹ His eugenic influence had purchase in both the United States and Britain, while his work on alcoholism seems to have been received in the United States more quietly than in the U.K. at the time of its publication.

significance for the eugenics movement than has been acknowledged previously. Finally, I will consider his impact on the development of a reductionist genetic model of alcoholism.

Sir George Archdall O'Brien Reid was born in Roorkee, India on April 7th 1860, only son to Capt. Charles Augustus Reid, of the 20th Bengal Native Infantry, part of the Honorable East India Company Service. Reid lived first in India, then in New Zealand, and settled finally in Southsea, Britain, a home-base from which he traveled to London or Edinburgh for lectures and presentations. The details of his early education are not known, aside from the fact that he was educated privately. He attended the University of Edinburgh and completed a Bachelor of Medicine and Master in Surgery at the age of 23.⁴² He married Florence Mahony in 1891 and remained married until her death in 1926. They traveled extensively in the Pacific and toured America. Prior to taking up a career in medicine, Reid held a variety of jobs including Schoolmaster, Kauri gum-digger, stockman, and hunter (Who Was Who 1929, p. 1132). Reid was elected a Fellow of the Royal Society of Edinburgh in 1902.⁴³ He helped found and edit the journal *Bedrock*, which began publication in 1912. He was even knighted in 1919 for his medical service during the First World War (Hamer 1929). His academic career is represented by a number of publications—five books addressing topics in evolution, disease, and modern psychology, as well as a number of articles.

Sir George Archdall Reid's role in the nebulous Eugenics movement spanned two important domains. As we will see, his publications reached both scientific and lay audiences,

⁴² He graduated August 1, 1887. Edinburgh University Records, Edinburgh, Scotland. These records also indicate he attended Crescent School, Margate for 5 years prior to enrolling at Edinburgh. I am grateful to Lawrence Dritsas for his help in locating these records.

⁴³ Obituary, *Proceedings of the Royal Society of Edinburgh*, v.50 p. 341. 1929-30.

thereby providing a crucial bridge between the new theoretical work in genetics and social problems.

Table 2.1 — Books by Sir George Archdall Reid, with initial data of publication.

<i>The Present Evolution of Man</i> (1896)
<i>Alcoholism, A Study in Heredity</i> (1901)
<i>The Principles of Heredity: With Some Applications</i> (1905)
<i>The Laws of Heredity</i> (1910)
<i>Prevention of Venereal Disease</i> (1920)

Reid's books (Table 2.1) display an interesting progression of subject matter. On the whole, he seems to move from the study of human disease in evolutionary context to judgments about the origins of modern mental, moral, and social characteristics. As I will show, there are two interesting features of this progression for the historian of alcohol and addiction studies that foreshadow some recent publications. The first is the argument for the "outbreeding of alcoholism"—a notion that resurfaced in the addiction studies literature as a "new discovery" as recently as 1989 (Carpenter & Ewing 1989).

Reid's first book *The Present Evolution of Man* was published in 1896, and was not subsequently reissued. In this text he parses the subject of man's evolution into two parts: Organic Evolution, and Mental Evolution. The first part is devoted to a discussion of the state of Darwinian evolutionary theory, and the question of Lamarckian "inheritance of acquired characteristics." Reid favors the selectionist interpretation of evolution (i.e., that natural selection is primarily responsible for evolution, rather than spontaneous or directed

mutation), and dispenses with the notion of inheritance of acquired characteristics for a variety of reasons. The second part of the book applies this selectionist lens to recent human evolution, in an effort to address the question whether there are significant inherited mental differences in humans. His answer to this question is odd, for in denying the inheritance of acquired characteristics, he concludes he cannot admit inheritance of mental characteristics. What is curious about this stance is the option with which he is left. Seeing mental characteristics largely as products of environment, he nevertheless carves out room for judgment on racial differences in intellect. Reid thinks there is a fundamental property of minds which is subject to evolution: *ability to acquire knowledge*. He claims that analogous to inherited resistance to indigenous disease, intellectual flexibility and openness to learning are a heritable factors. Finally, he extends his theory of inherited disease resistance to inherited resistance to, or tolerance for, *addictive substances*. Here he hints at a theory he develops in greater detail in his later writings, called “The outbreeding of Alcoholism.”⁴⁴ Reid summarized the theory this way:

Many races have been afflicted by alcohol for thousands of years. Some men are naturally more susceptible to the charm of alcohol than others. These, because they are more tempted, drink, on the whole, to greater excess, and thus are weeded out to a greater extent. As a consequence, every race is temperate precisely in proportion to its past experience of alcohol. Thus

⁴⁴ Reid’s use of the term “outbreeding” differs significantly from its familiar use in the context of animal breeding. For animal breeders “inbreeding” refers to the selection of mates from within a family pedigree, with varying coefficients of relationship between mates. In contrast, “outbreeding” refers to the selection of mates from outside of the family pedigree, or outside of the breed. Reid’s use of the term focussed on the presence of a character within a population, rather than mate selection from a family line. Treating alcoholism as one such character, Reid thought that if alcoholics did not mate, then the condition would be eradicated from the population. This same connotation of “outbreeding” appears in modern talk of natural selection and its effects on alcoholism.

west-African savages, who have possessed unlimited supplies of pal toddy, the Jews, and the inhabitants of the vine countries of the south of Europe are more temperate than the north-Europeans, and infinitely more temperate than most savages (Reid 1906b, p. 536).

The general idea is that many of the racial and individual differences in rates of addiction are a function of the length of time ancestors have been exposed to alcohol. Let's assume that some peoples indigenous to the Mediterranean coast have been producing and consuming wine far longer than peoples native to the British Isles. Over the course of time, Reid theorized, those who were susceptible to the addictive effects of alcohol would succumb to its influence, become "inebriate" and suffer from disease or death. As disease and early death are not traits conducive to a successful search for a mate, these individuals probably failed to bear offspring, and so their "weakness" was not passed along to future generations. This idea led one reviewer of *The Present Evolution of Man* to describe Reid's motto as "let the drunkard drink and perish, and his seed with him..." (Lankester 1896, p. 413).

A review of *The Present Evolution of Man* in *Science* sparked an exchange between Reid and the reviewer, T.D. A. Cockerell. Cockerell gained his notoriety in scientific circles for his voluminous work on bees, cataloging thousands of new species and subspecies from his post at the University of Colorado at Boulder. While Cockerell spoke highly of the first section of Reid's book, noting that "in the earlier part of the book there is given a very excellent discussion of the broad principles of evolution" (Cockerell 1897, p. 33), he took issue with several other aspects, particularly Reid's presentation of retrogression, or atavism--the emergence of ancestral characteristics in a species. He also voiced disagreement with Reid's assessment of the role of heredity vis-a-vis sanitation and "social efficiency" in

shaping different human population's development of resistance to disease. Cockerell felt that a society's ability to maintain sanitary living environments through technology, infrastructure, or simply social custom, would affect the rate of disease stronger than any inherited factors. Reid's suggestion piqued interest among his colleagues, and seemed to accord with epidemiological data for population differences in rates of disease. However, he received criticism over the degree to which he claimed inherited factors could determine immunity to certain diseases, toxins, and harsh environments.

Cockerell's final object of criticism was the last portion of Reid's book that presented his selectionist view of alcoholism and narcotic use. Cockerell wrote "It is assumed that a desire for alcohol is inherent in the human race, and that, since the substance cannot be banished, our only salvation is to gradually acquire a toleration of it, as of a zymotic disease... An obvious difficulty here is to explain how, under the influence of natural selection, this highly pernicious craving for alcohol arose." Cockerell is clearly not satisfied with Reid's supposition that the preference for alcohol and narcotics is a natural byproduct of human mental evolution. In a reply to Cockerell, Reid (1897) dwells almost exclusively on the issue of retrogression and atavistic characteristics, insisting that his theory of retrogression applies to cases of natural, not artificial selection: "wholesale reversion to the ancestral type must be extremely rare in the case of all species slowly evolved under the ordinary conditions of nature" (p. 368). Reid believes the core of the disagreement has only to do with the degree of retrogression a species might undergo in the complete absence of selective forces: Cockerell endorsing small amounts of retrogressive changes, Reid endorsing "absolute and unlimited retrogression" (p. 369), and therefore a disagreement of degree rather than of kind.

Having consumed over half of the space of his reply on the matter of retrogression and atavism, Reid turns to the more serious challenges of Cockerell's review. Reid likens

Cockerell's comments to those he has received (privately) from Alfred Russell Wallace, taking aim at Reid's supposition that the craving for alcohol has arisen naturally in evolution. In answer, Reid offers three "significant fact[s]". First he claims that "old records seem to prove that the classic races were anciently much more intemperate than at the present time. Second, he points to the example of narcotic opium and its long history of use in India and China compared to "Burmah" where "it has been recently introduced" (p. 372). The comparative intemperance of the Burmese offers support for his view, he claims. Third, Reid deploys the case of tobacco to support his view. He claims that "tobacco causes little or no elimination" in selective terms, and therefore the craving for it is equivalent across populations, irrespective of their length of exposure to it. These three examples seem to do little more than sidestep the brunt of Cockerell's criticism, offering no reasonable explanation how the initial craving for alcohol could have become established.

Cockerell reiterated his misgivings in a reply the following month (Cockerell 1897b), in which he shifted somewhat his stance on Reid's theory of alcohol as a selective force: "I do not deny that the general use of alcohol will lead to a process of evolution against it, but I do deny the desirability of any race undergoing such a process" (p. 563). Hence, Cockerell's reply removes the issue of alcohol and heredity from an historical context and resituates it in a normative context. Cockerell's main worry is now whether any nation should, as Reid suggests, revise their prescriptions for temperance and allow the forces of natural selection to weed the drunkards from among them. But Cockerell introduces a new wrinkle to the issue by equating craving for alcohol with a more general temperament in which men seek fulfillment of their desires. Assuming this equivalency, he underscores the potential homogenizing influence that any eugenic program aimed at alcoholics might entail: "As missionaries will say, give us a man who strongly believes something, however demoniacal,

and we can do something with him; but give us a man with no beliefs and we are almost helpless” (p. 563). With this remark the public debate between Cockerell and Reid comes to a close.

The topic of alcoholism was taken up more fully in Reid’s second book, significant also for the history of addiction research as it is the first English-language monograph to deal exclusively with the theory of the *hereditary origins of alcoholism*. First published by T.F. Unwin of London, this volume was reissued in 1902 by Bailiere, Tindal & Cox publishers of London, and by Wood publishers of New York. This text followed from his earlier work, with added consideration of a variety of data on the use and abuse of alcohol in various societal contexts across the globe and throughout history. For example, key chapters bear titles like “The Natural History of Man,” “Alcoholic Selection,” “Racial Differences” and “The Temperance Failure.”

Contrary to many temperance advocates and hereditary specialists alike, Reid maintained his selectionist view of the role of heredity in alcoholism. The majority of temperance advocates of the day held that alcohol was a *poison*, and was solely to blame for its toxic and addictive effects. Although some attempted to profit from cures for inebriety (e.g. “temperance pills” or regular Turkish baths), for many temperance advocates *abstinence* was the only cure, and national prohibition a proposal for achieving it on a large scale (Longmate 1968, pp. 200-2002). Meanwhile, for many eugenicists, alcoholism or inebriety served as a central example of degeneration of the national stock, whereby increasing numbers of social problems could surface with every generation of offspring of the drunkard. In the context of these two camps, Reid’s views clearly place him with the eugenicists rather than the temperance advocates. Reid believed the only solution for alcohol problems was to allow natural selection to cull from the nation those persons who were

most susceptible to the negative effects of alcohol. This put him at odds with the temperance advocates, because to cut off the supply of alcohol would only allow the spread of the inebriates “weakness.”

Reid’s next book *The Principles of Heredity* (1905) sold as a textbook on evolutionary theory and genetics. It was published in both the United States (by E.P. Dutton and Co.) and Britain (by Chapman and Hall) and it sold so well that a second printing was issued the following year, with a third printing in 1911 (by Methuen publishers). In this volume, Reid aimed to reach an audience of physicians.⁴⁵ Reid thus appealed to an elite group of professionals and pre-professionals in the medical community and university education. He even held a post at the University of Edinburgh from 1914 to 1916 as a lecturer specifically to their medical students. These facts may seem benign, until we note the eugenic tone of Reid’s remarks on the hereditary aspect of human social problems and diseases. Speaking specifically on the disease of “consumption,” Reid remarks:

As generations pass, as the numbers of the unfit increase through lessened elimination, as the race regresses, the task of the sanitary reformer will grow in magnitude and complexity.... Apparently, therefore, our only hope of permanently lessening the prevalence of the disease lies in a reduction of the number of people susceptible to it. In other words the problem presented by consumption is one which ultimately will have to be solved—if ever it be solved—by the student of heredity. Some method will have to be devised to

⁴⁵ In his own words (as noted earlier): “I have addressed the volume mainly to medical men. The evidence relied on is drawn largely from medical sources; medical men are the largest body of scientific workers; they deal constantly with questions of Heredity, a knowledge of which is of great importance to them; but in measure they have neglected the systematic study of the subject. Little or no direct instruction in it is given to medical students. There

lower the output of children by people predisposed to the complaint, otherwise the mortality from consumption cannot be greatly or permanently reduced. A few States of the American Union have already laws forbidding the marriage of consumptives. It is, however, no part of my present purpose to suggest remedies for the mere discussion of which the community is not as yet prepared. My principle object is to demonstrate that there are certain practical problems of great importance which cannot be dealt with until the public, and especially the medical profession, are in a position to bring an adequate knowledge of heredity to bear on them. The mere existence of such knowledge would be of incalculable benefit, inasmuch as the race would become alive to the dangers which menace it, and improper marriages, meeting, as they should, with universal censure, would become less frequent (pp. 337-8).

As we can see from these remarks, Reid alludes to the eugenic sterilization programs that were beginning to operate in the United States shortly after the turn of the century. This reference to the power of heredity to guide future public health policy, coupled with the medically-minded target audience is what situates Reid as a specialized promoter of eugenic ideals.⁴⁶

does not exist even a text-book to which they may refer. But a knowledge of Heredity is becoming essential to the educated doctor. I have sought to supply the want" (1905, p. viii).

⁴⁶ Also noteworthy is a remark capturing the logic of the eugenic argument quite nicely: "We reach thus two fundamental biological laws. The first law is that the germ-plasm is very highly indifferent to the action of the environment, and therefore that children are seldom affected by the influences to which their parents are exposed. The second law is that germ cells, and therefore the individuals that arise from them, vary spontaneously among themselves, just as the body-cells vary, and for the same reason. It follows that we cannot improve races of plants and animals by improving the conditions under which they exist. Such a course benefits the individual, but results in racial degeneration. The race can be

In a review in the weekly *Science*, William A. Locy offered a brief, somewhat mixed review of *The Principles of Heredity* (Locy 1907).⁴⁷ Although he praised Reid as “a medical man of high scientific attainments” offering a volume aimed at the study of heredity through examples afforded by disease phenomena, he saw Reid’s omission of recent work in cytology as a serious drawback. And although he thought the book would “assist materially in getting medical men to pay more attention to the matters discussed” (p. 62), his reservations about the level of the discussion were severe: “this book, although introducing much new matter, does not appear to rise to the level of current standards in the serious discussion of the principles of heredity” (p. 61). Locy’s central point of criticism had to do with Reid’s style of writing, which involves sentences of considerable length, and his argumentative tone. While such issues of style might serve to warn the laboratory experimenter away from the book, it is not clear that they diminished the book’s employment as an instructional textbook, as it was reprinted subsequent to this review in 1910.

Reid’s third book *The Laws of Heredity* saw initial publication in 1910 in both London (Methuen & Co.) and New York (MacMillan), and saw a second printing in London the following year. In a review essay titled “Heredity and Environment in Regard to Social Reform,” Reid’s volume received favorable mention from British mental deficiency expert A. F. Tredgold. His review approached the topic of social reform by touching on a total of six prominent texts, putting Reid in the company of notable scientists and writers like J.A. Thomson, Hastings Gilford, Arthur Dendy, Franz Boas, and Marcus Hartog. Tredgold situated the views of Archdall Reid opposite those of August Weismann on the mutability of

improved only by restricting parentage to the finest individuals. All the practice, if not the theory, of breeders confirms us in this belief” (Reid 1906b).

the germplasm, and cites Reid's work also as having established that the immunity of a population "to a morbid process is directly proportionate to its previous experience of that process" (Tredgold 1913, pp. 370-1). Thus, Reid's theory on the "outbreeding" of alcohol was known in wider circles other than those focussed on the temperance issue or hereditary science exclusively. Tredgold himself was an expert on the topic of feeble-mindedness whose published findings lent explicit support to eugenic social policies. In his own studies he claimed to have found that "between 80 and 90 percent" of cases of mental deficiency were "the result of a morbid inheritance, the remaining small proportion being caused by accident or some form of acquired cerebral lesion" (Tredgold 1913, p. 372). How exactly mental deficiency was transmitted through the germplasm Tredgold failed to articulate;⁴⁸ but this did not diminish his insistence on the importance of eugenic social policies for its future eradication. As he put it, "The nation must breed from its best; at all events, it must take especial care to prevent the propagation of those who are so inherently defective that their regeneration by the environment is out of the question" (p. 382).

⁴⁷ Locy, a zoologist, would later publish his own books on biological topics aimed at a popular audience. These included titles such as *Biology and Its Makers* (1915), *Main Currents of Zoology* (1918), *The Story of Biology* (1925), and *The Growth of Biology* (1925).

⁴⁸ In his widely used textbook *Mental Deficiency*, however, Tredgold went to great lengths to both document the existence of mental defects and argue for their hereditary origin. He held that mental deficiency offered evidence of the hereditary transmission of a "neuropathic diathesis," a constitutional state, rather than an individual genetic element. The success of his textbook was considerable--it was published in the United States in its second edition (1914), and reissued in 10 successive editions, the last of which appeared in 1967 (Source: WorldCat).

Table 2.2—Articles published by Sir George Archdall Reid.

Reid, G. Archdall. 1896a. "Reflex Action, Instinct, and Reason." <i>The Fortnightly Review</i> 65: 269-77.
Reid, G. Archdall. 1896b. "The Present Evolution of Man': A Reply." <i>The Fortnightly Review</i> 66: 755-768.
Reid, G. Archdall. 1897. "Characters, Congenital and Acquired." <i>Science</i> . Dec. 17th & 24th.
Reid, G. Archdall. 1903. "Human Evolution: With Especial Reference To Alcohol." <i>The British Medical Journal</i> . 818-820.
Reid, G. Archdall. 1905a. "Theory of Natural Retrogression." <i>Natural Science</i> 13: 396.
Reid, G. Archdall. 1905b. "The Causation of Variations." <i>Nature</i> 72: 318-319.
Reid, G. Archdale. 1906a. "Heredity and Social Problems." <i>The Independent</i> 60: 379-383.
Reid, G. Archdall. 1906b. "The Biological Foundations of Sociology." <i>American Journal of Sociology</i> 11(4): 532-554.
Reid, G. Archdall. 1912. "Recent Researches in Alcoholism." <i>Bedrock</i> . 1: 45.

From a glance at the venues in which Reid published his articles, and the topics he addressed, we can see that he connected certain aspects of the eugenics movement with research on his favorite case study, alcoholism and heredity. By writing in popular magazines like *The Independent*, *Fortnightly Review*, and *Bedrock*, he broadcast to a wide lay audience his vision of the importance of selection for modern human diseases and addictions. Simultaneously, publishing in journals like *Nature*, *Science*, *The British Medical Journal*, *The American Journal of Sociology*, and *Natural Science* he tested his views before specialized scientific audiences.

Aside from his 1901 book *Alcoholism: A Study in Heredity*, Reid offered a more detailed articulation of his views on alcohol and heredity in a review article titled "Recent

Researches in Alcoholism” published in the journal of which he was co-editor, *Bedrock* (Reid 1912). Reid begins this review with a lengthy (although not terribly illuminating) consideration of the abstract relations among interpretations, facts, causes, explanations, empirical laws—with the apparent aim of comparing the status of these in Kepler’s work versus Darwin’s. From this comparison he reaches the conclusion that biological phenomena represent a distinct and special category of physical phenomena, and that the “group of sciences which are included under the term biology offer a marked contrast to all other sciences” (Reid 1912, p. 28).

Articulating his evolutionary perspective on human traits, Reid offers three hypotheses for the role of natural selection in shaping modern alcohol problems. The first hypothesis holds that “the diverse behaviour of men indicates variations in their susceptibility to the charm of alcohol, and that those who are most susceptible tend to be eliminated.” Continuing, Reid provides two alternative hypotheses: “(1) that experience of alcohol causes racial degeneration; and (2) that it has no racial effect at all” (Reid 1912, p. 41). Given the content of his previous writings, it is clear that he intends to endorse the first hypothesis and discount the alternatives. As a way of testing these hypotheses, Reid launches into a lengthy arm-chair anthropological consideration of the differences between the “races” considering also differences among them in climate, potency of national beverages, environment, civilization and education. Weighing the difference among these elements against the current role of alcohol in cultures across the globe, Reid builds support for his evolutionary hypothesis on alcoholism. The glaring shortcoming of this approach lies in his attempt to infer biological facts about these races from the basis of his largely unsubstantiated anthropological overview. For example, in discussing the role of education in shaping racial differences in alcohol use, he remarks

Country folk are usually more temperate than urban populations, and Mahomedans than peoples of the same race but of another religion. But, granting all this, it is true nevertheless that indulgence in alcohol produces in people who are susceptible to its charm particular feelings, sets of sensations and emotions which to them are very delightful. Education cannot alter sensations (Reid 1912, pp. 43-4).

Moving so swiftly from anthropological generalizations to conclusions about the sensory and emotional character of vaguely defined groups of people betrays the degree to which Reid had committed his intellectual energies to his evolutionary perspective on alcoholism.

Correspondence and Controversy

In 1896 the *Fortnightly Review* carried an exchange between Reid and Edwin Ray Lankester, Professor of Comparative Anatomy at Oxford. The exchange began with Professor Lankester's review of Reid's first book, *The Present Evolution of Man*, in which Professor Lankester offered a mixed opinion (Lankester 1896a; Lankester reported that his review had been produced at the request of the journal's editor). While he noted that Reid was developing an intriguing and somewhat original theory on the role of hereditary selection in the development of modern human diseases, he noted what he took to be several errors of fact, and expressed reservations over Reid's failure to consider the phenomenon of "correlation of variation" in this context. Correlation of variation is a concept first articulated by Charles Darwin, which refers to the problem of distinguishing between sets of traits that are acted on by selective forces in the evolution of an organism, versus those sets of traits that are linked to these "acted on" traits by some developmental process. In the

case of Reid's selectionist views of human disease, the force of the objection is that Reid may have considered some traits to be driven by selection (e.g., human resistance to various diseases) when they were instead correlated results of selection acting on other features of the organism.

Faced with this objection, Reid used the opportunity to restate his views and guide readers to his book for further clarification.⁴⁹ On four separate occasions he restates the core element of his outbreeding thesis and offers examples culled from observations of differences in drinking behavior in different countries. Using these comments as a springboard, he lauds the importance of his theory for the future of human affairs on an international scale. Reid also struck back against Professor Lankester on a number of occasions, offering in conclusion "I think, therefore, I am justified in slightly altering one of [Lankester's] own sentences, and saying that he shows plainly enough that he is himself, in spite of his learning, not a very acute student of the matters of which he treats" (Reid 1896b, p. 768). Reid's comments betray his motive: having attracted the attention of a prominent reviewer, he sought to engage in an extended exchange in order to both broadcast awareness of his book and to bolster his stature with the educated lay audience of the journal. Professor Lankester replied in the next issue of the journal, and pointed out these motives, writing "...he is apparently anxious to engage in a controversy with me on some special points of zoological observation and doctrine in the pages of this Review" (Lankester 1896b, p. 914).

⁴⁹ Reid's summary of the outbreeding thesis went as follows: "In countries where [alcohol and opium] are abundant and easily accessible to the mass of the population, they weed out, with more or less certainty and speed, during generation after generation, those that crave most for deep intoxication, leaving the race to the descendants of those that crave least for it. And therefore, generation after generation, a race cursed by the prevalent use of any of them grows less and less mentally susceptible, till, at length, the great majority of its members crave so little for excessive indulgence or crave for so mild a degree of indulgence, that they are no longer prejudicially affected in the struggle for existence" (Reid 1896b).

After attempting to address some of Reid's concerns, Lankester concludes "Adequate discussion of so wide a range of speculations is impossible under the conditions offered by the pages of this Review, and, for my own part, I shall not here write anything further in reference to Mr. Reid's performances" (p. 916). In retrospect, Reid's ploy for the attention of the educated lay readership of the *Fortnightly Review* appears to have been relatively successful.

Aside from this published exchange, there is at least one other significant dialogue in which Archdall Reid's views on heredity were aired alongside those of other prominent figures of the era. *The American Journal of Sociology* in 1904 printed a paper presented by Francis Galton to the Sociological Society at a meeting in London University's School of Economics on May 16th of the same year (Galton 1904). Galton, the father of eugenics, read his essay "Eugenics: Its definition, scope and aims" before an audience and invited comments, all of which were published along with the paper. Galton's short essay articulated five areas of attention for the growth of eugenics as both a science and a sound social policy. While the first four of these topics relate to areas of study, the fifth point takes a more political tone. He maintained that eugenics must be rendered familiar to a wide and intelligent audience, and therefore "must be introduced into the national consciousness like a new religion" (Galton 1904, p. 5).

Responding to the paper were a number of key figures in social and political circles (G. Bernard Shaw and H.G. Wells), as well as reputable doctors and genetic scientists (Karl Pearson and William Bateson). Below is a full list of respondents, in order of successive comments:

Karl Pearson
Dr. Maudsley
Dr. Mercier
Prof. Weldon

H.G. Wells
Robert Hutchison
Dr. Warner
Mr. Elderton
Benjamin Kidd
Mrs. Dr. Drysdale Vickery
Lady Welby
Mr. Hobhouse
G. A. Archdall Reid, M.D.
W. Leslie MacKenzie, M.A., M.D.
G. Bernard Shaw
John M. Robertson
W. Bateson, M.A., F.R.S.
C.S. Loch, B.A.⁵⁰

Pearson and Bateson both offered praise for the Galton's views. Voices of dissent were of two stripes: on the one hand, Dr. Maudsley, Dr. Mercier, and Mr. Hobhouse urged caution in delving into eugenics due to the complexity of human minds and morals and the limited state of knowledge of hereditary influences in this regard; on the other hand H.G. Wells, Dr. Warner, and Benjamin Kidd challenged the primacy of hereditary forces over the more obvious influence of environmental factors. Said H.G. Wells "I have been impressed by the idea...that our analysis of human faculties is entirely inadequate for the purpose of tracing hereditary influence. I think we want a much more elaborate analysis to give us the elements of heredity..." (p. 10).

Reid took the opportunity to reiterate his views on the role of heredity in shaping social problems of all sorts. Putting it rather strongly, he writes "At the root of every moral and social question lies the problem of heredity" (p. 16). Reid next turns to the question of the proper audience for eugenic teaching, arguing that members of the medical profession are best situated to both gather evidence and promote eugenic ideals. He offers in evidence

⁵⁰ See "Discussion," *The American Journal of Sociology*, 1904, vol. 10, pp. 6-25.

of the truth of Galton's eugenic teachings a reference to his work on the "outbreeding" hypothesis of disease. Using the examples of malaria and consumption in colonial contexts, he reiterates "it may be taken as a general rule, to which there is no exception, that every race throughout the world is resistant to every disease precisely in proportion to its past experience of it, and that only those races are capable of civilization which are resistant to the diseases of dense populations" (p. 18). Finally, Reid endorses two policies designed to "raise the standard of our race." The first is the requirement that society "improve the conditions under which the individual develops"--hence, enriching the human environment. Second, Reid embraces the policy of negative eugenics, stating "we must endeavor to restrict, as much as possible, the marriage of the physically and mentally unfit" (p. 19). Appearing in a published discussion including Francis Galton and other eminent figures of science and popular thought of the era offered Reid a rich opportunity to convey his selectionist view of human disease and social ills to a wide and intelligent audience.

Information on Reid's private correspondence is unfortunately not as forthcoming. Reid is listed in the United Kingdom National Register of Archives only twice, first in a correspondence held with Charles C. Hurst over three years from 1905 to 1908 and second with birth control reformer Marie Stopes toward the end of his life. Given Hurst's interests in botany and evolution, their discussions probably centered on general theories of evolutionary mechanisms, rather than human heredity or alcoholism. Mary Stopes principally wrote on topics of birth control, marriage, venereal disease. As Reid's publications around the time of this correspondence centered on venereal disease in evolutionary context, there is reason enough to think that alcoholism was not among the topics they debated. Unfortunately, at the time of this writing I have not been able to locate a private collection of his correspondence.

Professional Practice

Reid lived in London and Southsea, with only one University teaching affiliation at the University of Edinburgh during the years 1914 and 1915. Serving primarily as a physician and writer on the biological foundations of modern social problems, he did not leave a legacy of archives, manuscripts, or memoirs for historical study, and not much is known about the details of his practice.⁵¹ It would be interesting to know, for instance, whether he attempted to treat patients with alcohol problems, and what method of treatment he may have developed. Lacking solid documentation, I will avoid speculation on the details of his practice.

Reid died in 1929 at the age of 69. In an obituary in the *Times*, one friend recalled “As he used to say to me, ‘While others have been gazing at the far horizon, I have observed the obvious things lying just near my feet’” (Hamer 1929). Perhaps he was looking too close to his feet, perhaps at the drunkards in the park, or the sick and fallen. As one obituary stated it “he had not Darwin’s broad view of natural selection, and in all his writings he appeared to recognize only disease selection in mankind.”

Reid’s Contribution

By appealing to a wide audiences including technical, scientific and educated lay public, Reid broadcast the doctrine of eugenics and further entrenched the medicalization of social problems. Addressing issues like alcoholism, venereal disease, mental traits and

⁵¹ Although his Edinburgh University records indicate that he completed a three month practical in Midwifery, as well as two residencies between November 1883 to November 1887. Records of his final examinations indicate that he also qualified specifically to perform vaccinations.

morals from the perspective of evolutionary theory, he brought legitimacy to the scientific study of social problems in Britain and the United States. He aired his views on the hereditary aspects of alcoholism and narcotic use in contexts like the Sociological Society, where luminaries and future cultural leaders included Francis Galton, G. Bernard Shaw, and H.G. Wells. Also, by singling out medical students as an audience for his work, he enrolled a powerful group of future professionals perfectly poised to act on eugenic guidelines and principles. His books were distributed widely,⁵² and no doubt influenced the hereditary and eugenic outlook of future physicians.

Of course, there are other prominent members of the eugenics movement who used their medical and scientific positions of authority as pulpits to preach eugenic doctrines. The list of notable figures includes names like Francis Galton, Erasmus Darwin, David Jordan, Karl Pearson, Charles Davenport, Edward East, William Castle, etc. There are a number of features that set Reid aside from such figures, particularly for the burgeoning view of alcoholism as a hereditary phenomenon. First, as a physician working directly with patients, he was not concerned with model organisms but with real human medical and social problems. His day-to-day concerns focussed on human problems rather than laboratory-based experiments. Yet he rubbed shoulders and published exchanges with figures at the forefront of biology and evolutionary theory. This may have lent him greater authority with lay audiences, for whom human tales were more relevant than experiments on rats or mice.

⁵² Records from the publishing houses Reid used are not currently available. However, a recent search of WorldCat electronic database of library catalogs shows 100 copies of *Laws of Heredity* and 84 copies of *Principles of Heredity* remaining in circulation in the United States. This pales in comparison with Punnett's *Mendelism*, of which 608 copies (from all 6 editions) remain housed in libraries. However, both of Reid's textbook efforts went through only 2 editions, so a balanced comparison is difficult to make. As for copies currently in private hands, a similar search on abebooks.com (a database of used bookstore holdings) returned 19 copies of Reid's texts currently for sale.

Second, Reid had a limited role as an organizer in the eugenics movement. Reid took no obvious steps to publicly recruit new members. Instead, he primarily used his publications and textbooks to convey eugenic principles to the educated public. This may have enhanced his stature in the public mind—for he appeared only as a legitimate man of letters, writing scientific textbooks and articles for intellectual periodicals, rather than solely editorial opinions lacking the weight of scientific backing.

Finally, Reid straddled the gap between the study of heredity and etiology of social and behavioral problems, something that few of these other figures managed. By arguing for the relevance of evolutionary theory for both medicine and psychology, Reid provided a seamless solution to the presumed gap between the scientific and manifest images of social problems. Biology and evolutionary theory could be counted on to solve troubles in domains of both infectious disease and social deviance. In his own words:

Hardly a social, moral, or intellectual question can be thought of but we find that in its deeper aspects it is a problem of heredity. Heredity concerns not only the philosopher and the man of science, but also the parent, the teacher, the doctor, and even the statesman, the social reformer, and the historian
(Reid 1906a, p. 532).

In light of this review of his activities during the late 1890s and early 1900s, I believe Reid's contributions to the medicalization of alcohol and other social problems exceeds the proportion of scholarship that notes his impact in this arena.

2. Connections between the Eugenics Movement and the Origins of Hereditary Research on Alcoholism.

Eugenics refers to the notion that animal breeding principles can be applied to humans to enhance the overall well-being of a population. In 1883 Francis Galton coined the term *eugenics*, literally meaning good breeding. The proposed methods for achieving better human stock are commonly parsed into two categories: *positive* eugenics—encouraging acceptable individuals to procreate, and *negative* eugenics—denying reproductive rights to unacceptable individuals using methods ranging anywhere from social stigma to compulsory surgical sterilization. On first learning about the eugenics movement in the United States, many are shocked that such actions could have taken place here, in this century of such presumed scientific, medical, and social “progress.” But the fact remains, the eugenics movement was neither small, nor isolated. At one point 24 states had passed sterilization legislation, and the principle aims and methods were endorsed by politicians, professionals, and even supreme court justices.

The eugenics movement has received considerable attention in a variety of areas of scholarship within the fields of history of biology and medicine. Despite this fact, the *origins* of this movement remain difficult to pin down. While it is safe to say that the core ideas of the movement entered popular circulation during the last quarter of the nineteenth century in the United States, and somewhat earlier in Britain and France, precise analyses of the social and political dynamics responsible for the movement’s growth remain tentative and obscure. The most interesting period of growth in the American scene is roughly 1900 to

1930, although the movements' effects on reproductive rights clearly continued as late as the mid 1970s in some states.⁵³

What can we learn from twentieth-century textbooks on genetics?

The aim of this section is not to review in detail the history of the eugenics movement in the United States (a task beyond the scope of the current project). Rather I present an examination of one means of disseminating eugenic propaganda that has not received attention by researchers to date—college textbooks on genetics and heredity. Such an examination serves to underscore the central point of this chapter: that the history of both intellectual and common folk-knowledge of heredity in relation to alcoholism is deeper and crucially more relevant to the conceptualization of modern alcoholism than most current alcohol-related literature would suggest. For at the core of eugenic thinking is the notion that social problems can be both understood and potentially alleviated by placing them under the lens of hereditary science. To be sure, modern research on alcohol problems does not endorse forced sterilization for those thought to be predisposed to alcoholism by virtue of heredity. However, the fact that the core idea of genetic reduction of human social problems has persisted through a century of textbooks presentations of genetic science underscores the importance of probing as deeply into historical context as possible when seeking to explain the current state of affairs in alcoholism research.⁵⁴

⁵³ The last surgical sterilizations performed at the state-run clinic “The Lynchburg Colony” (Lynchburg, VA) occurred in 1972 (*The Lynchburg Story* 1994). Between 1927 and 1972 over 8,000 sterilizations were performed at this clinic.

⁵⁴ As I show below, the first serious work on alcoholism and heredity emerges within the context of early textbooks on hereditary principles and eugenic propaganda.

So far the growth of the movement in the Anglo-American cultural landscape has been attributed in part to an ideological climate favorable to biological metaphors for society and progress, on the one hand,⁵⁵ and to the legitimacy conferred by backing from much of the scientific and progressive intellectual community, on the other hand. Daniel Kevles' (1985) highly regarded text, *In The Name of Eugenics*, attributes the movement's rapid growth in part to a number of its promotional techniques. Prominent scientists delivered public lectures at civic clubs and reading groups, clergy members sermonized on the topic of healthy progeny and the fate of the race, state fairs displayed exhibits on the subject, proponents staged "fittest family" contests at fairs and expositions, distributed pamphlets, and published many popular books discussing the topic (pp. 60ff). All of these techniques of promotion employed socially legitimate forums and figures as channels for broadcasting to the public the movements' methods and aims. With the rediscovery of Mendel's analysis of patterns of hereditary transmission of characters in 1900, what was for decades only a vague metaphor for progress acquired scientific legitimacy and grew, achieving credibility among lay public, intellectuals, policy makers, and prominent public figures across the political landscape.

As Lisa Pine (1996) shows in a study of the spread of Nazi ideology and education, textbooks occupy a particularly crucial position for impressing political and social ideas on nascent members of educated classes. Similarly, Steven Selden shows how high school biology textbooks can endorse the application of biological metaphors to public policy issues (Selden 1985). In the case of university and medical school contexts, the science textbook holds considerable ideological potential as a result of the nature of the readership these texts reach. Throughout the nineteenth and twentieth centuries, one consequence of the industrial

⁵⁵ Due in large part to the influential work of Herbert Spencer.

revolution was that individuals with training in the physical sciences became valuable consultants for (and in many cases active designers of) industrial, public health, and social policy concerns. For some, the college degree amounted to an entry ticket to the professional and managerial classes. In this state of affairs the policy content of collegiate scientific coursework could influence the tenor of discussion over public policy, eventually influencing the outcome of state social policy decisions (Allen 1974, p. 36ff; Allen 1975). College textbooks deserve attention as instruments of social change, particularly when they explicitly endorse social policies in the context of otherwise (presumably “value-free”) scientific instruction.⁵⁶

In the following section, I survey a sample of early 20th century textbooks in heredity and genetics and then turn to disturbingly similar social-policy rhetoric that appears in recent (1990s) genetics textbooks. In the texts from the early 20th century it is interesting to note the frequency with which alcoholism is included among the list of problems for which eugenic solutions are proposed. While no such remarks appear in the texts from the 1990s, they do contain vague and potentially misleading statements on the value of genetics for social and health problems. Given the role college genetics textbooks played as propaganda instruments of the eugenics movement, these recent associations of genetic science with social policy should give us pause.

3. Eugenics and Education: the Textbook

By reviewing a few central points in the history of the eugenics I will illuminate the link between education and intellectual scholarship on the one hand, and the success of this

⁵⁶ On the question of the role of values in modern scientific education, see e.g., Burkhardt (1999). Burkhardt reexamines the assumption that scientific and technical instruction is void of value judgements.

social movement on the other hand.⁵⁷ Eugenic ideas found their most popular and direct introduction in the work of Francis Galton as early as 1865. He went on to coin the word “eugenics” in 1883 in *Inquiries into Human Faculty*. The movement gained institutional support when in 1904 when Charles B. Davenport established the Laboratory for Experimental Evolution at Cold Spring Harbor (with funding through the Carnegie Foundation). At this and the Eugenics Record Office (established in 1907 and also directed by Davenport), many early workers in genetics also sought to establish eugenic policies throughout the United States. These policies were realized first in 1907 in Indiana, where the country’s first sterilization legislation appeared, recommending surgical sterilization of criminals, the feeble-minded, prostitutes, alcoholics, and other undesirables (Kevles 1985, p. 99ff). Laws also recommended restrictions on marriage by “habitual drunkards” the “mentally deficient” and those with transmissible diseases. By the mid 1920s such legislation had spread through at least 24 states, and even affected federal immigration policy (through the Johnson Act, which limited Eastern European immigration).

Before the movement began to grow, it needed a way to spread its teachings and beliefs to potential initiates. College textbooks on genetics served this purpose remarkably well. Two considerations support this view: First, eugenicists were quite vocal about the value to the movement of texts that appealed to a broad lay audience. Textbooks for college courses clearly reached a large and socially mobile audience of middle and upper-middle class students. Second, there are as many as 30 texts that were promoted for use in introductory courses on biology, genetics, or eugenics. Prior to the rediscovery of Mendel’s work, heredity and genetics were not an integral part of the curricula of universities and medical

⁵⁷ Paul (1998), Ludmerer (1972) and Kevles (1985) offer extensive historical and political accounts of the eugenics movement.

schools. The first decades of the 20th century saw steep growth in the number of such courses, as well as texts written for them (Cravens 1978; Herndon 1956; Selden 1978). At the time these texts were being written, many eugenicists pined for the incorporation of their teachings into college curricula, thinking this was the best way to insure the success of the movement. George Archdall Reid advocated including eugenic doctrines in college curricula. His landmark medical textbook on heredity in 1905 argued for the inclusion of eugenic doctrines in medical college curricula, in addition to standard undergraduate curricula. In the introduction to his text he describes the promise that physicians hold for the movement.

I have addressed the volume mainly to medical men. The evidence relied on is drawn largely from medical sources; medical men are the largest body of scientific workers; they deal constantly with questions of Heredity, a knowledge of which is of great importance to them; but in measure they have neglected the systematic study of the subject. Little or no direct instruction in it is given to medical students. There does not exist even a text-book to which they may refer. But a knowledge of Heredity is becoming essential to the educated doctor. I have sought to supply the want (Reid 1905, p. viii).

By targeting the so-called “medical men” it is clear that he meant to target those in positions of authority, those who had the means and opportunity to weigh in on matters of state health policy, and possibly influence public opinion.⁵⁸ In 1912, at the First

⁵⁸ To be sure, the authority of “medical men” in the early decades of the 20th century was not organized into professional societies, and did not have the influence it does today. However, the fact that physicians in this era were relatively unspecialized meant that they applied their skills and opinions in a wider variety of roles than at present. Frequently blurring health and social policy boundaries, coupled with a history of political aspirations, contributed to the view of physicians as experts on social policy (Starr 1982, p. 82-3). Also, the “educated were few and physicians a relatively high proportion of them. Since medicine was much less

International Eugenics Congress H. E. Jordan⁵⁹ argued for the incorporation of eugenic doctrine into the curriculum of medical schools. He claimed there was both a need for such incorporation, and that its effects would greatly shape the future of the eugenics movement. He wrote:

The coming physician must have adequate training in matters relating to heredity and eugenics. And the medical curriculum that includes these subjects (properly combined as one) and provides for their clear scientific presentation is, other things being equal, the one which best meets the needs of the very near future (Jordan 1921, p. 396).

On the subject of the impact such measures will have on the growth of the eugenics movements, his comments are similarly positive and direct.

[D]ue to their peculiar position of influence and respect, if properly advised about eugenics, *physicians* could be the most potent factors in spreading, and giving proper direction to, the eugenic propaganda (p. 397, emphasis added).

renumerative, the incentive to become politically engaged was relatively greater” than at present (Starr 1982, p. 83).

⁵⁹ H. E. Jordan is introduced at the Congress as “Chairman of the Eugenics Section of the American Association for the Study and Prevention of Infant Mortality.” See Jordan (1921, p. 396). This First International Congress of Eugenics was held in London between July 24 and July 30. It was organized by the Eugenics Education Society of Britain. Although the meeting took place in Britain, eugenists from the United States were well-represented, and plans for the New York meeting of the Second International Congress of Eugenics were made by the American Consultative Committee. For details on the organization of both meetings, and their importance for the development of the movement, see Mehler (1988), as well as Ludmerer (1972).

Broadcasting the eugenic doctrine to the public in addition to incorporating it into the college and especially the medical school curriculum were understood by the eugenicists themselves as methods to secure the continued spread of eugenic doctrines and the growth of the movement as a whole. At the very least, we must recognize their belief that the movements' future was tied to the use of the college curriculum as a courier of propaganda.

When it comes to this view of education, Jordan was not alone at the First International Congress. Oxford philosopher F.C.S. Schiller also argued that eugenic views be placed more prominently in college education (Schiller 1921). But Schiller goes further than both Jordan and Reid, by suggesting that the educational system be re-organized to become an *instrument* of positive eugenics, rather than merely its trumpet. He held that educational institutions ought to be selective with respect to the heredity of their students, decrying the loss of the genetic heritage of the upper class. Schiller advocated reforms that would provide these eugenically fit with suitable incentive to apply their inherited qualities. Schiller suggests this as a means of “conserving resources” on a society-wide scale (Schiller 1921).

In Britain, the Eugenics Education Society (E.E.S.) formed earlier than its American counterpart, with its first meeting in 1907 (Farrall 1969, p. 206) and by 1914 it boasted 1047 members. This organized body was directed by a council comprised of 41 members. Donald MacKenzie (1981) analyzed the occupational background of this council, showing that 12 of the 41 directing members were either university professors or school headmasters. The prominence of educators on the governing council of the E.E.S. further betrays the extent to which eugenicists were involved in spreading their beliefs through education. If the occupational affiliations of the Society's directing members is any indication, we might expect to find that similarly a third of the members of the Society were active educators in some respect.

Official organization in the American context lagged somewhat behind the efforts of the British. The American eugenics effort organized in 1921 as the American Eugenics Society (AES). It sported a Popular Education Committee which recommended curricular reform through much of the 1930's.⁶⁰

Assembling a comprehensive sample of textbooks in English from early in the twentieth century is a mammoth undertaking. Using online databases, word-of-mouth, and inter-library loans, I was able to find and review eighteen textbooks on Mendelian genetics published between the years 1900-1922. Although this clearly falls short of an exhaustive sample, it is a place from which to start. And as I show, these texts are telling with respect to the presence and scope of the eugenic propaganda in biology and medical texts. Also noteworthy for the purposes of this dissertation is the frequency with which alcoholism is listed among the cardinal evils of society that eugenic methods aim to eradicate (other items on the list usually include feeble-mindedness, criminality, and general moral weakness; See Valverde 1998 for an excellent historical tracing of arguments for moral weakness).

4. Genetic Textbooks: 1900-1920s

Steven Selden (1985) has analyzed the content of three prominent texts published during this period: Charles P. Davenport's *Heredity in relation to Eugenics* (1911), Edwin Grant Conklin's *Heredity and Environment in the Development of Man* (1923), and Paul Popenoe and Roswell Johnson's *Applied Eugenics* (1918). His findings suggest that it was primarily through texts such as these that eugenists were able to appropriate "science" for their ideological purposes in the public mind. Davenport, in particular, made clear attempts to "capture the

⁶⁰ After which it declined in the public arena, due in large part to the withdrawal of support by prominent scientists as well as the appropriation of sterilization legislation and policies by Adolf Hitler (Kevles 1985; Allen 1975; Farrall 1969).

minds” of his students and so disseminate his racial agenda (Selden 1985, p. 43). My argument is different, however, in that I am interested in a particular subset of genetics textbooks, viz., those that incorporate Mendelian genetics. The rediscovery of Mendel’s work marks a crucial turning point for both the science of heredity in its own right and the manner in which hereditary information was perceived. With Mendel’s quantification of patterns of trait transmission, biological metaphors for society and its problems achieved remarkable purchase.

Of the 19 texts I survey, 15 of them (79%) contain positive references to eugenic policies. Given this feature of the sample and the nature of their content, these texts represent a remarkably unified stance on eugenic ideas. As a result, when a professor assigned any textbook on Mendelian genetics, he or she would likely be complicit in the spread of eugenic doctrines.⁶¹ As far as the students of genetics of the time are concerned, this may have been enough to set in their minds an association between the two, apart from the beliefs and political commitments of individual researchers in genetics.

A number of these textbooks were re-published in new editions several times during the period. Therefore, one of the simplest ways to trace the shifting eugenic content is to follow a single text through a number of its revisions. Let’s begin with one of the earliest such texts. Reginald Crundall Punnett’s name is mentioned in college genetics courses even today, in conjunction with the so-called “punnett square” he designed for representing the possible combinations of inheritance of characters. Punnett (1875-1967) worked with William Bateson on poultry and green peas during the first decade of the 20th century to

⁶¹ Which is not to say that professors followed the texts without critical commentary. As for the events in the classroom, we can only speculate in the absence of evidence that professors *endorsed* the eugenic tone of these texts. My claim is not that there was such endorsement; however, there clearly was *exposure* to the eugenicists biological *reduction* of social problems.

confirm Mendel's analysis of inheritance experimentally. He played an important role in the development of the discipline of genetics, as founding member of the Genetical Society, and founding the *Journal of Genetics* with Bateson. In 1905 Punnett published the first edition of a slim, 63 page textbook titled simply "Mendelism." It was the first such textbook devoted explicitly to the popularization of Mendel's work.⁶² Because it is a specifically focussed text, Punnett's *Mendelism* can provide a glimpse into the perceived importance of Mendel's findings for the study of living organisms, and most importantly, for the study and management of human beings. Fortunately, this text underwent many changes between the years 1905 and 1922, during which there were six editions published (substantive changes occur in only four of these editions).⁶³

a. Mendelism, 1st Edition (1905)

Punnett's text explicitly targets students of the life sciences in an attempt to reach as broad an educated audience as possible. The text covers a variety of experiments on plants, as well as some examples which extend the patterns of inheritance to animals. But it is in the final four pages of the book that we find a discussion of the human implications. Punnett begins by discussing the economic benefit breeders and farmers might reap by adopting certain practices suggested by Mendelian patterns of inheritance. After this talk of profit-through-genetics, Punnett moves on to larger goals.

⁶² Although William Bateson's text *Mendel's Principles of Heredity, A Defence* (1902) appeared three years earlier, its aim was to defend Mendelian principles against Prof. Weldon's criticism. In this respect it is neither a textbook nor a straightforward popularization and so has been left aside in the present survey.

⁶³ Since not all editions were available through inter-library loan, or other sources, it is not possible to estimate the total number of print runs and total volumes printed of each successive edition of this text.

On the subject of applying Mendelian inheritance to humans, Punnett's comments fall into two categories: policy recommendations, and the relationship between education and heredity. As to matters of politics, his remarks seem to be directed at those who may someday be in a position of power—such as government officials, legislators, or policy makers. He remarks,

That [the principle of gametic segregation] must apply to man also—that most complex of living forms—there is little reason to doubt. If there is aught in these matters the time is coming when they must be taken into account by those whose business it is with the ruling and advising of their fellow-men, whose wish is to leave the world a little *less aimless* than they found it... (Punnett 1905, p. 60 emphasis added).

This remark suggests that readers of the text, predominantly college undergraduates, would stand to benefit by exploring how Mendelian analysis could be used as a means of *governance or control*. Punnett continues:

Most of us are agreed that the circumstances of modern life are susceptible of change and of improvement. That end we seek to attain by better teaching and better sanitation. And in this direction we have made a start by concentrating attention upon the lower strata of society. Speaking broadly, our present policy aims at raising the standard of the less fit, at attempting to bring them closer by such means to those who are richer in natural endowment (p. 60).

What he means by “less fit” is not clear because we cannot be sure how widely accepted Darwinian and Spencerian ideas of “fitness” were at the time. Similarly, what he means by “lower strata” is rather vague. On the one hand, since the above remarks appear immediately

after a discussion of the economic virtues of this analysis of heredity, it may refer to an economic underclass. On the other hand, it could refer to a class distinction that is culturally based, rather than strictly economic. On either interpretation, Punnett's remarks suggest to the reader a use of Mendelian principles as a problem-fixing tool for elites. As such, these statements on the policy value of Mendelian genetics are certainly consistent with, if not suggestive of, policy measures undertaken in the name of eugenics.

On the relationship between heredity and education in humans, Punnett says:

Education is to man what manure is to the pea. The educated are in themselves the better for it, but their experience will alter not one jot the irrevocable nature of their offspring (p. 60).

That education will not "alter one jot" the "irrevocable nature" of a child is as clear a statement against the long term value of education as any. He continues,

Permanent progress is a question of breeding rather than of pedagogics; a matter of gametes, not of training. As our knowledge of heredity clears and the mists of superstition are dispelled, there grows upon us with an ever increasing and relentless force the conviction that the creature is not made but born (p. 61).

In short, his view on the relationship between education and genetics is one in which education is secondary to heredity.

b. Mendelism, 2nd Edition (1909)

Punnett's second edition of *Mendelism* is slightly longer, with 85 pages total, a new preface, and a single-page index in the back matter. Apart from these additions, very little of

the text has changed. Among the prefatory remarks, however, is what may be a change in the intended audience:

As year follows year, and experiment succeeds experiment, there is forced upon us a sense of what it all may come to signify for ourselves, of the tremendous *powers of control* that a knowledge of heredity implies. The prologue is nearing completion; the drama is yet to be written—and played (p. vii, emphasis added).

What's remarkable here is how rhetoric of “power” and “control” enter into his general account of progress in the field—thus suggesting the connection between experimental progress, on the one hand, and “powers of control” through knowledge of heredity on the other.⁶⁴

Since Punnett's remarks at the conclusion of the book focus on the potential for progress through human breeding, these remarks clearly may be directed at a future cohort of elites—those at the helm of society in professional and governmental positions. On such a reading, these suggestions of an increased “control” afforded by Mendelian heredity are consistent with the larger effort to direct the future of social policy by instilling a eugenic sense in students. I will return to this issue shortly.

⁶⁴ It may also be interesting to note how the history of eugenics renders ironic the last sentence in the quoted passage. The “drama yet to be written” may be read as the sterilization laws written in the mid-1920s, while the “playing” of this script could be the use of these laws as models for institutional practice by the Third Reich in the 1930s, culminating finally in the genocide of the Second World War.

c. *Mendelism, 3rd Edition (1911)*

Punnett's third edition boasts a wealth of additions and changes ("Entirely Rewritten and Much Enlarged," p. i).⁶⁵ The number of pages is double that of the previous editions (192, to be exact), and it contains a new preface as well as a more detailed index. The most prominent modification for our purposes is the division of the book into topical chapters, facilitating the addition of a final chapter simply titled "MAN."

In the preface to this edition Punnett offers definitions of two terms "genetics" and "eugenics." Genetics, he says, is the "term applied to the experimental study of heredity and variation in animals and plants..." (p. vi). Eugenics, on the other hand, "deals with the improvement of the human race under existing conditions of law and sentiment" (p. vii). Hence, *by the third page of the text he forges an association between genetics and eugenics.*

On the topic of what Mendelian genetics offers for the study of man, Punnett's position is straightforward: Genetics provides justification for adopting selective breeding as a policy to regulate human reproduction. Certain unions can now be understood as harmful to the "racial stock" of the nation, while other unions can be shown to be beneficial. In the former cases, state intervention is encouraged since this is the only way to ensure that potentially destructive reproductive combinations do not occur. Conversely, those matings which seem to be beneficial are to be encouraged. When one reads through Punnett's discussion of these matters, the blatant endorsement of the eugenic agenda is striking. Intervention into the reproductive choices of human beings is essentially prescribed as a *panacea* for society's shortcomings.

⁶⁵ This edition is published by Macmillan, rather than Bowes & Bowes, which indicates a larger print run than previous editions, as well as an American printing.

A final piece of reasoning that marks Punnett as an advocate of eugenics is a statement of the strategies and policies one might use to regulate mating in human populations. These remarks could easily have appeared in a eugenics pamphlet of the mid-1920s:

We are in the hands of the gamete; yet not entirely. For though we cannot influence their behaviour we can nevertheless control their unions if we choose to do so. By regulating their marriages, by encouraging the desirable to come together, and by keeping the undesirable apart we could go far towards ridding the world of the squalor and the misery that come through disease and weakness and vice.... Whether we are prepared to make use of [genetics] will depend in great measure upon whether we are prepared to recognize facts, and to modify or even destroy some of the conventions which we have become accustomed to regard as the foundations of our social life (Punnett 1911, pp. 184-5).

Policies of “regulating marriages,” prohibiting some and encouraging others were understood at the time as “negative” and “positive” eugenics, respectively. This is as clear a statement of the eugenic agenda as one can find. It is worth mention here because it appears in a widely-distributed textbook which, by title alone, would appear to be a neutral introduction to Mendelian genetics.⁶⁶

⁶⁶ Interestingly enough, Punnett was also active in the eugenics movement beyond simply writing textbooks. In 1912, at the First International Eugenics Congress in New York, Punnett gave a paper titled “Genetics and Eugenics” (Punnett, 1912). Here, Punnett is more reserved than in his textbooks on the rosy scientific status of eugenic doctrines. Observing the dearth of experimental data on heredity in humans, he warns that “our knowledge of heredity in man is at present far too slight and too uncertain to base legislation upon” (p. 238). Nevertheless, he suggests that science can see its way around the difficulties posed by

d. Mendelism, 4th Edition (1912)

This edition of the text contains few substantive alterations. Many plates have been altered, and there are some new examples to support Mendelian analysis of plant and animal experiments. However, the final chapter on “MAN” remains wholly intact.

e. Mendelism, 5th Edition (1919)

This edition also boasts more pages (219), and carries the preface from the 4th edition. Here the only addition appears to be a chapter devoted to discussion of some of T.H. Morgan’s experiments with *drosophila*.

f. Mendelism, 6th Edition (1922)

In this text few alterations have been made from the 5th edition. In the final chapter on “MAN” the discussion of sex-linked hereditary diseases has been altered slightly. But aside from this, the chapter remains unchanged. The comments on heredity in humans and their eugenic implications thus remain unaltered between 1911 and 1922.

At this point, it may be prudent to restate the objective of this textual analysis. I am concerned to show that the use of the educational system was instrumental in the rise of eugenics in the U.S. and Britain—a point that has been suggested but not substantiated in the literature to date. What I have shown occurring in these six editions of an introductory textbook on Mendelian genetics is *an increase in the mention of eugenic policies and an increased*

the unavailability of experimental human subjects by instead collecting “accurate pedigrees” and comparing these with “standard cases” exemplified by animal studies. And while he claims the category of “feeble-minded” is a viable one, he offers no strategy for comparisons of such non-physical attributes in animals.

connection between knowledge of genetics and the need for these policies. In the first edition (1905), there are only sparse remarks on the application of genetic knowledge to humans. Yet even then, the basic idea was to alter the distribution of social classes by adopting eugenic restrictions on the “lower strata” of society. By 1922 the same argument is deployed — but it describes in more detail the control of reproduction as a means to eugenic improvement.

While I have used Punnett’s text here to illustrate the manner in which eugenic social policies were promoted in the course of introductory works on Mendelian genetics, we need to remember that Punnett was by no means the sole crusader for the movement. Table 2.3 lists all of the textbooks I have found from the period, a total of 20 books. Of these, McKim’s book *Heredity and Human Progress* (1900) can be dismissed for it was published contemporaneously with the rediscovery of Mendel’s work and thereby does not associate the Mendelian analysis of heredity with eugenic aims.⁶⁷ William Bateson’s text also is of little relevance for it is not clearly intended as a textbook. These other texts are worth a closer look and so I will continue to survey some of the more prominent ones. For the purposes of this dissertation it is important to note that in nearly all cases where eugenists describe the target groups of sterilization, they mention alcoholism, inebriates, or drunkards, in addition to prostitutes, criminals, and “feeble-minded.”⁶⁸

⁶⁷ Which is not to say that the tone of the text was anti-eugenic.

⁶⁸ While the textbooks are curiously vague when it comes to description of the target groups, the pamphlets and marriage guides offer far more detail (although it falls outside of the purview of the textbooks under study, a good example of such a marriage guide is *You and Heredity*, Scheinfeld and Schweitzer 1939).

Table 2.3 Assessed Texts by Date, including Country of publication. Bold items indicate positive reference to both Mendel and Eugenics.

Date	Author	Title	Edition	United States	Britain	Mendel	Eugenics
1900	McKim, W. Duncan	Heredity and Human Progress	1	✓	✓	No	No
1902	Bateson, William, F.R.S.	Mendel's Principles of Heredity: a Defence	1		✓	Yes	No
1905	Punnett, Reginald Crundall	Mendelism	1		✓	Yes	Yes
1905	Reid, George Archdall	The Principles of Heredity: with some applications	1	✓	✓	Yes	Yes
1906	Lock, Robert Heath	Recent Progress in the study of variation, heredity, and evolution.	1	✓	✓	Yes	Yes
1908	Thomson, J. Arthur	Heredity	1	✓	✓	Yes	Yes
1909	Punnett, Reginald Crundall	Mendelism	2	✓	✓	Yes	Yes
1909	Whetham, William C. and Catherine Durning Whetham	The Family and the Nation	1	✓	✓	Yes	Yes
1911	Punnett, Reginald Crundall	Mendelism	3	✓	✓	Yes	Yes
1912	Doncaster, Leonard	Heredity in the light of recent research	2		✓	Yes	Yes
1912	Punnett, Reginald Crundall	Mendelism	4	✓		Yes	Yes
1913	Bateson, William, F.R.S	Problems of Genetics	1	✓		Yes	No
1913	Walter, Herbert Eugene	Genetics: and introduction to the study of heredity	1	✓		Yes	Yes
1915	Conklin, Edwin Grant	Heredity and Environment in the Development of Man	1	✓		Yes	Yes
1915	Pearl, Raymond	Modes of research in genetics.	1	✓		Yes	No
1916	Castle, W. E.	Genetics and Eugenics: a textbook for students of biology and a reference book for animal and plant breeders	1	✓	✓	Yes	Yes
1916	Guyer, Michael F.	Being Well-born: an introduction to heredity and eugenics	1	✓		Yes	Yes
1917	Lull, Richard Swann	Organic Evolution	1	✓		Yes	No
1919	Morgan, Thomas Hunt	The Physical Basis of Heredity	1	✓	✓	Yes	No
1922	Conklin, Edwin Grant	Heredity and Environment in the Development of Man	4	✓		Yes	Yes
1922	Punnett, Reginald Crundall	Mendelism	6		✓	Yes	Yes

21 texts total

19 recognize Mendel's work (leaving Bateson 1902 aside, as it is not really a textbook, *per se*).

15 present Mendelian Genetics *and* Positive Assessment of Eugenics. = 79 %

g. Recent Progress in the Study of Variation, Heredity and Evolution, Robert Lock (1906)

Robert Lock's prominent text takes a bleak view of the role of education in the progress of society: "The principles of heredity teach us that education and training, however beneficial they may be to individuals, have no material effect upon the stock itself..." (p. 287). This juxtaposition of education and heredity appears again and again in the sample of textbooks examined in this essay—with heredity represented as the more powerful force in nearly every case.⁶⁹

b. Heredity, J. Arthur Thomson (1908)

J. Arthur Thomson held the position of Regius Professor of Natural History at Aberdeen. Although he began his career with promise as a researcher, his work writing textbooks and scientific books for popular audiences soon outpaced his research projects (Bowler 2002).

In this textbook introduction to the biological study of heredity, Thomson argues that human heredity needs to be considered as a national political issue. In the conclusion of the book he puts it this way:

As the general results of biological investigation must apply, *mutatis mutandis*,
to man as well as other organisms, we naturally look to Biology for some

⁶⁹ Robert Heath Lock worked within the community of scientists aiming to clarify and extend the mendelian analysis of heredity. In this vein he assisted in clearing up a confused issue in genetics during the first decade of the 20th century, involving the proper understanding of some exceptions to Mendel's rules. Prior to the independent efforts of Cuénot, Castle and Lock, individual characters of organisms were assumed to be transmitted in heredity. Lock's book helped to establish the notion that characters are controlled by functional elements, or alleles, that are subject to transmission. Lock's experiments with

practical guidance in relation to human affairs...When all is said, however, we cannot but feel that the application of biological results is *only beginning*, and beginning with a tardiness which is a reproach to human foresight. There can be no doubt that it would ‘pay’ the British nation to put aside a million a year for research on eugenics, or the improvement of the human breed (pp. 506-507, emphasis in original).

This remark exemplifies the shift from progress in the biological study of heredity to calls for application of this knowledge to national health policy.

The topic of alcoholism arises in Thomson’s discussion of the effect on heredity of changes in the germ cells. Alcoholism poses a particular problem for the researcher, for there are two distinct pathways in which alcohol and heredity may be linked. First, there is the possibility that the human embryo is damaged before birth by the mother’s use of alcohol during pregnancy. This possibility actually became the subject of a significant research project by Karl Pearson and resulted in something of a controversy. Pearson’s 1910 Eugenics Laboratory research (with Elderton and Barrington) explored the question whether “alcoholism of parents had any marked influence on the mentality and physique of the offspring as *children*.” This study concluded that there was no evidence for effects on offspring so claimed, which elicited hundreds of angry responses from clergy, temperance advocates, and others concerned with alcohol and heredity. Mr. Montague Crackanthorpe (chairman of the Eugenics Education Society) wrote a letter to *The Times* of London that blasted Pearson and his study. Francis Galton himself responded to the letter in an attitude of “complete dissent” from Crackanthorpe, and was almost forced to resign his honorary

mice supported the view that these units could combine to exhibit more than simply two alternatives (Dunn 1965, p. 101).

presidency (Pearson 1938, pp. 171-77; Barrington and Pearson 1910; Elderton and Pearson 1910). Second, there is the possibility that “what is really inherited is a specific degeneracy of nature, an innate deficiency of control, perhaps, which led the parent to alcoholism, and which may find the same or some other expression in the child” (Thomson 1908, p. 189).

i. Heredity in the Light of Recent Research, Leonard Doncaster (1912)

At the time of the publication of this text Leonard Doncaster was a Fellow of Kings College, Cambridge, where he would later hold the endowed Derby Chair of Zoology (1919-20). Doncaster’s text is remarkable in the context of the development of hereditary theory for its contributions to the debate over the role of X chromosomes in sex-linked traits.

Couched within this text are comments on eugenics and education. Doncaster’s text, like so many others of the era, deploys the ironic dual role of education for the eugenics movement: on the one hand education is seen as a force which cannot rival that of heredity, and on the other hand it is described as the principle the means by which knowledge of heredity and eugenic ideals can be realized. Doncaster writes:

The father may educate his children because he himself was educated, but the mental powers of his children will be the same whether he had a good education or none. And the effects of special care given to the weakly or feeble-minded may be absolutely harmful to the race, if the improvement so effected leads to more frequent marriage among such unfortunates than would otherwise be the case, for then an increased number of defective children may be born, and the race average lowered. Hence has arisen the study known as ‘Eugenics,’ the study, that is, of the methods by which the race may be improved both physically and mentally. The whole trend of the

results obtained is that in order to produce exceptionally gifted men in both body and mind, those with high development of the characters desired should be encouraged to marry; and that to prevent the production of the weakly and feeble-minded, the only method is to prevent such from having offspring (pp. 50-51).

Continuing, Doncaster links eugenics with the issue of world politics and even national defense:

It is admitted that at present these things hardly come within 'practical politics,' but there is little doubt that the nation which first finds a way to make them practical will in a very short time be leader of the world (p. 51).

Among the traits eugenic principles might rid from society is alcoholism. In rapid succession Doncaster offers several possibilities for eugenic focus, including alcoholism (pp. 113-5), insanity (p. 102), slum populations (pp. 115-6), and criminality (pp. 117-18).

j. Heredity and Environment in the Development of Men, Edwin Grant Conklin (1915)

This text represents a collection of the "Norman W. Harris Lectures" for 1914, delivered by Edwin Grant Conklin. Conklin played a key role in the development of genetics as both a research program and academic discipline. He chaired the Department of Biology at Princeton University, where he lectured to thousands of undergraduate students and developed a number of research programs. One component of his investigations highlighted the structure of the cytoplasm of eggs and the role of its organization in the development of embryos—this during the growing focus on genes at the expense of other

cellular components (Fankhauser 1978). Conklin was elected to the American Philosophical Society and later served terms as both executive officer and president.

Although Mendelian inheritance is not mentioned in the title of this book, it is clearly the topic around which the entire volume is organized. Beginning with introductory descriptions of the factors of development, the cellular basis of heredity and development, and patterns of inheritance, the text sets the stage for the final three chapters that evaluate the relative contributions of heredity, discuss methods for the control of heredity, and ponder the ethical consequences of genetics. Structured in such a way, the text serves not only as a sound introductory text on the general science of heredity, but also offers recommendations as to how and why such methods can be applied to human affairs. Apparently the ethical quandaries posed by this application are second to the aim of continuing the process of natural selection in human populations, despite the unpleasantness it might entail:

The method of evolution by the elimination of persons, the destruction of the weak and cowardly and antisocial, which was the method practiced in ancient Sparta, is repugnant to the moral sense of enlightened men and cannot be allowed to act as in the past; but the worst types of mankind may be prevented from propagating, and the best types may be encouraged to increase and multiply. This is apparently the only way in which we may hope to improved permanently the human breed (Conklin, 1915).

In the course of this discussion of heredity in relation to human affairs the topic of alcoholism arises a number of times. In three instances alcoholism appears as an “inherited character” in pedigree charts, alongside other traits such as feeble-mindedness, criminality,

insanity, hysteria, and epilepsy (pp. 206, 295, 450). All other mentions relate to the role of alcohol as a poison on reproductive cells (i.e., the germplasm).

What bears mention about the appearance of alcoholism as a topic in these texts on eugenics is the nearly universal inclusion of alcoholism in lists of hereditary problems. And while we have made considerable headway since the early decades of the 20th century in removing “feeble-mindedness” and “criminality” from these lists, why have we not made similar progress with alcoholism?

Perhaps we would be wise to heed some of Conklin’s concluding advice: “We need to return to the joys of a childhood age in which men believed themselves free to do, to think, to strive, in which life was full of high endeavor and the world was crowded with great emprise. We need to think of the possibilities of development as well as the limitations of heredity” (p. 481).

k. The Physical Basis of Heredity, Thomas Hunt Morgan (1919)

Of the more influential texts in scientific circles the *Physical Basis of Heredity* by T.H. Morgan ranks among the more prominent. Published in both the United States (Princeton University Press) and Britain (Oxford University Press), this text includes a discussion of Mendel’s analysis of hereditary transmission, as well as some comments on their relevance to humans.⁷⁰ However, each of these comments serve only to illustrate the point that the germ plasm material in humans and other animals is the same, and appears to be located on the chromosomes. Morgan is careful not to delve into the arena of social policy in the course of his discussion of the details of hereditary science.

⁷⁰ Comments on “man” are listed in the index on pages 136-7, 170, and 300.

Nevertheless, Morgan remained deeply committed to the ideals of eugenics. He felt that eugenic aims were important, but had reservations about both the cases singled out for eugenic attention, as well as the ideal methods for achieving eugenic progress (Carlson 1981). While he voiced dissatisfaction with the methods proposed in the United States early in the century, his criticism was aimed only at the methods chosen by eugenic proponents, not their ultimate aims. In 1932 at the Eugenics Congress in New York, Morgan took issue with the limited state of knowledge on the part of eugenicists concerning exactly how genes could be connected with conditions as nebulous as intelligence or poverty (the latter particularly in the context of the great depression; Carlson 1981, pp. 178-180).

The place of Morgan's textbook in this analysis of eugenic social policy content is mixed. While Morgan was personally committed to eugenics in principle, his reluctance to endorse it in his textbook on genetic principles conveys some measure of his commitment to his profession. The elements of his text that mention humans serve to connect our hereditary situation with that of model organisms under study by geneticists. In this limited regard, his text does not undermine or call into question the connections between eugenics and genetics forged by other introductory texts of the era.

1. Genetics, An Introduction to the Study of Heredity, Eugene H. Walter (1920)

By 1920 some members of the eugenic movement had softened their stance somewhat. This accompanied a divergence between staunch advocates of eugenic policies and the increasingly narrowed focus by genetic scientists on physical and chemical characterization of germplasm. Some researchers in genetics began to distance themselves from direct involvement in social policy discussions, and considered those who sought the public eye with suspicion (Bowler 1989, p. 168).

In this text, Walter flips the earlier education goal on its heads, arguing that education is a better policy than legislation for achieving eugenic ends.

A far more effective means of restricting bad germplasm than placing elaborate marriage laws upon our statute-books is to educate public sentiment and to foster a popular eugenic conscience, in the absence of which the safeguards of the law must forever be largely without avail (p. 251).

However, in discussing the effectiveness of Mendelian analysis of human problems, Walter articulates a certain impatience for the ability of science to adequately model social problems.

“For practical purposes it is unimportant to know whether or not feeble-mindedness, or any similar defect, is Mendelian in behavior. The fact that it is hereditary is enough” (p. 311).

Here he refers to a chart taken from Goddard, where two defective parents exhibit only defective offspring. The list of traits that count as “defective” in this respect is important for the purpose of this dissertation: “alcoholic, criminalistic, feeble-minded, and tubercular.” Situating alcoholism as a prime behavior in need of eugenic attention exhibits the ease with which eugenic policies could be made relevant to alcohol-related behavior. Earlier in the text (prior to discussion of Mendelian patterns of trait transmission) Walter remarks “when alcoholism ‘runs in a family,’ its reappearance in the son is probably due to the fact that he is derived from the same weak strain of germplasm as his father” (p. 81).

5. College Student Cohort

Who might have been reading these eugenic-minded textbooks? Can we possibly estimate the true impact of these texts? Answering these questions is difficult, but not impossible. If we view the situation in terms of the students exposed to eugenic doctrines while in college, we can distinguish a cohort that may have been exposed to eugenic

principles in biology and medical coursework.⁷¹ As this cohort entered the middle-class and professional workforce, it would gain increasing control over the governance of the nation, become more active in politics, and generally have a greater influence on the cultural milieu. The fact that eugenic sterilization policies were put into practice in the United States through the mid 1970s can only suggest the extent of the legacy we can attribute to the use of education by eugenists.

This cohort would be unique from previous graduates in that they would have greater exposure to producing social policy from genetic science and theory, as the eugenists argued in their textbooks. So educated, they would more readily support eugenic policies than a cohort that lacked such exposure in college curriculums. And by the 1930s, when this cohort is disrupted by the disintegration of the dialogue between genetic researchers and eugenic propagandists, the fading of the movement in the face of developments in international politics would follow naturally.

The connection between eugenists' use of textbooks and the success of the program cannot be firmly established without some measure assessing how the new converts to the movement were first exposed to the central idea. On the basis of the analysis of Punnett's *Mendelism* I have presented so far, and the sample of 15 other textbooks in this period, I can only suggest that college textbooks may have been an additional element contributing to the success of the movement. However, the continuing influence of such texts on ideas of the role of heredity in the evaluation of social ills should not be underestimated. One way to assess whether such notions live on involves inquiring of the *modern* textbook whether social

⁷¹ The story of medical school curricula during this period is related, but not specific to our concerns here. Briefly, Archdall Reid published a text in 1905 directly targeted toward medical school courses on heredity. This marked an abrupt transition from earlier considerations of medical school curricula (e.g., see Puschman 1966) which entirely overlooked topics of heredity and genetics.

and reproductive policies are still espoused, particularly in texts that introduce students to the principles of genetics.

6. Genetics Textbooks from the 1990s

In order to assess whether the zeal for the gene-myth, or even for eugenic social policies, persists in textbooks written today, we can employ a simple survey similar to that undertaken above. In some ways this is easier for the new texts, for modernity has rendered the textbook an orderly and systematic learning tool, free of the rambling, polemic and diatribe so common to texts from early in the century. The texts also compile vast amounts of technical and theoretical information, most of which was unavailable for earlier writers. As a result, we can look to the introductory remarks and sections that deal with human behavior, disease, or social patterns, in order to mine these texts for social commentary.

a. Genes and Genomes, Maxine Singer and Paul Berg (1991)

The authors of this text are two of the most prominent members of the genetic research community. Maxine Singer was named president of the Carnegie Institution of Washington in 1987, this following nearly ten years as Chief of the Laboratory of Biochemistry. Paul Berg developed the first recombinant DNA molecules in 1972, an achievement for which he was awarded the Nobel Prize in chemistry in 1980. Developing recombinant DNA molecules essentially underwrote the field of genetic engineering. Yet Berg was presciently aware of the potential hazards posed by the use of his methods, and penned a letter proposing a voluntary one year moratorium on recombinant DNA research in order to forestall experiments that might develop new and potentially devastating viruses.

Singer and Berg's textbook projects an enlightenment tone, with frequent reference in the introductory remarks to "progress," "opportunity" and the growth of knowledge.⁷²

The authors also present Bergs recombinant techniques with pride:

The genotypes, and thus the phenotypes of individual proteins and of whole cells and organisms, can be altered, providing future opportunities to investigate fundamental biological processes as well as to address critical problems facing our species and the planet we inhabit (p. xxi).

This is part of an optimistic and uncritical tone that continues throughout the text. The authors also attempt recast Berg's earlier call for a moratorium as unnecessary.

The early public fears about the biological revolution engendered many negative attitudes about the research. Biologists then feared the worst: highly restrictive laws or regulations that would seriously hinder further experimentation and its promise of new *knowledge* and beneficial applications to medicine, agriculture, and industry. Many scientists regretted the initial open discussion of the issues in the face of successful demagoguery by critics and the tendency of newspapers to build hype rather than carefully explore difficult issues. Yet it has turned out well. The science described in this book attests to that, as do the growing number of important *products* being produced by the young and energetic biotechnology *industry*. Perhaps the moratorium and initially *restrictive* guidelines held things up, but only briefly.

The early caution in the face of *ignorance* was prudent, even though hindsight

⁷² For example, in the preface they write "...we have focused on selected specific areas that have already been studied in some depth and that illustrate the progress that is being made" (p. xx).

suggests that the risk scenarios were far less likely than we had supposed (p. xxv, emphasis added).

Even though these statements clearly exaggerate the success of science and industry (especially in genetic modification), the text is otherwise devoid of comments of a political or social policy nature. In short, the authors safely ignore the contentious history of attempts to manipulate human heredity and their ensuing consequences. Singer and Berg offer no speculations on how the science of heredity might influence social or public health policy.

b. Elements of Medical Genetics, Alan E.H. Emery and Robert F. Mueller (1992)

Emery has worked on both sides of the Atlantic. He holds a Ph.D. in human genetics from Johns Hopkins University awarded in 1968, subsequently chaired the department of Human Genetics at the University of Edinburgh, and following this worked with the Division of Neurology at Duke University Medical Center. During the early 1980s he served as president of British Genetic Society. He has published a number of books on methodological issues and diagnosis in medical genetics. The second author, Mueller, holds a position in Clinical Genetics at the University of Leeds, and currently is the regional Head of Genetics Service at St. James Hospital in Leeds.

In this textbook targeted toward medical students, the authors discuss the association between eugenics and genetics early in their introductory remarks. In a section on “the beginnings of human genetics” they review Galton’s role in the eugenics movement, casting the aims of the movement as admirable, if unreachable given the state of the science at the time. They write:

Galton had many interests and among them was the advancement of the idea of hereditary improvement of men and animals by such methods as selective

breeding, for which he coined the word *eugenics*. Over the years a eugenics movement developed which had fervent followers both in Europe and the United States. It seemed reasonable at the time that a desirable aim of human genetics should be the improvement of the human species by selective breeding. For many years, therefore, human genetics and eugenics were linked in people's minds, but even today our knowledge of human genetics is too rudimentary to advocate drastic eugenic policies. What information we do have suggests that such measures would be largely ineffective anyway. This is not to say, however, that there is no place for warning those who are at risk of passing on or having children with hereditary disorders and explaining the reproductive choices available to them (p.7).

There are two troubling aspects of these remarks. First, note the authors depiction of the problems inherent in the eugenic approach to human behavior: "even today our knowledge of human genetics is *too rudimentary* to advocate drastic policies." At what point would knowledge of human genetics be *sufficient* to underwrite eugenic policies? The authors leave this to the imagination, clearly suggesting that such knowledge is not beyond our grasp. Why focus on the "drastic" methods employed by eugenicists? Are there non-drastic, perhaps "sensible" methods to which the authors would not object?

Second, what role are we to think modern human geneticists will serve? Do they exist only to "warn" those in danger of hereditary disorders, informing them of their "reproductive choices?" It is not at all clear that such warnings should be free from directives to refrain entirely from natural procreation. The line between such warnings and eugenic guidance is a fine one. To the extent that warnings against reproduction fall short of state-supported sterilization, they do not constitute the worst of the eugenic policies. But

are these hypothetical experts in human genetics merely consultants, or do the authors envision a degree of power to their decisions? This is left to the reader's imagination. In this respect these textbook statements are dangerously ambivalent with respect to the complicity of genetic science with eugenic policies, both past and future.

Further exhibiting this ambivalence, the authors present eugenics as a potentially promising but historically unfortunate episode in a section toward the end of the book titled "Man's Future Evolution." They write:

Because of improved medical care we are prolonging the life of many individuals who would otherwise have died before reaching child-bearing age. The result will be an accumulation of 'bad genes' in the population which would have normally been eliminated. This is the price we have to pay for being civilized. The question now arises, 'What can we do about this?' It would be unethical not to treat persons with many of these conditions and there is no doubt that advances in medical treatment have helped to relieve an enormous amount of personal suffering. But if we are to continue treating individuals with such diseases we must also be prepared to face the consequences of having to deal with more affected persons in each succeeding generation. It has been argued that affected persons might be persuaded not to have children. Voluntary or even legislative sterilisation has been proposed. The former is a choice which some individuals may choose to exercise but should never be advocated from some misguided eugenic viewpoint, while it is doubtful if the latter is ever appropriate (pp. 260-261).

The authors make no effort to dispel the notion that "bad genes" are accumulating in the population. In fact, all of their subsequent comments on treatments are based on the

assumption that such bad genes exist. What might “bad genes” be? Are they related only to disease, or do they affect human performance in more general and abstract terms? This assumed reduction of badness to genetic underpinnings is as good an example of the gene-myth at work that one could concoct. For a textbook on medical genetics, it is surprising to see this association of loaded moral language with talk of genetic counseling. It is more surprising to see that the authors might wish to dismiss the *methods* employed by the eugenicists, they tread dangerously close to endorsing the eugenic *aims* of ridding “bad genes” and “low intelligence.” On the matter of intelligence, they write:

Another related problem is that of controlling the fertility of persons with severe mental retardation. It has often been suggested that in our society a slow decline in general intelligence is to be expected since those with low intelligence tend to have large families whereas those with high intelligence have small families....There is no reason to believe that our natural intelligence is declining and no reason for sterilising the severely mentally retarded because they are usually subfertile anyway (p.261).

In their view, we do not need to resort to eugenic policies with respect to the mentally retarded, *not* because it might be unethical to do so, rather because these people are “usually subfertile” in the first place. We can see that the authors are comfortable with the eugenic aims, and perhaps relieved to find that natural patterns allow them to avoid resorting to the negative eugenic sterilization methods.

On the subject of *positive* eugenic (encouraging breeding among the gifted), the authors seem to be committed to *both* the aims and the methods. Continuing, they write:

Finally, there is the question of producing outstanding people by selective breeding, or so-called positive eugenics. One way of doing this might be to

give tax relief and family allowances to persons with those attributes which we wish to propagate. But here we are faced with the problem of deciding which genes are the ones we wish to increase by selection. There is no guarantee that we will still consider the qualities preferred today important in future generations. Also there is the much more serious problem of how we are going to control such selective breeding programmes. These are complex problems which might well occupy the whole of a book this size (p. 261).

These remarks not only endorse the aims of positive eugenics—they also propose methods. The only mentioned “problem” stems from the difficulties we might encounter when *managing* the eugenic programs. In essence, their reasons for putting off a eugenic program is procedural and lacking entirely in normative content. That the procedural or management problems would require “the whole of a book this size” further suggests that these problems are surmountable if merely given the amount of attention the authors currently devote to medical genetics. In short, the authors seem to have no qualms about the eugenic aims themselves, nor any deep reservations over methods and policies designed to realize these aims. This text is unequivocally the strongest endorsement of eugenics among those I have sampled.

Finally, the authors leave us with a vision of the importance their own discipline will have in the future, encouraging their student readers to believe their role is largely preventive:

In the future, the physician may become more involved in treating people who appear perfectly healthy but are genetically predisposed to a particular disease. He may even find himself treating mothers during pregnancy in

order to prevent certain hereditary diseases in their offspring. Medical genetics seems likely to become the preventive medicine of the future (p. 315).

c. Essential Genetics, Daniel Hartl (1996)

Daniel Hartl studied genetics at the University of Wisconsin and held a number of prominent posts prior to his appointment as Higgins Professor of Biology in the Department of Organismic and Evolutionary Biology at Harvard University (where he remains as of this writing). He has worked with the National Institutes of Health Genetics Study Section, and served on the editorial boards of a number of key journals in genetics and molecular biology.

In the prefatory remarks of this widely used textbook the author projects what we might call an “enlightenment tone”—promising limitless progress, with no vision of an end to the relevance of genetic information:

Genetics is endlessly fascinating. It is a unifying theme that cuts across every aspect of biology from structural biochemistry to evolution. Genetics is relevant not only to biologists, but to all members of our complex technological society. Understanding the principles of genetics will help you to make informed decisions about personal issues affecting you and your family, as well as broader issues of social and political concern (p. xix).

The details of genetic science’s relevance to “social and political” issues are left unarticulated. It is unclear whether this author is paying homage to eugenics or simply ignorant of the history genetics shares with the eugenic movement. Looking to the future of applications derived from genetic principles and clever tinkering, the author touts the lucrative promises

of genetic engineering: “Currently genetic engineering is providing us with new tools of great economic importance and of value in medical practice” (p. xx). While this may appear to some as too rosy a picture of the consequences of genetic engineering, it is also noteworthy because it follows the same rhetorical blueprint we noted in Punnett’s texts from the beginning of the century. The discussion begins with praise for agricultural and economic benefits, then moves to the principles of genetics themselves before turning to human genetics.

While the text makes no direct mention of eugenics, there are comments in the discussion of genetic counseling that do nothing to dispel belief in eugenic aims. Here the author limits his discussion to inherited diseases (no list of these is provided), and seems to envision a preventive role for such counselors. Hartl writes:

Human beings are heir to several thousand different inherited diseases.

Married couples can be informed of the possibility of their producing affected offspring and can now make choices between childbearing and adoption. What a relief for a woman and a man to learn that they do not carry a particular defective gene and so can produce a child without worry.

When an offspring might be affected with a genetic disorder, techniques are available to determine *in utero* if a fetus is, in fact, affected (p. xx).

Aside from this vague mention of human genetic disorders and in utero techniques, there are no further suggestions of eugenic aims or methods throughout the text.

d. Basic Human Genetics, E. Mange and Arthur Mange (1999)

Arthur Mange is currently emeritus professor of the biology department at University of Massachusetts. In this prizewinning textbook on human genetics first published in 1994, the

authors offer another rosy picture of genetic counseling and “humane” human genetics, perhaps to dispel any association with eugenic aims or policies. In the introduction they write:

But human genetics can be employed humanely at the personal level.

Increasingly sophisticated prenatal testing and genetic counseling are able to avert family tragedy or reassure those individuals who turn out not to be at risk for certain hereditary disorders. Great caution, care, and sensitivity are required, however, when considering or carrying out the large-scale testing of populations that are known to be at increased risk for certain conditions.

Even for those who oppose testing, there is still the problem of just maintaining our current genetic endowment, as when, for example, medical advances preserve detrimental genes that would otherwise be eliminated by the death of these individuals. The possible long-term genetic and social consequences of medical practices will be debated for years to come (p. 12).

While these authors clearly worry about the persistence of a negative genetic endowment, they confusingly present two very different hopes. First they present optimism for social and economic environmental betterment strategies, then shift to optimism for the potential of managing human breeding. It is best to quote this confusion in its entirety:

Because of meiotic segregation, polygenic inheritance, and especially environmental effects, the specific characteristics of a parent are rarely passed on intact to a child. For example, using Babe Ruth as a sperm donor would only slightly increase the probability of producing a slugger. (Indeed, none of Ruth's 15 descendents has shown any particular talent for baseball.) We wish to emphasize that phenotypic variability is influenced by differences in both

genes and environment. Rather than trying to control the genes with which a person is born, society would be better served by providing the enriching environments that foster optimal expressions of the genotypes that currently exist. The benefits of a multitude of public and private programs for betterment—socioeconomic, health related, educational, cultural—have never been fully realized (p. 455).

Turning to eugenic aims and methods, they continue:

Still, there is some eugenic potential (depending in part on the heritabilities of the traits in question) in currently feasible and safe technologies that are voluntarily undertaken with minimal, if any government oversight.

Technologies such as genetic testing and reproductive assists can contribute in humane ways to the universal desire of parents for healthy, normal children. Because of its offensive history, however, the term eugenics should perhaps be discarded. ‘But the judicious use of genetic knowledge for the alleviation of human suffering and increase in the well-being of future generations is a novel ideal, whatever it is called’ [quote from Crow 1988] (p. 455).

The authors clearly want to distance themselves and the future of human genetic tinkering from eugenics, wishing to discard the term itself. However, they leave up to the reader the boundaries of what might constitute “humane” realizations of the “eugenic potential.” It is unclear what methods fall under the category of “reproductive assists.” We can charitably assume that these methods do not include sterilization, and the author’s use of the word “voluntary” clearly sets their vision apart from a severe program of negative eugenics. But while they distance themselves from the aims and methods of negative eugenics, it is clear

that by seeing a positive role for genetics in shaping our human future they embrace the *aims* of positive eugenics.

To summarize this section, we might ask: What does this survey of textbooks from opposite ends of the 20th century show with regard to the gene-myth of alcoholism? The connection that I have articulated between eugenic propaganda within these texts, as well as the frequent use of alcohol problems in lists of targets for eugenics policies in the earlier texts, shows that the gene-myth has remained entrenched in genetic instruction for a considerable span of time. The extent to which the continuity of the gene-myth guides modern views of the hereditary nature of alcoholism remains to be settled. It has been shown elsewhere that textbooks may have served as particularly potent vehicles for the dissemination of ideology, as in the cases of Nazi Germany, Mussolini's Italy, and Fascist Austria prior to the outbreak of WWII (Pine 1996). Textbooks in general, and biology textbooks in particular, have long been recognized as sites in which the natural / social divide is formulated, with the usual result that social problems are given "natural" explanations (Kuhn 1962; Haraway 1997). The policy content of these textbooks on genetics warrants the attention of today's scholars, particularly those concerned with the further dissemination of the gene-myth.

The eugenics movement has been described by historians as only one expression of the rising educated professional middle-class in the U.S. and Britain at the turn of the century (Allen 1975; Farrall 1969; Karier 1975; Selden 1978; Pickens 1968). At the beginning of this chapter I argued that this group, well educated and cognizant of the power of technical and scientific information, may have identified with a movement defined by just such a technical foundation in what appears to be a scientific treatment of heredity. We saw

that eugenisists themselves at the turn of the century argued that the success of the movement depended on finding a place for their teachings in the curricula of college, medical schools, as well as secondary education (Jordan 1921; Schiller 1921). Moreover, many of the advocates of eugenics in both Britain and the United States at the turn of the century were educators, presidents of universities, college professors, or curriculum specialists (MacKenzie 1981). It follows that there was a significant educational component during the first two decades of growth of the eugenics movement. While scholars have traditionally attributed this growth to grass-roots methods and powerful professional affiliations, I maintain that the college biology and genetic textbook remains an important factor in the rise of this movement focussed on articulating and spreading the gene-myth.

By reviewing this sample of 20 texts I have shown that between the rediscovery of Mendel's work in 1900 and the organization of the Popular Education Committee in the United States in 1921, a number of authors broadcast the eugenic doctrine to a cohort of college and medical students using college textbooks on heredity and Mendelian genetics. As we saw above (in Table 2.3) fifteen of nineteen (or 79 %) textbooks on Mendelian genetics published between 1900 and 1922 employ the rhetorical value of Mendelian genetics for the service of enhancing the credibility and urgency of eugenic aims and methods.

The eugenics movement stood to benefit from such exposure in textbooks in two ways: First, a tight association in these texts between the Mendelian principles and eugenic policy served to legitimize the latter by means of the scientifically sound status of the former. Second, the movement grew because the cohort of students exposed to such texts in the first two decades of this century were arguably members of a rising professional middle

class, if not also future elites.⁷³ This is not to deny that the eugenics movement was gathering considerable momentum prior to 1900. Rather, in addition to this extant momentum, eugenicists deployed textbooks as devices to reach a growing and potentially powerful segment of society, while simultaneously benefiting from the association with scientific theory and technique.

Encapsulated within this association of eugenics and genetics is the widespread assumption that alcoholism ranked as one of the chief problems of heredity to which eugenic negative eugenics might be applied. The full list of problem groups for eugenic solutions ranged included insane persons, epileptics, paupers, criminals, unemployables, habitual slum dwellers, prostitutes, and inebriates (Kevles 1985, p. 113). In textbooks focussed on Mendelian genetics, many of these groups are mentioned and connected with evidence for patterns of inheritance. While “feeble-mindedness” probably ranks as the most frequent category of eugenic focus, there is nonetheless persistent mention of alcoholics, inebriates, and drunkards as candidates for hereditary analysis.

The presence of eugenic propaganda in textbooks suggests that we ought to be on the lookout for reproductive policy recommendations in genetics texts presently. The survey of four textbooks from the 1990s shows that eugenic aims and even methods continue to appear in college texts. While the focus is now on building healthier humans through heredity (rather than “better” humans), and despite authors’ efforts to distance their

⁷³ While it stands to reason that college and medical degrees were secured mostly by the upwardly mobile or current elites, we lack hard data to show the extent to which this cohort actively shaped policy in both Britain and the United States. Physicians continued to expand their influence on public health in the first three decades of the 20th century (Starr 1982, p. 83). Legislators, political leaders, progressive organizers, and many others played significant roles in the success of the eugenic movement as well (Kevles 1985, p. 58ff). My claim is that there is a high likelihood that this cohort was exposed to positive considerations of eugenic aims and methods if they took virtually *any* college course on heredity.

suggestions from the name of eugenics, policy recommendations and favorable consideration of the *aims* of both positive and negative eugenics persist. If we can attribute the growth of the eugenics movement in part to popular and scientific attention gathered from the rediscovery of Mendel's work at the beginning of the century, the recent completion of a draft genome from the Human Genome Project conjures an intriguing parallel. With modern genetics prominent in the public eye, we might stand to benefit from a careful and considered appraisal of current social policy claims made on its basis, wherever they occur.

So far in this chapter I have argued that ideas concerning heredity have an entrenched history. I have also displayed how some of these ideas have been put to use by the eugenics movement, and that textbooks arguably played a significant role in the early success of this movement. The fact that alcohol related problems receive frequent mention in both eugenic and genetic contexts during this period in key texts on the subjects suggests the extent to which the gene-myth for alcoholism is entrenched at present.

7. Curious Continuities

This chapter began with a review of Sir George Archdall Reid's work on heredity and alcoholism. Using Reid as a guide, I broadened my scope for surveying texts on hereditary aspects of human problems, noting the eugenic tone of genetics texts at both ends of the 20th century. But this is not the only continuity across the century that is suggested by Reid's work. Indeed, there are two further aspects of his work that clearly foreshadow, if not pre-empt, recent developments in the field of alcohol studies. The first of these is the

aforementioned “outbreeding of alcoholism” notion. In one formulation of the theory he puts it the following way:

All races had a common origin, and, therefore, had once a common temperament, which, to judge by the analogy of primitive peoples, was of that kind which renders intoxication delightful. They have since diverged widely. In every case the temperate races of modern times have suffered prolonged elimination through alcohol; the drunken races little or none (Reid 1901, p. 124).

Curiously enough, this theory re-emerges in 1989 in an article authored by two addiction researchers, published in *The American Journal of Drug and Alcohol Abuse* (Carpenter and Ewing 1989). These authors trot out much more recent but intrinsically similar evidence in support of the idea that ethnic and racial differences in rates of alcoholism and disease due to alcohol consumption may be the result of differences in ancestral exposure to alcohol. Some recent findings can be marshaled to support the idea, most notably the presence in large portions of Asian populations of a variant form of the enzyme Acetaldehyde. This variant form of the enzyme causes accumulation of a metabolic by-product of ethyl alcohol, which causes irritation, swelling and flushing of the skin. Despite this finding an empirical test of the theory of “outbreeding” has yet to be undertaken, and even so, it is unclear how one would go about determining both ethnic ancestry as well as historical exposure to alcohol. Still, researchers in alcohol studies cling to the idea that alcohol is an agent of natural selection. Said one recent writer, “Alcohol-caused motor vehicle accidents are a major cause of death in the early reproductive years, so natural selection should gradually

decrease the human preference for alcohol. The time course of changes from natural selection is so slow, however, that cars will likely be extinct before such selection eliminates our taste for alcohol” (Nesse 1994).

Reid’s work is remarkable also in its resemblance to claims of novelty from practitioners of “Darwinian Medicine” (and also “Darwinian Psychiatry”). This new approach claims to refresh medicinal and psychiatric perspectives on human diseases and disorders by consideration of the evolutionary contexts thought to be responsible for the diseases. One writer decries the lack of evolutionary theorizing around alcoholism: “Evolutionary origins of alcohol consumption have rarely been considered in studies of ethanol addiction” (p. 3), and elsewhere “evolutionary perspectives are conspicuously absent from the literature on human alcoholism” (Dudley 2000, p. 7). Similarly, in *The Present Evolution of Man* Reid claimed “never before have diseases been considered from the standpoint of the evolutionist.” These statements are separated by 103 years, but the resemblance is striking.

As historians and philosophers of science, it is our duty to investigate continuities and discontinuities across stretches of time and cultural milieu. We have learned so much about evolution and genetics in the last 100 odd years that it seems obvious to apply its teachings to our modern diseases. However, the parallel story on alcoholism suggests that we need to take a much closer look not only at our disease-classifications and definitions, but also the deeper assumptions built into their formulation. Evidence I have presented in this chapter further suggests that the stories of alcoholism and eugenics may not be historical parallels, but rather interwoven articulations of a singular zeal for characterizing human problems by reference to heredity.

The fact that eugenic ideas—the most widely influential and deepest genetic reduction of social problems—are represented in textbooks on genetics at opposite ends of the 20th century suggests an acceptance of the gene myth that could be coloring our modern conceptualization of social problems like alcoholism. Furthermore, the fact that G. Archdall Reid, one of the first to focus on alcoholism as a hereditary problem, was an ardent supporter of eugenics points to a stronger connection between eugenics and the desire to attribute to genes causal primacy in alcoholism scholarship. To be sure, modern alcohol researchers are not eugenicists. However, the undercurrent of genetic reductionist solutions to social and psychological problems that persists in introductory texts on heredity throughout the century coupled with the clear central focus on genetic accounts of alcoholism in the present day warrants careful attention. It is this persistence of the gene myth throughout the 20th century that beckons a careful examination of the structure of genetic accounts of alcoholism with an eye toward uncovering the plausible alternatives to the reductionist model.

Chapter 3: Fundamental limits to genetic explanations of alcoholism⁷⁴

“How would you differentiate between an alcoholic and a gifted wine taster?”

—Joseph Pitt, 2002

Developing a perspective on the depth at which the genetic reduction of alcoholism is entrenched, I showed in the last chapter how textbook presentations of genetic science are still not free from potentially eugenic content. I noted also the eugenic tendencies of Sir George Archdall Reid, the author of the first English-language book devoted to the hereditary question of alcoholism. The close parallel between his theories at the beginning of the century and those offered eighty years later suggests the extent to which researchers even now operate within a paradigm of genetic reductionism, and how powerful a guide of inquiry this paradigm is despite the lack of understanding regarding a mechanism. In this context, genes are imbued with power to explain multi-faceted and socially amorphous features of human condition, even though it remains impossible to articulate the causal pathway on which the explanation must be built. The purpose of the following chapter is to consider key conceptual and empirical difficulties encountered in attempts to analyze mechanisms that can bridge the gap from DNA to social deviance. Only with a clear picture of these limitations will we be able to move forward to a reassessment of the paradigm in which they are currently overlooked.

⁷⁴ Portions of this chapter appear in my article “Why Do We Drink? A History and Philosophy of Heredity and Alcoholism,” *Bulletin of Science, Technology and Society* February 2002, vol. 22 (1): 48-55.

1. Limitations to the formulation “the gene for”: The Norm of Reaction

The logic underlying modern attempts to delineate genetic from environmental factors in the causal account of a disease or disorder underwrites also the claims in the popular press (and in patent filings) for momentous discoveries of “the gene for” the disorder. However, as we will see, the logic applies only in certain ideal cases, yet the discovery claims continue.

There are two types of gene-related disorders that plague humans and other organisms. In the first class we can place those disorders that have as a cause a single genetic fault, the narrowest definition of which is a “single nucleotide polymorphism” or SNP. An SNP occurs when a single base element in the nucleotide chain (of say, “AGT”) of a gene is changed (to, “GGT”). The consequences of single data-point changes can in some cases be quite severe. Most inborn errors of metabolism (that is, enzyme deficiencies that render the body incapable of dealing with specific ingested molecules) result from alterations of a single genetic base, or a single gene locus (e.g., Tay-Sachs disease, Alkaptonuria, Phenylketonuria, etc). The approach of looking for genetic markers for diseases that fall into this category is largely successful. For a specific disorder we can isolate a specific genetic aberration that may be responsible. To do this researchers determine through statistical analysis of biological material which chromosome might carry the problematic genetic factor. By analyzing the material at a number of levels from chromosome loci to individual gene alleles, they are able to isolate and compare the relevant functional piece of DNA that produces an odd variant of the molecule in question.⁷⁵ Unfortunately for the genetic researcher, these types of disorders are rare.

⁷⁵ The steps in this process have changed somewhat as a result of the completion of the Human Genome Project, and will continue to change. The ability to articulate the genetic sequence of an entire set of chromosomes from an individual opens up the possibility for

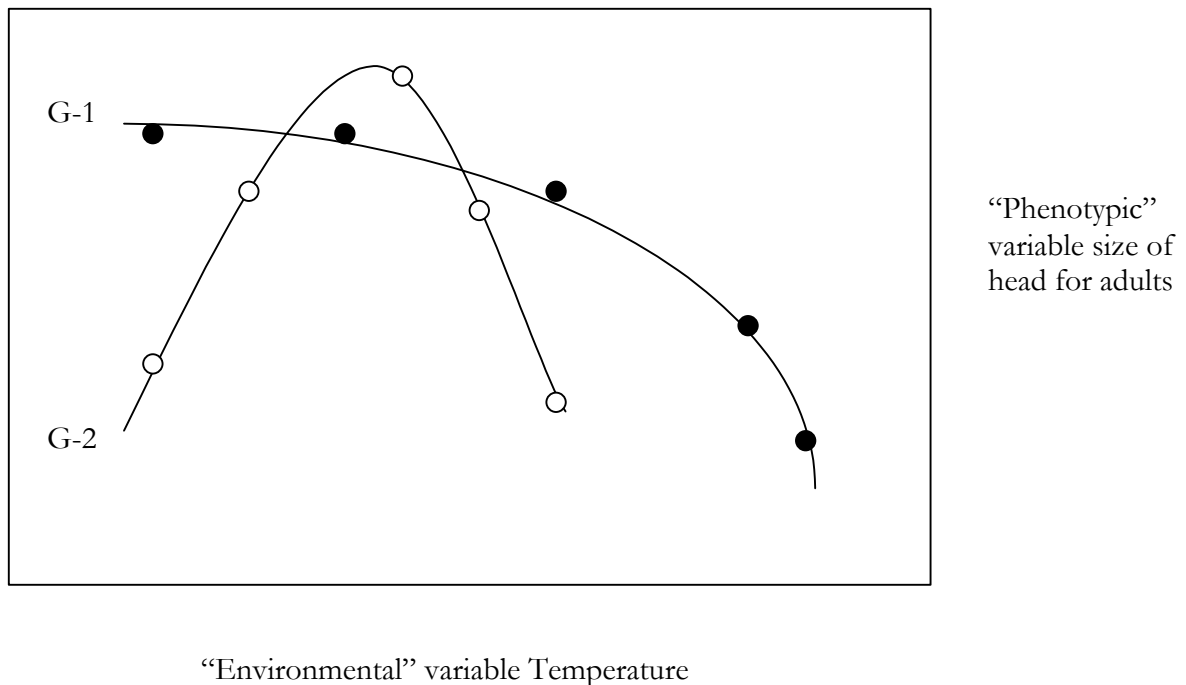
The second category of disorders are those conditions that are thought to be the result of collusion of many different genes dispersed across many genetic loci. Behavior genetic researchers admit that most of the conditions for which they seek genetic underpinnings probably result from the interaction of hundreds and possibly thousands of genes. Lewontin (1974) pointed out some time ago the difficulty for researchers in attempting to analyze such disorders with the methods traditionally used to analyze the first type. In order to present the full weight of this critique, we need to consider first just how complex the relationship between genome and environment is in the case of complex organisms.

For any organism we have studied since the rediscovery of Mendel's work, our analysis begins with the isolation of a particular trait or characteristic (i.e., an aspect of the organism's phenotype). A genetic reductionist technique proceeds by attempting to distinguish the relative influences of the environment from those of the genotype in the production of this phenotype. The very attempt to distinguish these forces betrays a fundamental confusion over the involvement of genotype and environment in the development of organisms. This confusion has been recognized in some corners of hereditary scholarship since as early as 1909, although only a few have appreciated its ramifications for the study of human heredity (Woltereck 1909).

reverse genomics, where researchers can compare genomes for subtle differences in a gene allele across a number of individuals, develop the molecular product these alleles would produce, and then characterize the function of these molecules in larger metabolic processes. I call this reverse genomics because it represents a reversal of the technique of genetic reductionism. This reversal, however, does not upset the primacy of the genetic material for generating explanations of disorders. By starting with fully articulated genetic sequences this research builds on the assumption that the causal chain begins at the level of the genetic material and radiates through to higher levels of the organism.

Richard Woltereck studied a group of pure-bred, self-fertilizing crustaceans (*Daphnia*) by segregating these organisms into lines that shared the same genetic material (although it would be sloppy to call these organisms “clones,” since they were self-fertilizing they could be grouped into clusters that shared the same genetic material). He noted that each singular genotype developed the phenotypic trait under study in a particular fashion, in reaction to changes in particular environmental variables (Sarkar 1999; Woltereck 1909). These reactions across different genotypes were not uniform—in the presence of the same environmental situation, each genotype would generate a different growth pattern. Furthermore, the differences among these growth patterns were not linear. If we plot each series of identical organisms graphically, these growth patterns appear as *curves* (see Figure 3.1 below). Originally termed *Reaktionsnorm*, these complex, non-uniform relationships between genotype-phenotype-environment are now known as “norms of reaction” (Lewontin 1974; Sarkar 1999).

Figure 3.1. The Norm of Reaction. Here the X axis represents an environmental variable (temperature during growth), and the Y axis represents a phenotypic endpoint (head size of adult organism). Each line in the graph represents the norm of reaction for a number of organisms with the same genotype (G-1 and G-2). The curving lines illustrate the non-linearity of genotype-phenotype-environment interactions in complex organisms. For example, if organisms are raised at a temperature in the middle of the X axis, the two genotypes construct similar phenotypic outcomes. But at either temperature extreme, the phenotypes diverge. Only by tracking the development of each genotype through a series of environmental sequences is it possible to determine the norm of reaction for any given genotype.



The epistemological difficulty posed by the existence of norms of reaction, especially in the description of such complex systems of heredity and development as underlie human behavior, is that for any one phenotypic outcome, whether it be skin color or alcoholism, there can be many possible underlying norms of reaction, each generated by a specific spatiotemporal sequence of interactions between a single genotype and the environment in

which these genes operate.⁷⁶ Or, to put it differently, “all individuals owe their phenotype to the biochemical activity of their genes in a unique sequence of environments and to developmental events that may occur subsequent to, although dependent upon, the initial action of the genes” (Lewontin 1974, p. 401). The causal situation is bi-directional. Both environmental and genetic elements collude to produce patterns of development and growth that, when plotted together, are not simply additive, but curvilinear. An attempt to trace the causal chain backwards (so to speak), from a phenotype to underlying genotype without both strict, precise control of the temporal sequence of environmental exposures and with other genotypes for comparison is folly. Moreover, lumping together groups of organisms that possess the same phenotype but different genetic material produces a set from which no causal inferences are valid. Since humans generally do not possess identical genetic material, true analysis of the norms of reaction responsible for the production of a particular phenotypic trait is impossible. Absent a wholesale strategy for rethinking the way we analyze the intermingling of causes in developmental systems, claims to have found “the gene for” a complex human trait remain dubious.

2. What is a “Gene?”

Even the most detailed attempts to implicate genes as causes of human alcoholism rely on rough schematic accounts of the function of genetic material in the organism. But this is to be expected, since going beyond the schematic requires articulation of processes we are only beginning to understand. To illustrate this complexity and the non-genetic factors that are necessary participants in the function of genetic material we can focus on the

⁷⁶ See Hogben (1933) for a remarkably prescient illustration of these epistemological problems.

production of a single molecule (e.g., an enzyme) by the interaction of nuclear DNA and other cellular material.

BASE COUNT	448 a	317 c	417 g	431 t		
ORIGIN						
1	gggcatgggc	gcggccaccc	cggatgtcag	ccccccgcgc	cgaccagaat	ccgtgaacat
61	ggcgaacgag	gttatcaagt	gcaaggctgc	agttgcttgg	gaggctggaa	agcctctctc
121	catagaggag	atagaggtgg	cacccccaaa	ggctcatgaa	gttcgaatca	agatcattgc
181	cactgcgggt	tgccacaccg	atgcctatac	cctgagtggg	gctgacctcg	agggttgttt
241	tccagtgatc	ttgggacatg	aaggtgctgg	aattgtggaa	agtgttggtg	agggagttac
301	taagctgaag	gcgggtgaca	ctgtcatccc	actttacatc	ccacagtgtg	gagaatgcaa
361	atthttgtcta	aatcctaata	ctaacttttg	ccagaagata	agagtcactc	aagggaagg
421	attaatgcca	gatggtacca	gcagatttac	ttgcaaagga	aagacaattt	tgcattacat
481	gggaaccagc	acatthttctg	aatacacagt	tgtggctgat	atctctgttg	ctaaaataga
541	tcctthtagca	cctthtgata	aagtctgcct	tctaggttgt	ggcatttcaa	ccggttatgg
601	tgtctgtgtg	aacactgcca	agttggagcc	tggtctctgt	tgtgccgtct	ttggtctggg
661	aggagtcgga	ttggcagtta	tcatgggctg	taaagtggct	ggtgcttccc	ggatcattgg
721	tgtggacatc	aataaagata	aatttgcaag	ggccaaagag	tttggagcca	ctgaatgtat
781	taaccctcag	gatttaagta	aaccatcca	ggaagtgtc	attgagatga	ccgatggagg
841	agtggactat	tcctthgaat	gtattggtaa	tgtgaaggct	atgagagcag	cacttgaggc
901	atgtcacaa	ggctggggcg	tcagcgtcgt	ggttggagta	gctgcttcag	gtgaagaaat
961	tgccactcgt	ccattccagc	tggtaacagg	tcgcacatgg	aaaggcactg	cctthggagg
1021	atggaagagt	gtagaaagt	tcccaaagtt	gggtgtctgaa	tatatgtcca	aaaagataaa
1081	agttgatgaa	tttgtgactc	acaatctgtc	ttttgatgaa	atcaacaaag	cctthgaact
1141	gatgcattct	ggaaagagca	ttcgaactgt	tgtaaagatt	taattcaaaa	gagaaaaata
1201	atgtccatcc	tgtcgtgatg	tgataggagc	agcttaacag	gcagggagaa	gcgcctccaa
1261	cctcacagcc	tcgtagagct	tcacagctac	tccagaaaaat	agggttatgt	gtgtcattca
1321	tgaatctcta	taatcaagga	caaggataat	tcagtcatga	acctgttttc	tggatgctcc
1381	tccacataaa	taattgctag	ttataaggat	atttaacata	ataaaagtaa	ttctacattg
1441	tgtgaattgt	cttgthttatg	ctgtcatcat	tgtcacgggt	tgtctgcccc	ttatcttcat
1501	tctgcaagg	aaagggaagg	gaagcagggc	agtgggtggg	gtctgaaacc	tcagaaacat
1561	aacgttgaac	ththtaagggt	ctcagthccc	gthgattaaa	gaacagatcc	ccg

Table 3.1 DNA base sequence of allele for human alcohol dehydrogenase class III (ADH5). The “base count” refers to the number of times in the sequence that each base appears. Source: Manowitz, Poretz et al. (1998).

In Table 3.1 above we have a gene allele for the human form of the enzyme alcohol dehydrogenase-5. This enzyme facilitates one step in breaking down the ethanol alcohol molecule. At the outset it is important to keep in mind that there is far more involved in the

production of the enzyme than simply a sequence of bases⁷⁷ on a strand of the DNA double helix. The relevant strand of DNA containing the sequence must be “readable,” that is, have an “open reading frame” (ORF) where the surface of the molecule is accessible to other cellular machinery. The DNA chain must “unwind” itself slightly in order to provide space between the two strands of paired bases for other molecules to do their work of *transcribing* the DNA sequence into an RNA sequence. The RNA polymerase molecule binds to a site within this open reading frame and matches a base on the DNA to an RNA nucleotide and adds this to a messenger-RNA (mRNA) strand that is growing alongside the unraveled DNA sequence. This mRNA strand is released by the RNA polymerase molecule and freed to participate in *translation*, the process of constructing a series of amino acids bound together. In just these initial stages of the process the DNA is dependent on the existence and activity of the complex RNA polymerase molecule, of which there are three distinct varieties (in eukaryotes). This is what is meant by “cellular machinery”--there are a number of factors other than DNA that are necessary for making anything of the DNA sequence. In addition to RNA polymerase, the cellular context in which DNA is made useful includes agents like enhancers and promoters, and further processes such as formation and splicing of introns and exons. While the complexities of transcription and translation have been worked out sufficiently well to allow for a bevy of predictions, the amino acid molecules this process constructs undergo still further processes that currently defy precise prediction. The sequence of amino acids produced through translation will collect together and “fold” to form a polypeptide, or molecule that can eventually serve a biological function (such as an enzyme). Richard Lewontin articulates the problem this way:

⁷⁷ The amino acid bases of which the genetic material is comprised are paired as A-T, G-C (adenine and guanine, cytosine and thymine).

The full explanation of the path between gene and organism needs to include known phenomena that influence the way in which the string of amino acids coded by the gene becomes a protein, that is, a folded three-dimensional structure. The sequence of amino acids is insufficient to explain this folding, and there are many alternative stable folded states for any sequence, only one of which is the physiologically active protein” (Lewontin 2000, p. 115).

In the folding process this collection of amino acids may even discard some of its parts, while also picking up molecules produced by similar methods from other regions of the DNA strand. Finally, the folded molecule must be made available for interacting with other molecules, functioning essentially as an enzyme (Jablonka and Lamb 1995; Beurton, Falk et al. 2000).⁷⁸

In claiming to have discovered “the gene for enzyme x” discoverers must articulate what relevant cellular machinery *in addition to DNA sequence(s)* collude to produce the enzyme. Because there is considerable variance in the cellular machinery that is relevant to a particular set of products, there is complex heterogeneity of causes within the level of “genes” themselves, entirely aside from the causal heterogeneity of the larger biological systems in which the genetic material functions. Only by ignoring the intricacies of this causal situation at the molecular-genetic level can one claim to have found “the gene for” complex outcomes.

⁷⁸ See also Burian (1997) for aspects of the problems determining the relationship of genes to the development of organisms.

3. The Looping Effect of “Human Kinds”

Now I will shift from considerations at the molecular levels of phenomena to issues arising at a level we might call macro-sociological. Here I describe another vexing feedback-system that must also be taken into account if we are ever to construct a truly robust model of the role played by heredity in complex human behaviors such as alcoholism. Since the genetic accounts discussed above make no attempt to consider interrelationships at this level, the goal of a full account of alcoholism and heredity is clearly far from realization.

Ian Hacking has called attention to an aspect of the human condition that is capable of producing considerable epistemological difficulties for anyone attempting to explain it. He has named the problem the “looping effect of human kinds” (1995). He introduces “human kinds” as a particular type of meaningful classification. They differ from their philosophical counterpart (in epistemological talk, “natural kinds”) only in that they are subject to this looping effect (i.e., it may be possible to confuse natural kinds and human kinds). First let’s consider human kinds on their own terms and then see what use they may have for our analysis of problems in developing an understanding of the causes of alcoholism. Human kinds are defined according to the following four criteria; human kinds are

- (i) kinds that are relevant to some of us,
- (ii) kinds that primarily sort people, their actions, and behavior,
- (iii) kinds that are studied in the human and social sciences, i.e., kinds about which we hope to have knowledge,
- (iv) 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 (Hacking 1995).

For our purposes the last three of these criteria are most important. These criteria betray that these kinds are human creations, distinct from their more grounded counterparts natural kinds. They are historically contingent, but socially real kinds. That is, they constitute the classes in 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 (even though, in the short term, they may be confused with them).

Most interesting for our purposes is that human kind-designations, such as “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, while people often alter their behavior when they have been assigned to a kind designation—e.g., “labeled” as an alcoholic, or learning disabled, or depressed, and so on. But Hacking’s proposal goes beyond mere labeling theory in arguing that the kinds act on aggregate at more than an individual level, generating effects at institutional levels, thereby complicating our schemes of classification and the empirical studies reliant on them. That “alcoholism” exists as a category (or kind) 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 end-point ebbs and flows over time, and across geographic location.⁷⁹ But most importantly, the kind

⁷⁹ Alcoholism is diagnosed at a lower rate in Finland than the United States, although alcohol consumption per capita, and liver disease are both higher in Finland. To be sure, this may be the result of purely professional differences (e.g., how doctors are trained, which diagnostic criteria they prefer, etc.). However, differences like this point to something different about our collective views on acceptable behavior and the point at which one is considered deviant. Given the long historical avenue that generates and distinguishes our

classification can underwrite its own expansion, legitimacy, and objectivity. This is the looping effect.

As a human “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 force that can corrupt or influence any patterns observed among populations of humans so designated—even genetic studies. Although perhaps difficult to see at first, I maintain that awareness of such interlooping dynamics *at all levels of phenomena* is crucial to any successful resolution to the issue of alcoholism’s purported origins in heredity.

It may seem that I’ve outlined a largely negative project—criticizing accepted ideas and the links among them, while avoiding positive claims. This is not my intent. Of course, if my criticism does anything to unseat the gene-myth in relation to alcoholism and, by implication, other complex human behaviors, it will be welcome. But I have in mind a positive project also. From a philosophical perspective, the above historical episodes and reflections may supply a useful insight: despite technical progress in answering the questions posed by society’s problems over the past century, our preference for *posing only certain questions* requires consideration. Why, after all, are we fascinated with and accepting of 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 render me curious whether there is something deeper in the history of the enlightenment scientific project that privileges *simplicity* as both an epistemic and

views on deviance, doesn’t it seem odd to search for genetic differences between these two populations by which to explain the situation?

ontological virtue.⁸⁰ According to this assumption of simplicity, good theories are elegant, reductive, and account for a wide range of phenomena with the fewest, or simplest theoretical entities. Such explanations may have steered the course of inquiry 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 due.

One way to render the normal strange is to suggest plausible alternative explanations that account for the phenomenon in question, but without reliance on the usual causal factors. Such thought-experiments can aid in unseating flawed assumptions, or causal stories that have been for one reason or another become reified. In the next section I present an alternative account of personality and heredity, with a suggestion on how such an account could upset the gene-myth with respect to alcoholism. What is more, rather than introducing the obvious non-biological factors such as educational histories or peer social groups, this account relies on a purely biological foundation of parasite-influence. This thought experiment is meant to show that even thinking “biologically” there are clear alternatives to the genetic-reductionist explanation of human behaviors and deviance.

4. One Empirical Alternative: Parasites, Personality, and Alcoholism

How could findings about patterns of human behavior be complicated by parasite infection? A growing body of research suggests that infection by some parasites results in behavioral changes in the host organism (Barnard & Behnke 1990). Parasite influence on host behavior appears in situations in which parasites complete their life cycle in a predator host, but are capable of completing some phases of their life cycle in their host's prey. By

⁸⁰ Some initial steps in philosophical examination of the notion of simplicity in science can be found in Goodman (1972), Pitt (1990), and Rescher (1990).

altering the behavior of prey in ways that make them more prone to predation, parasites effectively increase their chances of completing their life cycle. I discuss two examples of this phenomenon, the first illustrating the functional utility of behavior alterations caused by such parasites, the second illustrating how one parasite in particular may directly alter human behavior. These examples suggest the existence of general non-genetic mechanisms for the alteration of human behavior (the body of research on human behavior discussed here is that corner of work focussing on personality). The role of these mechanisms needs to be explored in any research (such as that on alcoholism) that attempts to link behavioral / personality traits to socially problematic behaviors, on the one hand, or to underlying hereditary / genetic structures, on the other.

Our first example is *Dicrocoelium dendriticum*, a species of fluke that lives in many grazing mammals, but can also infect some insects. Infected ants, in particular, have been consistently observed climbing stalks of grass and clinging to the tip, where they are far more likely to be eaten by a grazing mammal than they would be on the ground (Zimmer 2000b). This behavior stands out against the background of normal ant behavior: constantly on the move. Once the infected ant is ingested by a mammal, the fluke can complete its life cycle in its host's gastrointestinal tract, reproduce, and disseminate its progeny through its host's feces. The mechanism for altering ant behavior in this case remains to be discovered. Nonetheless, the observable behavioral difference between infected and uninfected ants is so strong as to indicate parasite manipulation of the host organism with the advantageous result of furthering its own life cycle (advantageous for the parasite, that is).

The second example concerns a protozoan by the name of *Toxoplasma gondii* (Zimmer 2000a, 2000b; Flegr, Kodym et al. 2000). Infecting a wide range of mammals

(including bovines, rodents, and humans), this parasite completes its life cycle and reproduces only in the bowels of felines.

The *Toxoplasma* parasite lays oocytes (eggs) in its cat host that are deposited to the ground through the feces, where they await contact with a new host. From there, they find their way either into another cat, or to a mammalian host. When *Toxoplasma* oocytes invade a human host (through handling cat litter, eating undercooked meat from an infected animal, or even contact with the soil when gardening), the immune system mounts a forceful response causing the oocyte to form a thin-walled cyst around itself. The cysts appear in a wide range of tissues, concentrating particularly in the muscles and brain of the host.⁸¹

It has been shown that in rodents there are not only general differences of behavior between infected and uninfected animals (such as level of activity, Hutchison et al. 1980a; Hutchison et al. 1980b), but quite specific behavioral changes. In Berdoy and Webster's (2000) study, infected and uninfected rats were tested in a maze for avoidance of nests containing strong predator odors. The maze offered nests with choices of clean straw, straw with cat urine, straw with rabbit urine, and straw with rat odor. During their nocturnal explorations, the infected rats visited the cat-scented nest significantly more often than their uninfected counterparts, exhibiting no avoidance of the potential cat presence. Moreover, the infected and uninfected rats acted differently only in the cat-scented nests—their behavior in all other circumstances being nearly equivalent. Such specific alteration of predator-avoidance suggests direct parasite manipulation. Although the mechanisms of this manipulation are still obscure, the presence of parasite cysts in the host's brain has led

⁸¹ These cysts have been found to be life-threatening in only two circumstances: (i) cases of severe immunosuppression (e.g., AIDS), or (ii) early fetal development. In the first case death results from unchecked and widespread cyst growth that eventually disrupts the host's critical functions, while the result in the second case is spontaneous abortion.

researchers to postulate blocking of specific neurochemical receptors as one likely mechanism (Luft and Remington 1986 discuss details of *Toxoplasma* infection in the central nervous system).

The possibility of behavioral changes induced by *Toxoplasma gondii* infection in humans was first postulated in the 1950s (in this case, connections were sought between infection and “mental deficiency” Burkinshaw et al. 1953), and has seen renewed interest in recent years. Since the worldwide rate of infection in humans is relatively high (by a diversity of methods, estimated to be 84% in Paris; 22% in the UK; 20% in Finland; 27% in Denmark; 40% in southern Italy), researchers have clear warrant to investigate the extent and severity of possible behavioral effects (Desmonts and Couvreur 1974; Flegr, Kodym et al. 2000). Recent research has focussed on measures of personality differences between infected and control groups as a function of length of infection. The results are surprising, for they indicate that simple genetic accounts of some behaviors may have to be revised in light of the possibility of behavior caused by parasite infection.

Jaroslav Flegr performed studies of *Toxoplasma*-induced differences in men (Flegr, Zitkova et al. 1996) and women (Flegr, Kodym et al. 2000) using Cattell's personality questionnaire on infected and control groups. As reported in *Science* and *Biological Psychology*, the differences between the infected and control groups point to opposite effects for men and women (Flegr, Kodym et al. 2000; Zimmer 2000a). Table 3.2 illustrates the nearly opposite personality differences by gender. The strongest of the findings center on factors A, G, and Q3. *Toxoplasma gondii* would seem to lose the battle for the completion of its life cycle in humans, for detached, socially obtuse, impulsive men still run a rather small risk of being eaten by a cat. Equally bleak is the future of the parasite that renders its female host more outgoing and self-confident.

Sizothymia Reserved, detached, critical	A	<i>Affectothymia</i> <i>Warm-hearted, outgoing, easygoing</i>
Low intelligence	B	High intelligence
Ego weakness Affected by feelings, emotionally less stable	C	High Ego Strength Stable, mature, faces reality, calm
Submissiveness Obedient, mild, easily led, docile	E	Dominance or ascendance Aggressive, competitive, stubborn
Desurgency Sober, taciturn, serious	F	Surgency Enthusiastic, heedless, happy-go-lucky
Low superego strength Disregards rules, expedient	G	<i>Superego strength, character</i> <i>Conscientious, persistent, moralistic, staid</i>
Threctia Shy, timid, restrained	H	Parmia Adventurous, “thick-skinned”, socially bold
Harria Tough-minded, rejects illusions	I	Premisia Tender-minded, sensitive
Alaxia Trusting, accepting conditions, tolerant	L	<i>Protension</i> <i>Suspecting, jealous, dogmatic</i>
Praxernia Practical, has ‘down-to-earth’ concerns	M	Autia Imaginative, bohemian
Naivete Forthright, unpretentious	N	Shrewdness Astute, worldly, polished
Untroubled adequacy Self-assured, placed, secured	O	Guilt proneness Apprehensive, self-reproaching, insecure
Conservatism of temperament Conservative, respecting	Q1	Radicalism Experimenting, liberal, analytical
Group dependency Sociably group dependent, ‘joiner’	Q2	Self-sufficiency Self-sufficient, resourceful, prefers own decisions
Low self-sentiment integration Uncontrolled, lax, follows own urges	Q3	<i>High strength of self-sentiment</i> <i>Controlled, exacting, will power, socially precise</i>
Low ergic tension Relaxed, tranquil, torpid	Q4	High ergic tension Tense, frustrated, driven, overwrought

Table 3.2 — Cattell’s 16 Personality Factors (1970). Items in **bold** represent data for infected Males (Flegr, Zitkova et al. 1996); Items in *Italics* represent data for Females (Flegr, Kodym et al. 2000).

How is this parasite capable of producing measurable personality differences in humans? The mechanism is presently unknown. When the immune response causes the parasitic oocyte to seclude itself inside cysts in the host's brain their mere physical presence might interrupt specific neural pathways, blocking the work of certain neurotransmitters and receptors. Unfortunately, differences between rat and human neurophysiology only render the problem more complex. I must put aside the question of the mechanism for now and focus instead on the implications of these parasite-induced behavior changes for research programs in the human sciences.

Parasite-induced behavior can act as a third cause, complicating correlational studies of personality traits and hereditary patterns. *Toxoplasma gondii* cannot be transmitted hereditarily, for early infection of the human fetus results in spontaneous abortion. Infection can, however, tend to run in families. Only after birth, through contact with the contents of a litter box, infected soil, or raw meat, can humans be infected. But such infections are likely to go unnoticed, having as benign of an effect on the well-being of human hosts as they do. These silent infections can be concentrated geographically and, I propose, even familialy. Since many people keep cats as pets, interacting daily with a possible *Toxoplasma* host, there is a good chance that entire households can be affected. These family-centered infections, while inconsequential for normal behavior, hold great potential for skewing hereditary-behavioral research in a given pedigree. Instead of correlating sex-linked patterns of behavior in a human pedigree with genetic markers on the Y chromosome, researchers could actually be correlating hidden infections with otherwise unrelated patterns in behavior (at one level) and genetic composition (at another level). Making causal inferences between these two levels without controlling for the possibility of influences by third causes in this intermediate level will produce at best an incomplete

picture of the human situation. At worst, such efforts will further entrench the notion that genes are directly responsible for specific features of our brains like personality, disposition, and musical ability.

With respect to alcoholism, the possibility of parasite-confounded personality alterations needs to be explored. For decades alcoholism researchers have made attempts to define, measure, articulate and subcategorize the “alcoholic personality.” Some of these studies have employed Cattell's personality questionnaire. For a crude exploration of similarities between *Toxoplasma* infected personality traits and alcoholic personality traits, we can refer to a meta-analysis of alcoholic-personality research (Spotts & Shontz 1991) and compare it against the patterns exhibited in Table 3.2. The effectiveness of this comparison is hampered by the lack of gender controls in the alcoholic-personality research—a problem only magnified by the gender-polarized results for *Toxoplasma*-infected subjects. Also problematic are the wide-ranging criteria used to select the sample of “alcoholics” for personality study.⁸²

⁸² The question whether there is an “alcoholic personality” is problematic on its own terms. Only in the last two decades have researchers (but clearly not the popular press) come to think of the alcoholic personality as a misguided notion. Attention is instead focusing on the distinction between the “natural history” of alcoholism as a disease in isolation from individual “drinking careers”—of which personality factors arguably play a significant, if mysterious, role.

Sizothymia Reserved, detached, critical	A	<i>Affectothymia</i> <i>Warm-hearted, outgoing, easygoing</i>
Low intelligence	B	High intelligence
<u>Ego weakness</u> <u>Affected by feelings, emotionally less stable</u>	<u>C</u>	High Ego Strength Stable, mature, faces reality, calm
<u>Submissiveness</u> <u>Obedient, mild, easily led, docile</u>	<u>E</u>	Dominance or ascendance Aggressive, competitive, stubborn
Desurgency Sober, taciturn, serious	F	Surgency Enthusiastic, heedless, happy-go-lucky
Low superego strength Disregards rules, expedient	G	<i>Superego strength, character</i> <i>Conscientious, persistent, moralistic, staid</i>
<u>Threctia</u> <u>Shy, timid, restrained</u>	<u>H</u>	Parmia Adventurous, “thick-skinned”, socially bold
Harria Tough-minded, rejects illusions	I	Premisia Tender-minded, sensitive
Alaxia Trusting, accepting conditions, tolerant	<u>L</u>	<u>Protension</u> <u>Suspecting, jealous, dogmatic</u>
Praxernia Practical, has ‘down-to-earth’ concerns	M	Autia Imaginative, bohemian
Naivete Forthright, unpretentious	N	Shrewdness Astute, worldly, polished
Untroubled adequacy Self-assured, placed, secured	<u>Q</u>	<u>Guilt proneness</u> <u>Apprehensive, self-reproaching, insecure</u>
Conservatism of temperament Conservative, respecting	Q1	Radicalism Experimenting, liberal, analytical
Group dependency Sociably group dependent, ‘joiner’	Q2	Self-sufficiency Self-sufficient, resourceful, prefers own decisions
Low self-sentiment integration Uncontrolled, lax, follows own urges	Q3	<i>High strength of self-sentiment</i> <i>Controlled, exacting, will power, socially precise</i>
Low ergic tension Relaxed, tranquil, torpid	<u>Q4</u>	<u>High ergic tension</u> <u>Tense, frustrated, driven, overwrought</u>

Table 3.3 — Alcoholic personality data shown as underlined items (Spotts & Shontz 1991); Items in **bold** represent data for infected Males (Flegr, Zitkova et al. 1996); Items in *Italics* represent data for Females (Flegr, Kodym et al. 2000).

Nevertheless, in Table 3.3 the alcoholic-personality trends have been added. From this crude comparison, the lack of overlapping affected variables suggests that we need not worry that parasite infection could be operating in the fashion of a third cause between personality features (as measured by Cattell's questionnaire) and alcoholism (as measured by hospitals, jails, and clinics in which the inmates were housed).⁸³

In summary, what this single example of fallout from research on parasite infection makes clear is that there are other features of biological systems that can generate effects at the level of behavior, comportment, and personality and whose appearance in patterns through family history can be mistaken for hereditary factors. Consequently, studies of complex traits that attempt to move from personality measures to underlying hereditary or genetic causes need to consider and control for more proximate influences of personality traits such as the parasite infections considered here. Only with a robust model of the complex labyrinth of systems that constitute an adult human being can we ever hope to move to describing likely sources of such subtle features of their behavior as personality or the desire to drink. Since we currently lack this robust model, time is better spent actively investigating all the relevant systems (which requires first establishing means of determining relevancy) that might ultimately play a constitutive role in the model. Considerable waste occurs in one-dimensional speculations about the genetic “cause” of the behavior in question. If we are to make any progress in putting flesh on the physico-chemical bones of

⁸³ These sorts of caveats are necessary when toying with data from different sources. It should be noted that even the criteria Sottz and Shontz (1991) relied on in their meta-analysis of drug use allows considerable room for corrupting influence by other factors. For instance, do the subjects have other more central issues that land them in jail or clinics, issues that affect their use of alcohol? These are the types of questions that need to be addressed when pursuing these lines of explanation. In constructing this example, however, I have attempted to stay true to the form of analysis and writing that sadly characterizes much of the social scientific endeavor to understand alcohol-related behavior.

hereditary science, we need to consider the human subject in more complex terms than those such as Dawkins' gene-carrier model where "genes are the replicators and we are their survival machines."⁸⁴

5. Darwinian Medicine

The remainder of this chapter considers in detail the latest formulation of an evolutionary and genetic model for understanding alcoholism, and the impetus behind it. The Darwinian Medicine paradigm⁸⁵ holds that biomedical science, and even clinical medical practice, will gain new power to explain aspects of human disease by application of an evolutionary perspective in which natural selection and adaptation function as explanatory elements. The explanatory power boasted from this approach is claimed to derive from previously unpredicted mechanisms underlying disease that become obvious when researchers articulate the evolutionary context in which the disease or disorder first arose.

Generally speaking, "adaptation" is a controversial term. Debates continue over both the proper definition of the term, and, more to the point, over the difference between *ad hoc* construction of "just-so" stories and properly supported claims of pinpointing a particular

⁸⁴ Dawkins' model is the epitome of the gene-myth. "Individuals are not stable things, they are fleeting. Chromosomes too are shuffled into oblivion, like hands of cards soon after they are dealt. But the cards themselves survive the shuffling. The cards are the genes. The genes are not destroyed by crossing-over, they merely change partners and march on. Of course they march on. That is their business. They are the replicators and we are their survival machines. When we have served out purpose we are cast aside" (Dawkins 1976, p. 35).

⁸⁵ When speaking of Darwinian Medicine throughout this dissertation I am concerned not with a specific group of researchers or institutions—my focus is rather with the methodological and theoretical commitments central to the *paradigm* that underlies a specific set of attempts to apply evolutionary theory to modern medical and psychological problems. For various articulations of this paradigm and its application to specific problems, see Dudley (2000); Williams and Nesse (1991); Nesse (1999); Nesse and Williams (1994); Nesse and Berridge (1997); Nesse (1990); McGuire and Troisi (1998); Nesse (1984).

evolutionary process. Further complicating matters is the recognition that adaptation can apply to events in evolution at three distinct but complexly interconnected levels: genes, organisms, or groups (Brandon and Burian 1984; Burian 1992). Although genomes, organisms, and populations are interrelated sets, selection can favor, separately, expression at any one of these levels. Consequently, only those adaptive stories for traits that specify the level or set in which the adaptive trait resides warrant our attention.

Consider an example. Over the course of thousands of years in an environment with little stability, selection may promote greater genetic diversity within a species, thereby allowing the species greater chances of survival in the face of drastic changes. Variance at the level of the population's genome could be the adaptation here. But just as easily selective pressures might have led to other changes in the organism, such as rapidly maturing reproductive organs, allowing each generation to keep pace with environmental changes. A change in age of reproductive maturity can take place without affecting overall genomic diversity. In this scenario the adaptation would be a feature of organisms, not genomes. This illustrates that when speaking of "adaptations" in an evolutionary context, one must specify both the level at which selective pressures have written their effects, and the course of evolutionary events by which they were written.⁸⁶ Without presenting a consideration of these factors, valid accounts of adaptations cannot be separated from *ad hoc* evolutionary stories for modern traits.⁸⁷ This requirement is often left unfulfilled by adaptation stories proffered by Darwinian Medicine writers.

⁸⁶ For some ways of meeting this explanatory requirement, see Kaplan and Pigliucci (2001).

⁸⁷ For more detailed accounts of the conceptual requirements of adaptation and the adaptationist program, see e.g., Gould and Lewontin (1979); Mayr (1983); Reeve and Sherman (1993); Sober (1984).

In the flagship article introducing the Darwinian Medicine paradigm, Williams and Nesse (1991) describe adaptation as follows:

An adaptation is some sort of biological machinery or process shaped by natural selection to help solve one or more problems faced by the organism.

The phenomenon may be interpreted as a necessary component of the imagined machinery, or as an unavoidable manifestation of its operation....

This search [for adaptations] can start either with a trait or a function (p.3).

It would be difficult to build an empirical program of research on as vague a definition of adaptation as this. But as I will show, when Darwinian Medicine is applied to a specific disease or illness it is easier to pinpoint how the term functions. To date, a wide range of human disease patterns have been supplied with evolutionary origin-stories according to this paradigm—including human emotions, depression, substance abuse, eating disorders, breast cancer, Alzheimer's, malaria, even rape, and most recently, alcoholism (Nesse 1984; 1994; 2000; Williams and Nesse 1991. By focussing on particular types of disorders and phenomena, some writers have developed distinct explanatory models. Williams and Nesse (1991) have articulated four general domains of disease phenomena that are amenable to adaptationist interpretations.

a. Infection.

In bacterial and viral infections, selection affects pathogens during their reproduction within the host organism. Bacteria and viruses reproduce and often mutate at rates faster than the systems they attack. Some of the human body's defense mechanisms can be compromised or simply outpaced by the rate of mutation of the infecting organisms.

Therefore, some common symptoms, defense mechanisms, and signs (cough, fever, swelling, etc.) may be evolutionary relics—defensive measures aimed at organisms once susceptible to the tactics, but now are capable of evading the defense. Although it would be a complex task, a program of research could establish which defense mechanisms are outdated for particular infections, providing new insights for clinical practice and pharmaceutical development. One component of this focus on infectious disease deals specifically with *parasites*. Evolution by natural selection is clearly a significant force shaping host-parasite relationships. Here, the evolutionary interrelationships of the host and parasite life-cycles and immune responses can be investigated to provide insights leading to new strategies for defense.⁸⁸

b. Injuries, Breakdowns, and Toxins.

The Darwinian hypothesis for these phenomena holds that the body's response to mechanical damage (sprains, broken bones, bruises) may constitute evolved strategies for repair. For example, swelling of a sprain may serve in some way to immobilize and further protect the joint. As a result, attempting to reduce swelling may be unnecessary, or worse, counter-productive. Investigation of possible evolutionary adaptations for such bodily responses to damage (including pain) may thus shed light on which (if any) modern methods of treatment are in need of revision, or could be exploited for new treatment strategies. Similarly, bodily response like nausea, allergy, and diarrhea after exposure to a toxin suggest encounters with toxins in our evolutionary past. Investigations of the mechanisms underlying these toxin-responses could provide insights into how we might exploit existing

⁸⁸ This line of work has been growing recently, with no small degree of success. See, e.g., Hamilton, Axlerod, et al. (1990); Moore and Gotelli (1996); Zimmer (2000); Poulin, Morand

defense mechanisms for the novel toxins (of our own creation) that we encounter today. Some current attempts to deal with toxins in evolutionary context have postulated chemical hormesis as a generalizable adaptive response in living systems (Calabrese and Baldwin 1998; Parsons 2000). I will return shortly to the specific hypothesis that situates the origins of alcoholism in the evolution of hormetic responses to ethyl alcohol in humans.

c. Genetic Factors.

Widespread genetic abnormalities could, like sickle-cell anemia, be the outcome of adaptations to evolutionary environments. In a modern context, however, many such abnormalities are no longer adaptive. Furthermore, selective forces may have given us genes that are beneficial early in life, but also cause serious complications later in life. On the basis of the recognition that genes rarely serve singular purposes, Darwinian Medicine proposes that some genes linked to diseases of senescence may play important roles in basic development much earlier in life. Therefore, an adaptationist perspective on senescence could affect the future course of research into disorders like Alzheimer's, for if research could establish that such "double-edged" genes were responsible, then straightforward genetic solutions to these disorders may no longer be feasible. Altering the negative effects of these genes might concomitantly negate the positive aspects of their expression, unless the change could be restricted to the desired portion of the life cycle.

d. Abnormal Environments.

The Pleistocene savanna is thought to be the physical environment associated with evolutionary adaptations for modern humans (Williams and Nesse 1991, p. 13). This

et al. (2000).

environment differed considerably from the modern human environment, in aspects including population size, geographical isolation, family structures, reproductive age, availability of nutrients, dietary constraints, energy expenditures, and so on. If modern humans are adapted to an evolutionary environment in which an essential nutrient or vitamin was scarce (e.g., vitamin D prior to the advent of large-scale dairy farming and supplementation in milk), they may have developed metabolic mechanisms for manufacturing them. But when situated in the modern context where the nutrient is widely available, its abundance may contribute to diseases or otherwise negative outcomes (e.g., obesity in environments with abundant dietary fat, or possibly alcoholism in the presence of a new abundance of fermented and distilled spirits). In this way many diseases may be nothing more than “diseases of civilization”—brought about by the stark difference between modern and evolutionary environments. Diseases thought to stem from this mismatch of environments include “atherosclerosis, breast cancer, substance abuse, eating disorders, and probably depression” (Nesse and Williams 1999, p. 21).

I turn now to the task of reconstructing the arguments that tie this general Darwinian Medicine paradigm to the quest for an evolutionary explanation of alcoholism in frugivory (‘frugivory’ refers to feeding in large proportion on fruits). I limit my comments here to the last of these four domains, for the frugivory hypothesis rests mainly on the mismatch of evolutionary and modern environments. The above review of Darwinian Medicine’s is intended to exhibit the scope and methods of the approach, and to make it clear that there are certainly reasonable applications of evolutionary reconstructions to matters of human disease and infection. Alcohol is not an infection, injury, or breakdown,

but its role as a toxin does figure into the frugivory hypothesis (as it has in previous theories of alcoholism's origin by specific toxic effects of alcohol exposure in individuals).⁸⁹

The first line of the abstract for the article entitled "Evolutionary Origins of Human Alcoholism in Primate Frugivory" (Dudley 2000) claims that "Evolutionary origins of alcohol consumption have rarely been considered in studies of ethanol addiction" (p. 3). This is misleading. Since the publication Darwin's *Origin of Species* (1859), there has been a continuous and prolific intellectual commitment to the idea that many social problems (alcohol addiction, or "inebriety" chief among them) stem from hereditary sources.⁹⁰ Hereditary theories imply an understanding of evolutionary considerations. Dudley's claim therefore misleads. Insofar as authors advance theories on alcoholism's *hereditary* origins after the initial reception of Darwin's theory of evolution, they attribute the modern manifestation of the disorder to some aspect of *evolution*. The differences between these theories lie in which particular evolutionary forces are invoked to account for an observed hereditary pattern. Dudley's claim would more accurately be articulated as "seldom have authors attempted to described precisely in what circumstances human ancestors encountered ethyl alcohol and developed adaptive behaviors (and subsequent physiological modifications) to take advantage of it." Speculations on the evolutionary origins of alcoholism are by no means novel. Other writers within the Darwinian Medicine paradigm have made exactly such attempts to deal with the possible evolutionary origins of alcoholism and substance abuse (Nesse and Berridge 1997; Nesse and Williams 1994).

⁸⁹ One early claim that alcoholism resulted solely from the toxic effects of exposure to alcohol was advanced by Leslie E. Keeley in *The Non-Hereditary of Inebriety* (1902). Since then, this claim has appeared as a component of more sophisticated theories on the etiology of alcoholism.

⁹⁰ For some historical perspective on this intellectual trend see Degler (1991); Kevles (1985); Lewontin (1992; 2000); Ludmerer (1972); Sournia (1990); Valverde (1998).

On the one hand, Dudley's is a welcome attempt to articulate an evolutionary setting that could have served as a selection mechanism acting on genes that somehow function to induce preference for alcohol consumption—welcome because discussion of *specific* mechanisms is decidedly rare in the last century of writing on the role of hereditary in alcoholism. On the other hand, with close examination even this attempt is not specific about the mechanisms involved, leaving room for more detail and elaboration. In order to examine the Frugivory hypothesis more closely, I have reconstructed three distinct but overlapping arguments that appear in publication (Dudley 2000), and the considerations on which they rest.

H1. “Behavior and Sensation”

- a. Hominids and many other animals regularly ingest ethanol in the course of frugivory.
 - 1. Ethanol exists in trace amounts in the husks of ripe and over-ripe fruits.
- b. There are probably ways of detecting the presence of ethanol in fruits.
 - 1. Some other animals (*drosophila*) locate plumes of vapor from fruit, of which ethanol is usually a component.
 - 2. Olfactory sensory localization of ethanol may have occurred in hominid ancestors, as it occurs in our closest living cousin, the chimpanzee.
- c. These methods of detecting ethanol's presence may have been selected for over the history of the hominid lineage.
- d. There are now molecular pathways common to frugivorous taxa for sensing the presence of ethyl alcohol in the environment

Hypothesis #1. One important feature of alcoholism in modern humans may be the “co-option of molecular pathways and sensory biases common to many frugivorous taxa” (Dudley 2000, p. 7).

H2. “Metabolism, Nutrition, and Abnormal Environment”

- a. Hominids and many other animals regularly ingest ethanol in the course of frugivory.
 - 1. Ethanol exists in trace amounts in the husks of ripe and over-ripe fruits.
- b. Frugivory probably developed as a major nutritive strategy among anthropoid taxa sometime in the late Eocene.
- c. Based on reconstructions of evolutionary habitats, it is likely that our hominid ancestors developed frugivorous feeding strategies sometime in the Pleistocene.
- d. Modern humans exhibit variable rates of metabolism of ethanol and its immediate metabolic products, variations that are correlated with the presence of variant forms of the enzymes alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH).
- e. Ethanol has caloric value, potentially imbuing it with nutritive reward.
- f. Variance in metabolism of some toxic substances may be the outcome of natural selection acting on specific sub-populations with greater exposure to a toxin so as to minimize its harmful effects.
- g. Natural selection may have favored the evolution of (hormetic) metabolic adaptations that maximize physiological benefits and minimize any costs associated with ethanol ingestion in the hominid lineage leading to modern *homo sapiens*.

Hypothesis #2. Alcoholism in modern humans stems partly from the existence of (possibly hormetic) metabolic mechanisms previously selected in evolution to maximize the nutritive benefit of ethanol in small doses, but which now operate in an environment in which ethanol is available in abundance.

H3. “From Genes to Behavior”

Hypothesis #3. “Genetically-based behaviors adaptive in the ancestral environment become disadvantageous in a modern human environment that provides *ad libitum* access to nutritional substrates, including ethanol” (Dudley 2000, p. 7).

Now that we have in hand a general outline of the Darwinian Medicine paradigm and the Frugivory hypotheses, I will return briefly to the continuity in the history of the quest for an evolutionary explanation for alcoholism.

6. Evolutionary v. Historical context

As I stated earlier, there is promise for increasing our understanding of the social status of modern alcohol use by looking to the history of previous attempts to classify and study it.⁹¹ While the Darwinian Medicine paradigm and the Frugivory hypothesis seek *evolutionary context* for enhancing our understanding of psychological disorders, there is considerable *historical and cultural context* ignored in these accounts. The degree to which our behaviors in the presence of alcohol may depend on the traditions, rituals, and habits in the roughly twenty centuries following the Pleistocene is not addressed by such evolutionary reconstructions as the frugivory hypothesis. Given the great flexibility of human forms of life and custom, the development of human exposure to and use of substances containing alcohol in this great stretch of time is clearly significant for any attempt to understand the current state of affairs surrounding alcohol problems. Instead of reaching back to our

primate ancestral line for an explanation of modern alcoholism, we would be better served by a careful appraisal of the place of alcohol and intoxication in our rich cultural heritage.

7. Continuity between Darwinian Medicine and historical approaches.

One significant historical parallel with the Darwinian Medicine approach to alcoholism can be seen in the aforementioned work of British physician G. Archdall Reid, who speculated on possible evolutionary and hereditary roots of ethnic distributions of diseases, predisposition to rare deformities, and even geographical distribution of immune response to common infections (Reid 1906b; recall chapter 2 for more detail on Reid's life and work). Reid hypothesized that alcohol problems ultimately arise from the forces of natural selection in our evolutionary past. Written just months prior to the rediscovery of Mendel's analysis of trait transmission, Reid's book on alcoholism describes the "natural history of man" and performs comparisons of "racial differences" in the preference for alcohol. Over many generations exposure to alcohol would act as a filter, limiting the reproductive success of those whom it rendered unfit. In this way exposure to alcohol could produce a population nearly invulnerable to the problems associated with alcohol use, essentially *outbreeding* alcoholism in that population. Reid tried to relate this possible selection effect to the worldwide patterns of alcohol problems in different populations, a task that still concerns public health research today.

Darwinian Medicine's explanatory aims parallel Reid's to a point in that they attempt to situate present-day patterns of pathology, disease, infection, and behavior within the context of heredity—i.e., scientific reconstructions of human evolutionary origins, past

⁹¹ See Nye (1984, esp. Chapter 1) for a compelling account of the relevance of broad-scope history for modern medical, psychological, and criminal designations. Also relevant to this topic is Maienschein (2000).

environments, and adaptations. The difficult task for modern society is to determine responsibly and fairly whether public health policy should be conducted on the basis of evolutionary theory.

8. The Genetotrophic Theory

Another interesting landmark in the quest for evolutionary accounts of alcoholism is Roger J. Williams's "genetotrophic" theory of the hereditary origins of alcoholism that he advanced in the 1940s and 1950s (see e.g., Williams 1947; 1949; 1959). Within this theory, alcoholism was defined as a nutritional deficiency, stemming ultimately from (unspecified) metabolic features unique to alcoholics. This focus on metabolism and nutrition was part of a larger trend towards biochemical analysis of illness in the middle of the twentieth century. The biochemical and nutritional analysis of metabolism built on the work of Archibald Garrod's theory of "inborn errors of metabolism" (1909), and his application of the theory to the disease Alkaptonuria. Garrod argued for the "metabolic specificity" of individual human digestive and metabolic systems. Garrod's work supported the supposition that the metabolic system was ultimately susceptible to genetic influence that could render one vulnerable to specific substances. Building on this foundation, Williams argued that there were genetic controls of metabolic systems and therefore must be a unique configuration of the alcoholic's metabolism that contributed to the disease. To support his theory, Williams invoked examples of human cravings after being deprived of essential nutrients, behavioral effects of some vitamin deficiencies, and rodent models of alcohol withdrawal symptoms. As a result of this focus on nutritional requirements, metabolism, and digestion, Williams's picture of alcoholism underscored the role of heredity in developing the alcoholic's metabolic individuality. In the genetotrophic theory, alcoholism was identifiable by the

appearance of cravings, and the loss of control over drinking quantity, frequency, and duration.

Ultimately, the genetotrophic theory of alcoholism suffered at the hands of pointed criticism, even if it was not without its followers. Popham (1953) attacked the theory on a number of levels, including the lack of evidence that alcohol functioned as a nutrient in humans and the lack of an identifiable biochemical pathway by which a gene (or suite of genes) could alter metabolism so as to depend on alcohol for nutrition. Popham's criticism focussed on the fact that, unlike Garrod's work, Williams' theory did not have roots in empirical observations of disease patterns, *deducing* metabolic mechanisms from them. Rather, the genetotrophic theory of alcoholism relied on *analogy* with other metabolic conditions—suggesting biochemical mechanisms for alcohol metabolism as similar to known metabolic mechanisms for other molecules, instead of establishing these mechanisms empirically. This methodological criticism was well placed, and Williams subsequently found a focus in the more practical endeavors of treatment and counseling.

As I will show, the genetotrophic theory shares much with the *nutritional hypothesis* (Hypothesis #2) advanced as part of the Frugivory hypothesis, including its weaknesses in dealing with genetic controls of alcohol cravings.

When compared with these precursors, what seems to be new about the Darwinian Medicine paradigm is the attempt to trace the biochemical and metabolic individuality in modern humans to *specific events* or *environments* in our evolutionary history that may help account for their presence. But in doing so, Darwinian Medicine accepts wholesale the genetotrophic view of human metabolism and biochemical individuality, attempting to support it with added speculations on its sources in evolution. The admitted lack of empirical studies to support the hypothesized selective importance of alcohol in our

ancestral lineage is a weakness that threatens the viability of this extension of the genetotrophic view. Given the history of hypothesis and criticism in the case of the genetotrophic theory, it is unclear why proponents of Darwinian Medicine are at pains to claim to have such a novel perspective.

9. Weaknesses of Darwinian Medicine & the Frugivory Hypothesis

In order to see the depth of the problems facing the Darwinian Medicine paradigm we need to examine the arguments for the Frugivory hypothesis closely. In what follows I describe difficulties that stem ultimately from evidential weaknesses, before moving on to deeper epistemological problems that plague the model of alcoholism and heredity on which the account rests.

The most sweeping claim made in the three hypotheses is that of **H3**—“*Genetically-based behaviors* adaptive in the ancestral environment become disadvantageous in a modern human environment that provides *ad libitum* access to nutritional substrates, including ethanol” (Dudley 2000, p. 7 italics added). This is simultaneously the most uncontroversial and the most problematic of the hypotheses. The phrase “genetically-based behaviors” implies a commitment to a missing premise: Genes influence behaviors in humans. On the one hand, this premise is trivially true and uncontroversial. There are undoubtedly genes without which a human could not behave as a human (such as genes involved in the development of the nervous system generally, and the brain specifically). But the phrase is not used with this general scope—it is specific: There are genes connected with preference for and consumption of certain nutritional substrates like ethanol. This is the sort of claim that can be supported or weakened with empirical evidence. But until such evidence is

collected, the third hypothesis rests solely on the evolutionary reconstructions expounded in the first two.

Recall the first argument and hypothesis **H1** (i.e., One important feature of alcoholism in modern humans may be the “co-option of molecular pathways and sensory biases common to many frugivorous taxa” Dudley 2000, p. 7). This is an attempt to establish a connection between sensation and perception of ethyl alcohol and “molecular pathways” in our evolutionary lineage. But where do these molecular pathways reside? Is it our olfactory sense organs that exploit these molecular pathways? Or is it rather a feature of our nervous system, perhaps a “module” hard-wired in the brain? Since the seat of all behavior is arguably in the brain, the first and third hypotheses depend on the notion that alcoholism is an outcome of a particular type of brain, or a module within the brain. Furthermore, these quirky brains are built by a particular suite of genes that increased fitness and became subject to natural selection. This assumption of a linear connection between genes, brains, and behaviors is the source of the greatest weakness in **H1** and **H3**.

The compelling argument against such linear genetic explanations of alcoholism follows the recognition that genes are individual components of complexly organized biological systems comprised of many levels of interacting components. The role of genes in these systems is often neither linear nor unidirectional—there is feedback between system components such that properties exhibited by the system as a whole (loosely “the phenotype”) cannot be reduced to the action of a single component of the system. Even at the level of individual alleles and the production of cellular products (amino acids, proteins, enzymes) the situation is one of feedback and non-linear cascades of events. Possessing a particular genetic sequence of bases is not sufficient to produce an enzyme, for example, for there is a host of cellular machinery that must be in place before translation and transcription

of the base-sequence can begin (Fogle 2000). As a result, the phrase “the gene for x” can mean different things to researchers with different areas of focus. Molecular biologists can view genes as clusters of DNA sequences and the necessary cellular machinery for their expression, while behavior geneticists focus on the statistical correlations between operationalized behaviors and gene markers or DNA sequences, considered without any well-defined cellular context.

This heterogeneity of gene concepts in theory and practice cannot be ignored in any philosophical analysis of the physiological features offered as explanations of any complex human behavior. While there is growing consensus among philosophers of biology that many fundamental concepts in biology are shifting in the face of greater access to the genomes of a number of organisms, there is no such consensus concerning the direction of such conceptual transitions. The perspective emerging from the study of gene concepts is that the gene concept in the future will be a plurality of contextually-restricted concepts rather than a single concept of “the gene.”

Emerging as one promising perspective is a reconceptualization of genes, organisms, and environments as interdependent and interactive components of a single system (Heather and Robertson 1997; Oyama 2000, esp. chapter 1). This “systems view” applied to alcoholism at a basic level holds that there are properties of systems that cannot be reduced to the properties of individual components of the system (McClean 1993). Applying this lens to alcoholism in humans, one way to conceptualize the system would involve labeling its principal features, e.g., “alcoholic human organisms.” Components of the system can be arranged under these three terms.

The “alcoholic” portion of the system includes observational and clinical settings, including such diverse elements as law enforcement, hospital emergency wards, psychiatric

clinics, rehabilitation clinics, counseling centers, and so on. In all these settings the act of *diagnosing* alcoholism takes place, which in itself is a result of the interactions between institutions and individuals (see Duster and Garrett 1984; Conrad and Schneider 1980). For instance, consider the case of a man arrested after attending a Washington Redskins football game at which he consumed a number of drinks with friends from work. While an arrest for “public intoxication” for loudly celebrating on the Metro ride home would be viewed by addiction professionals as evidence of possible alcohol dependence, the evidence is a result of a complex interaction. The interacting elements here are the law enforcement personnel who made the arrest (who may not be Redskin fans), the policies they follow (e.g., boisterous celebrations may be overlooked in the parking lot of the stadium, but not on the Metro), the letter of the law (e.g., the magistrate’s interpretation of statutes articulating conditions for both “public” and “intoxication”), and the relative socio-economic status of all of these individuals (e.g., the fact that our football fan is a landscaper from a nearby suburb, rather than a corporate heavy who watched the game from a box and consumed the same quantity of beer, will affect outcome of the interface between the individual and the police officer who that serves as a *de facto* representative of the state). Furthermore, this hypothetical incident will enter as a statistic into public policy evaluations of alcohol consumption that compare availability and cost of alcohol against arrests. In this respect our hypothetical arrest *follows from* previous public policy analysis (which may have supported availability of alcohol at public sporting events in the state) and also impinges on the continuance of such policies. E.g., in response to increasing arrest rates, public officials could recommend either excluding alcohol sales in the stadium, or altering tacit policies affecting action by law enforcement. In both cases there would be noticeable effects on the reported rate of alcohol-related arrests, as well as considerable differences for the conduct of

individual behavior. Such effects do not have biological origins, rather, they are contingently related features of the individual-society interrelationship. The simple fact of the matter is that the incidents included as evidence of alcohol-related behavior are outcomes of complex interactions between individuals and other individuals that represent an institution, profession, or state. As such, specific features of these institutions, profession or state can mask or amplify patterns that appear in the data they collect about individuals. Another component of this portion of the system is the diagnostic criteria and the clinical, historical, and scientific background against which it is constructed. Thus the *Diagnostic and Statistical Manual* (DSM-IV) criteria for the alcohol dependence figure as interactive components of this portion of the system. The designation “alcoholic” is a placeholder for the confluence of forces that bring an individual into interaction with institutional measurement systems, not depiction of a purely biological trait.

I’ve chosen to use two separate terms “human” and “organism” as individual components of the system as a way of cleanly distinguishing the history of our particular category of organism from features common to many organisms. This is useful because in this way we can parse the biochemical, genetic, and physical systems of which many organisms are composed (filed under “organism”) from the unique genetic-historical ancestry and subsequent environmental, cultural, and behavioral circumstances which to a large extent constitute what it is to be “human” (i.e., *homo sapiens* and some identifiable lineage of hominid ancestry).

According to this abbreviated introduction to a systems view of alcoholism, information garnered from the study of alcohol-related behaviors and metabolism in *drosophila* and rodents can give us insight into the “organism” components of the system, insofar as they allow us to model biochemical pathways that reasonably can be assumed to

function similarly in modern humans. But even this endeavor is not without its problems. Researchers have noted that an actual animal model of *human* alcoholism does not exist, and cannot seemingly be generated.⁹² In the lab, animal models of alcoholism are limited to exhibiting limited aspects of human alcoholism such as liver damage, withdrawal sickness, vitamin deficiency and preference for alcohol over another nutrient or toxin (see for example McMillan 1997; Mello 1973).

We can also investigate the lineage of hominids leading up to modern *homo sapiens* and we can look for environmental constraints or dietary resources that may have affected

⁹² As Paul Griffiths has argued, psychological categories stand on a different footing than other features of organisms that we attempt to explain by reference to evolution or genetics. Things like wings, eyes, limbs, claws, and feathers can be compared between two organisms (separated by evolutionary history, or modern species classification) and categorized according to whether they are *analogous* or *homologous*. Homology occurs when two traits share the same evolutionary origin (the genes that trigger construction of eyes in fruit flies and humans are the same genes, and therefore homologous). Homology implies a shared mechanism across species. Analogy occurs when the traits being compared have the same function but lack a shared evolutionary origin (so bat's wings and fruit fly wings are analogous, but not homologous). The use of animal models to develop a picture of human alcohol-related behavior relies on an ability to distinguish analogy from homology, and suffers from an insurmountable gap in our current knowledge. Given what we know about the plasticity and adaptivity of the modern human brain, comparison with far simpler systems such as those of the rodent offer poor chances for learning about behavior.

Not only do we lack empirical data about brain and behavior homology across species today, but we also suffer from a difficulty of describing the evolutionary development of the brain in our own lineage. The human brain seems to have been constructed to fill the need for spatial, social, and ecological reasoning. That is, what our brains share with our ancestors brains is the capacity to reason and solve problems. However, as a byproduct of our having and using brains in the context of cultural and technological development, our current reflections on ways to describe its function may differ wildly from how it might have been described in the past. To put it a different way, it is as if we developed hair in order to insulate our heads, but now use it purely for fashion and reproductive goals. Brains are probably the same--the categories we have constructed to describe aspects of their current function are not indicators of the purposes for which they were originally selected, nor are they indicators of the scope of possible function. When it comes to talk of the brain, we are condemned to reliance on modern categories of use whose relationship to properties of selection lies buried in evolutionary history. Consequently, to place our understanding of a complex behavior or emotion on models of evolutionary function is a form of whiggism. This points to the problem with the gene-myth for alcoholism, and the problems we saw with the frugivory hypothesis as well.

the design and function of our olfactory sense. But in both of these cases we would not learn about the function and behavior of the entire system “alcoholic human organism.” Rather, these investigations only serve to illuminate features of *components* of the overall system, and as many have pointed out, the dynamics of these complex systems are not cleanly reducible to these components (Anastasi 1958; Duster and Garrett 1984; Grene 1967; Lewontin 2000; McClearn 1993; Oyama 2000; Sarkar 1999). Complex biological and human systems rarely consist of additive or linearly related components.⁹³ Only by considering the behavior of the entire system and the mutual interactions of its constituent components can we move beyond speculation and hypothesis concerning genetic or evolutionary contributions to modern systems.

Here is the core of the problem: The Frugivory hypothesis takes a variety of data and hypotheses within the “organism” and “human” components of the system and constructs a hypothesis concerning the evolutionary origins of modern human alcoholism. To be sure, it is a remarkably elegant and appealing story about the origins of our modern taste for alcohol. However, the story thoroughly ignores a significant and highly unstable portion of the system: the definitional criteria for and practical application of the label “alcoholic.”

The designation “alcoholic” has suffered from conceptual vagueness and diversity of operational definition since its coinage in the nineteenth century. The difficulties stem from a number of sources: Who performs the diagnosis? According to what set of criteria or operationalized definition is the diagnosis made? How is this definition deployed in different cultural settings? These questions continue to pose serious difficulties for the study of alcohol problems (e.g., see Babor 1990; Heather and Robertson 1997; Jellinek 1960; Robinson 1971; Room 1972). Even the reflexive role of the drinker in documenting and

⁹³ This point is elaborated further in Chapter 4.

assessing their own drinking careers remain extremely problematic for characterizations of alcoholism (Edwards 1984; Kunitz and Levy 1994; Edwards, Brown et al. 1987). Reviewing the three hypotheses outlined earlier, and from the text of the article, it is clear that there is no effort to endorse a particular definition or operationalization of alcoholism. At no point is there an attempt to elaborate what counts as “modern human alcoholism.” Rather, the meaning of the phrase is left to the imagination. This gap threatens the viability of all three hypotheses, for without a clear idea of what constitutes “alcoholism,” hypothesizing on its origins in particular evolutionary environments would seem premature.

While this may seem to be a somewhat trivial matter, or one that Darwinian Medicine can rightly be expected to avoid, I maintain it points to a deep philosophical problem that threatens the viability of any evolutionary account of the origins of alcoholism. Patterns of alcohol production and consumption differ widely across the planet and in historical perspective. What people do, the way that they behave when they have been drinking also differs considerably cross-culturally (Alasuutari 1992; MacAndrew and Edgerton 1969). There have not yet been shown to be any genetic factors that underlie these cultural patterns of alcohol-influenced behavior, and it is doubtful that such factors exist. Humans are cultural and social creatures, and since alcohol serves different roles in different social and cultural contexts, the relationships that individuals forge with alcohol will be subject to these contextual influences. Explanations derived wholesale from biological considerations simply cannot account for the empirical character of alcoholism in these contexts.⁹⁴

⁹⁴ For example, studies comparing populations of northern European and Mediterranean descent suggest differences in *attitudes* about alcohol as well as rates of cirrhosis of the liver. However behavior genetic scholars might argue, we have at present no biological model that can predict human attitudes. Rather, the attitudes and behaviors across cultures seem to be a function of learning the role of alcohol in the culture: “cultures that teach children to drink

For the moment, let's leave aside these worries over the boundaries of the “alcoholic” designation. Let us assume that the Frugivory hypotheses rely on a stable, well-articulated definition of alcoholism. I argue that even in this case, the Frugivory hypotheses faces insurmountable challenges before it can be considered as candidates for the explanation of modern human alcoholism. What empirical observations would be required to validate the hypotheses? We can spell out the empirical requirements. Beginning with **H1** (...an important feature of alcoholism in modern humans may be the “co-option of molecular pathways and sensory biases common to many frugivorous taxa” Dudley 2000, p. 7). First it needs to be shown that there are differences between alcoholics and control groups in olfactory sensitivity to ethanol. This could be established perhaps by measuring responses to ethanol vapors to see if in fact alcoholics are biased toward greater awareness of the presence of ethanol. It is unclear how one would go about operationalizing sensory biases such as these. Is preference for the smell of alcohol the significant measure? Or is it rather the ability to notice alcohol vapors at certain distances? Even if significant differences were found for some measure of olfactory sensory localization in alcoholics versus controls, this would only serve to establish that alcoholics have greater sensitivity to alcohol. Of course, such sensitivity could be the product of physiological development *after* prolonged exposure to alcohol vapors. It could also be a feature of acquired skill in discerning the alcohol content of one's drinks. To support the claim of evolutionary origins, it needs to be shown that people with such sensory biases *prior to exposure to alcohol* are more likely to develop alcoholism sometime in their lives. In this vein, researchers could seek out genes associated with olfactory buds with particular sensitivity to ethanol and evaluate different

responsibly, cultures that have ritualized when and where to drink, tend to have lower rates of alcohol abuse than cultures that forbid children to drink (Vaillant 1995, p. 59).

populations for their possession. Additionally, this hypothesis requires evidence that ethanol played a positive role at some point in our evolutionary past (perhaps aiding in locating nutrients), and that this positive influence was significant enough to influence selection. This question of evolutionary origins is one of the most difficult empirical challenges facing the future of the hypothesis, as the chances are slim of finding fossil evidence of the lineage leading directly to modern *homo sapiens*, much less of evidence that could indicate exposure to alcohol within this lineage.

The second hypothesis (**H2**) holds that alcoholism in modern humans stems partly from the existence of (possibly hormetic⁹⁵) metabolic mechanisms previously selected in evolution to maximize the nutritive benefit of ethanol in small doses, that now operate in an environment in which ethanol is available in abundance. The empirical requirements for testing this proposal overlap with those of **H1**. In order for this proposition to be true we need far more information than we currently possess concerning not only our evolutionary lineage but also evolutionary environments.

Let us assume that Darwinian Medicine supporters are able to find empirical evidence exactly of this sort to support the first two hypotheses. Suppose new fossil evidence were found clearly indicating that the ancestral lineage of modern humans was intensely frugivorous, developed specific olfactory capacities for locating fruit by the

⁹⁵ Hormesis refers to a biological phenomenon where the relationship between dose-size and outcome is curvilinear (so called “U-shaped” or “J-shaped” curves). For instance, small doses of a substance for a population may reduce mortality overall, while at higher doses the substance increases mortality. The low-dose, positive reaction to such substances is termed a “hormetic response.” The concept has been the subject of much debate recently, with some researchers claiming that it is a feature of data collection methods rather than a true biological phenomenon (Calabrese & Baldwin 1998; Parsons 2000; See also Elliot 2000 for an attempt at conceptual clarification of different versions of hormesis). Dudley (2000) has hypothesized that evidence of health benefits in regular wine-drinkers actually points to a hormetic response in humans for some components of wine, possibly ethyl alcohol.

presence of ethyl alcohol vapors, acquired nutritive reward from the ingestion of alcohol, all of which resulted in reproductive advantage for those with greatest sensitivity to and ability to metabolize ethanol. Even in this extreme case, the evidence would not be sufficient to establish the third hypothesis.

The third hypothesis (**H3**) states “*Genetically-based behaviors* adaptive in the ancestral environment become disadvantageous in a modern human environment that provides *ad libitum* access to nutritional substrates, including ethanol” (Dudley 2000, p. 7 emphasis added). As shown above, this supposition fails because of its reliance on a picture of the genetic control of behavior that is inconsistent with what we are learning about the respective roles of genes and brains in living systems. To the extent that genes are involved in brain-building, there are relatively few genes, and the proteins they manufacture are deployed into structures by locally contingent, non-genetic cellular mechanisms (Buller and Hardcastle 2000).⁹⁶ The brain is overbuilt by such mechanisms, then unused neural pathways are pruned over the life of the organism. The consequence of this information from developmental neurobiology for the assumption of behavior-specific modules in hominid brains is straightforwardly negative. The *plasticity* of the human brain in the course of a single individual’s lifetime severely curtails the plausibility of there being modules designed to facilitate specific behaviors in response to specific cues. Articulating the role of genes in the construction and maintenance of these hypothetical modules presents even more complicated problems.

⁹⁶ The small number of genes involved in the process of building the brain through development does not suggest that the determining force of these genes is higher than previously thought. Quite the opposite is true: the small number of genes suggests a highly complex developmental pathway in which many other biological components interact with this genetic material to produce a variety of structures.

“Genetically based behaviors adaptive in the ancestral environment” must be either encapsulated in some modular structure in the brain, or should not really count as behaviors at all. Since the modularity option is not particularly plausible, one remaining option for the Frugivory hypothesis is to limit its scope to “genetically-based sensory structures.”

Following this line of thought, we could focus on our olfactory senses and look for sensitivity to certain molecules over others. Unlike modularity of the brain, this picture of genetically-based sensory specificity is consistent with what we are learning about the olfaction system in hominids (Pybus and Sell 1999).

This is where the Frugivory hypothesis has modest novelty: the olfactory-sensation molecular pathway and its role in human alcoholism. While other pathways implicated by the hypothesis have been shown to be extensions of previously suggested explanations, the role of olfactory stimuli specific to ethyl alcohol vapor has not, to my knowledge, been explored by the alcoholism research community. Despite the aforementioned difficulties of designing studies to test this relationship, this is perhaps where a component of the Frugivory hypothesis can be put to empirical test. But there remain a number of questions. How did this olfactory-sensory mechanism combine with a dietary preference for alcohol-bearing fruit stemming from its increased caloric value? Where would we locate such preferences in physiological terms? What system of genetically induced dietary behaviors exists that can support this supposed link in evolutionary context? How would this link operate to affect the behavior of modern humans? The only speculation offered on these questions is that modern alcoholism is an “appetitive behavior” (p. 8). Further elaboration on this front is clearly needed.

In this chapter I have explored the specific difficulties of explaining traits of organisms by reference to genetic factors alone. Coupled with the heterogeneity in disease definitions and proposed mechanisms we saw earlier for alcoholism, I have added considerations of the many levels of phenomena that may influence any complex human trait. Looking from the “top, down” we face the difficulty of the looping effect of human and label designations. Moving from the “bottom, up” all of our explanatory models face the problem of determining the pathways by which molecular mechanisms influence outcomes at higher levels of biological organization, and conversely how phenomena at these higher levels influences subsequent activity at the lower levels. The interrelation of these feedback systems and their role in aspects of the human condition like personality traits or alcoholism defy our current modeling abilities. Lacking a theoretical approach in which these non-linear causal systems can be modeled and evaluated, the prospects for progress and explanation are slim.

In the next chapter I consider one approach to the problems posed by complex biological systems, Developmental Systems Theory, with the aim of evaluating its promise for updating the genocentric view of alcoholism.

Chapter 4: Making sense of heterogeneity

“Rather than searching for radically different ways of studying organisms or for new laws of nature that will be manifest in living beings, what biology needs to do to fulfill its program of understanding and manipulation is to take seriously what we already know to be true. It is not new principles that we need but a willingness to accept the consequences of the fact that biological systems occupy a different region of the space of physical relations than do simpler physico-chemical systems, a region in which the objects are characterized, first, by a very great internally physical and chemical heterogeneity and, second, by a dynamic exchange between processes internal to the objects and the world outside of them. That is, organisms are internally heterogeneous open systems.

—Richard Lewontin (2000, pp. 113-4).

1. Steps toward understanding the complexity of alcohol problems without genetic reduction

As shown in the analysis of DSM criteria for alcohol dependence, “alcoholism” is a slippery concept to define. There are several ways we can come to grips with this slipperiness. One way of proceeding from this point would be to assume our current definitions and measurements of alcohol-related behavior, however deficient, will eventually home in on the essential aspects of the problem. Since the DSM criteria currently isolate as many as 35 distinct “alcoholisms” perhaps we can pursue a strategy of conceptual clarification by determining which of these definitions significantly overlap and then focus on a smaller number of distinct diseases, each with their own individual mixtures of social, biological and genetic associations. We might develop a new nomenclature for these and determine which biochemical pathways are most likely involved in their origin and progression, characterizing the diseases down to the level of gene alleles that associate strongly with them. In fact, this seems to be part of a trend in recent alcohol-studies research, as can be seen in the explosion of “sub-type” descriptions of alcoholism(s) and

contributions from genetic factors.⁹⁷ The proposed sub-types involve focus on very specific variables found in longitudinal and cross-sectional studies of alcohol problems, and include categories such as “early-onset Alcohol Dependence” (see e.g., Atkinson, Tolson and Turner 1990; Chou and Pickering 1992; Dawson 2000; Grand and Dawson 1998; Gonzalez 1989; Johnson, Cloninger et al. 2000; Sobock, Abbey et al. 2000; Turner, Cutter et al. 1993), anti-social personality disorder (Hesselbrock and Hesselbrock 1994), “obsessive-compulsive drinking” (Anton 2000; Anton Moak and Lantham 1996; Moak Anton and Lantham 1998), and other subtypes based on personality-trait analysis (Allen 1996; Von Knorring, Von Knorring et al 1987; Devor, Cloninger et al. 1993). While these have yet to be adequately fed back into the diagnostic criteria used to collect samples and gather data, they represent attempts to articulate with precision the varieties of phenomena that can be distinguished within the heterogeneous complex of modern alcoholism. However, such attempts retain the reductionist assumption that this complex feature of the human condition can be understood by breaking it into parts and analyzing each element for a linear, additive causal contribution. The same inaccuracies and flaws that plague previous genetic accounts of alcoholism will be re-produced in this attempt to find multiple, but distinguishable biochemical and genetic determinants.

I maintain that a more ambitious approach may be required. For example, we might come to grips with the conceptual slipperiness by backing away from the problem and taking a fresh view, re-characterizing the problem, its definition, means of measurement and ultimately address fundamental questions about its epistemic status. Articulating the

⁹⁷ See e.g., Mezzich, Tarter et al. (1993), Glenn and Nixon (1991), Glenn and Nixon (1996), Schuckit (1991), Caplan (1996), DelBoca and Hesselbrock (1996), Johnson, Van Den Bree et al. (1996), Lesch and Walter (1996), Anthenelli and Tabakoff (1995). Genetic sub-typing has been attempted in Hesselbrock (1995).

distinction between disease etiology and its place in our understanding of the human condition is no mean task. At a very fundamental level this approach has been pursued with respect to disease *qua* disease (Canguilhem 1978). Some attempts to deal with this philosophical issue in recent years have approached the problem as one of dealing generally with the role of genetic information in sickness (Childs 1999), and some have dealt specifically with a reassessment of the framework in which alcohol problems are observed (Heather and Robertson (1997). The issue remains unsettled, and a central question remains unanswered: “Is addiction an hereditary disease, or merely a choice with predictably negative consequences?” In order to develop language for an answer, one promising line of work suggests that we move toward the goal of articulating the difference between drinking careers and natural history aspects of alcohol problems.

2. Distinguishing between Natural History and Drinking Careers

The call for conceptual clarification of alcoholism was put to “those trained in the philosophy and logic of science” in 1984, when Griffith Edwards surveyed the confusion over methods for distinguishing aspects of “drinking careers” from “natural history” with respect to alcoholism (Edwards 1984, p.175). Natural history is a concept drawn from the arena of epidemiology, where it refers to the natural progression of a disease, infection, or pathology.⁹⁸ The difficulty over this distinction comes from the fact that every case-history of alcohol-related behavior is part of a life-long “drinking career,” involving sequential

⁹⁸ I should note here that “natural history” is used here in a restricted sense to refer to the progression of a disease state. This should not be confused with the meaning of “natural history” in a general history of science context where it refers to the enterprise of articulating the history of living things on our planet.

behaviors within a role. The distinction between these elements is most easily seen from a longitudinal perspective.⁹⁹

Analysis of behavior and the social realm by means of the notion of social “roles” has produced a mountain of material and advanced considerably the understanding of social and cultural forces.¹⁰⁰ A focus on “drinking careers” accepts this starting point of social roles and considers the career to be a sequential set of roles. Those roles that involve the individual’s behaviors with respect to alcohol can be plotted through the course of an individual’s life and give us a perspective on the turning points, progressions and reversals that distinguish certain drinking careers from others. The relevant information about turning points can even be assessed based on its source: reflections by affected individuals about what they consider to be turning points (e.g. Edwards, Brown et al. 1987), can be weighed against the traditionally recognized turning points like interventions by health care professionals, diagnoses, legal events, etc. (Conrad and Schneider 1980). The units of analysis are both individual and cultural elements, for in defining and characterizing an individual’s roles, reference must be made to the influences on the individual, be they from advertising campaigns, law enforcement strategies, self-help fads, or legislative developments. Collecting and searching for trends among individual histories comprising these drinking careers offers promise for analyzing the complex mixture of forces that shape alcohol problems, and offers a welcome opportunity to consider in detail the interactions of individual and social forces for specific cases. The crucial elements in this analysis are forces that influence the transition from one stage of a drinking career to the next. For example, if

⁹⁹ The summary of the notion of drinking careers that follows is drawn from the view expressed in Edwards (1984).

¹⁰⁰ See Erving Goffman’s pioneering work in this area, particularly his development of the “sick role” designation in *Asylums* (1961).

we have a reasonably large collection of individual drinking careers, the social and psychological elements associated with the individual transitions from moderate social drinking to psychological dependence are factors that hold considerable explanatory potential.¹⁰¹ Unfortunately, we are not yet near the stage of having in hand collections of longitudinal data regarding individual drinking careers.¹⁰²

The hope here is to develop strategies to distinguish drinking careers from the natural history of alcohol problems, and to empirically test the distinction on which these types of analysis can be performed. As a definition for “natural history” Edwards proposes

¹⁰¹ Despite a century’s long quest for genetic underpinnings of alcoholism, the possibility remains that this is a disorder without physical foundation. The variance, across cultures and even between members of the same culture, of drinking patterns, drinking problems, disease definitions, and visions of self-control suggest that there are social and cultural pre-requisites for medicalizing alcoholism(s). Any truly adequate understanding of the role of alcohol as an element in the human condition must address this cultural variance on its own terms, without appeal to biological differences across cultures. For some initial steps in this direction, see e.g., Babor (1986); Cahalan (1978); Dean (1997); Duster (1984); Fox (1977); Levine (1981); McAndrew and Edgerton (1969); Nye (1984); Pittman and Snyder (1962); Risjord (2000); Rothschild (1981); Shivelbusch (1992); Vallee (1998); Waddell (1981); Waitzkin and Waterman (1974); Williams (1994).

¹⁰² Some steps in this direction have been taken, see for example the prescient work of Martin Plant (1979). Another prominent longitudinal data-set has been compiled for comparison of alcohol-related behavior among ivy-league and inner-city samples of college-age men (see Vaillant 1995). Unfortunately the data so compiled features few variables that could be used to assess the social roles fulfilled by the subject’s drinking behavior, and therefore analysis in terms of drinking careers for this data-set is severely limited. Another limitation stems from the incredibly limited number of studies regarding women and minorities, as well as longitudinal research that includes information from early childhood. Researchers working to document drinking career aspects of alcohol problems may be dispersed, but their work is beginning to make its way into mainstream alcohol studies publications. See e.g., Neve, Lemmens et al. (1997), York (1995), Fillmore (1990), Taylor (1994), Kunitz and Levy (1994).

An historical footnote on the application of this method in a dietary context can be seen in the attempts to study the vitamin deficiencies caused by long-term ingestion of alcohol (e.g., B-complex vitamins and dietary Chromium Picolinate). Deducing the final stage of severe alcohol problems as one brought on by such deficiencies, researchers have even patented forms of delivery for these vitamins with the aim of defending against this final stage. See Dobbins (1990; 1991); Williams (1959).

the “sequential development of designated biological processes within the individual” (Edwards 1984, p. 178). The more obvious “biological processes” are things like cirrhosis of the liver, physiological withdrawal symptoms, muscular atrophy, and so on. These are clear biological consequences of heavy drinking and what bears attention are clear-cut stages in their sequential development. Mapping the development of these biological processes in large samples of affected individuals may produce an understanding of the disease as a series of well-defined, distinct, progressive stages. By focusing on the transitions between these stages, researchers can better understand the exact way in which specific systems are compromised, and subsequently develop strategies for intervening in defense of these systems. For example, Taylor (1994) argues that the analyses of drinking behavior over the long term that attempt to isolate predictors of end-states using the progressive disease model inherently obscure potentially meaningful events at the individual level that trigger an individual’s change from one status to another (p. 63). Once these individual changes are mapped, they may provide more insight into both the external and internal forces that affect individual drinking for specific sub-types.

The problem that remains for researchers willing to employ and further articulate the distinction between drinking career and natural history stem from the lack of a framework into which they can be placed as true interacting elements. Disagreements and divisions in the field of alcohol studies take disciplinary lines—with medically trained psychiatrists endorsing biological and genetic reductionist accounts of alcohol problems, while sociologists, psychologists and some anthropologists cling to statistical representations of variables that offer predictive value for alcoholism.¹⁰³ Part of the problem stems from the

¹⁰³ These disciplinary obstacles in alcoholism research only replicate the divisions that plague much of the larger landscape of the human sciences. Tensions among accounts of the

deep disciplinary rift over expectations of where the best explanation can be found.

However, the larger issue looms of finding a method of gathering data for analysis that is not biased by the very definitions the researchers seek to validate.

Even within the small corner of inter-disciplinary research, the overwhelming difficulty remains one of developing a perspective on the adult human condition in which biological, social, psychological, and cultural factors exist on comparable footing. The aim is not to model these factors (as researchers since the 19th century have attempted) as separate contributors to the end state. Rather, what is required is a model in which these factors truly interact over the life-course of individuals or groups.

Since it is unlikely that disciplinary divisions will allow for, or can reasonably be expected to produce, such a model, we are obligated to mine other interdisciplinary projects for theoretical frameworks that are capable of producing such a model. In what remains of this chapter I explore one strategy for borrowing from developments in philosophy of biology that may ultimately serve the required end.

The heterogeneity of gene concepts I have examined throughout this dissertation cannot be ignored in the philosophical analysis of complex human behaviors. While there is consensus among philosophers of biology that many fundamental concepts in biology are shifting in the face of greater access to the genomes of a number of organisms, there is no such consensus concerning the direction of such conceptual transitions.

Ultimately, many of the problems confronting evolutionary reconstructions such as the Frugivory hypothesis on the origins of human alcoholism have to do more with the

human condition generated from micro- and macro- sociological views compared with those generated from evolutionary biological and medical views are tensions that run deep. In this respect it will be intriguing to learn whether a new approach in the case of alcohol studies will have repercussions throughout the human sciences.

untidy state of the meaning of the designation “alcoholism” than they do with problems internal to evolutionary theory (although evolutionary itself is the source of much debate). The telling question for this writer remains “What would a proper definition of alcoholism look like?”

Earlier I argued that alcoholism can be conceived in terms of a system with a number of components, each capable of interaction, feedback and non-additive causal relationships. The system called “alcoholic human organism” needs to be filled in completely, especially the component loosely called “alcoholic.” In order to fully elaborate what is meant by this designation, we could do well to bear in mind that our current designation is the result of interactions through time of a number of previous designations, all of them imbued with cultural, behavioral, and even religious significance.¹⁰⁴ A robust and detailed picture of the development of these designations over time is what we will need in order to flesh out the final component of the alcoholic system as it appears in modern humans.

As Longino (1990) has shown, the fact that science is inextricably embedded in shifting social and cultural contexts is no reason to think that its claims suffer by this association. “Good science” and “bad science” judgments are not functions of the degree to which any particular scientific hypothesis or claim is connected with social or cultural values because all scientific claims are connected to these values, in addition to being formed in the context of social interactions within science. Claims that are perfectly reasonable from the perspective of scientific logic or rationality may still be deemed unacceptable by people wishing to build on them because of a failure to recognize the social and cultural historical

¹⁰⁴ Some preliminary features of such an investigation are elaborated by Hacking (1995) with respect to another troublesome designation, “autism.”

context in which they are advanced. This is precisely the case with alcoholism and evolutionary theorizing. Due to the sheer number of previous attempts to account for alcoholism's origins by reference to evolution and genetics, any new theory that follows this path must live up to—or live down—the expectations created by the weaknesses in the former attempts. Absent some effort to recognize what has gone before and to distinguish the new theory from it, current models like the Frugivory hypothesis suffer from exactly this failing. Treatment specialists are still looking for ways to characterize (and eventually minimize) the problems associated with alcohol use. In so doing, they are also actively testing new and different ways of defining what count as alcohol problems, destabilizing the designation “alcoholic.” Unless the Frugivory hypothesis can serve as a foundation for meaningful progress in research on the etiology and treatment of alcohol problems, it will be relegated to the brimming dustbin containing previous just-so stories about heredity and alcoholism.

Only with a historically and culturally informed perspective in hand can we begin to truly evaluate the relevance of details that science brings us from the other components of the system, “human” and “organism.” While the Frugivory hypothesis is an interesting elaboration of evolutionary theory and its consequences for “organism,” it fails to be of much help to those who deal with the full-blown system of modern alcoholism in research and treatment contexts today.

3. Developmental Systems Theory

Earlier I suggested two ambitious strategies for reassessing the role of genetic information in our understanding of modern alcohol problems. While the first of these, a careful and

probing review of the foundations for the distinction between social deviance and medical disorders holds considerable promise, it is a project the details of which remain beyond the scope of the current dissertation.¹⁰⁵ However, it should be noted that even this strategy is potentially susceptible to the same set of problems that plague accounts built on the gene-myth. By attempting to articulate the distinction between biological and psychological/social factors this approach runs the risk of reifying the view that genetic factors can in practice be separated cleanly from other factors, and their causal contribution quantified. While proponents of this approach may have a respectable scientific goal in mind, the analytic strategy required by the approach will only reproduce the same deficiencies that render simplistic genetic-reductionist accounts untenable.

The second strategy involves applying to our most nuanced models of alcoholism insights from developmental systems theory (DST). In what follows I argue that this approach holds greater promise overall and deserves a closer look. In order to assess this promise, allow me to first introduce the key tenets of the approach.¹⁰⁶

One steady proponent of DST is author Susan Oyama. Some of the approach's core ideas are presented in the writings of Richard Lewontin, and there is evidence that the central notions have been in circulation as far back as the 1930s in the work of some scholars (e.g., Hogben 1933; Sarkar 1999). In Lewontin's book *Not In Our Genes*, he describes the problem for biological researchers posed by complex organisms and the limits to the reductionist paradigm brought on by these obstacles. The problems stem from the focus of technological tools on a specific level of biological analysis..

¹⁰⁵ See, e.g., Heather and Robertson (1997); Conrad and Schneider (1980) for some initial attempts in this direction.

¹⁰⁶ Robert (forthcoming) documents some historical figures whose ideas foreshadow some central concepts in developmental systems theory.

The limitation of experimental biology to manipulating one or a small number of causes by large perturbations has had a profound effect on the kinds of explanations that are offered by biologists. The methodological limitations of experiments are confused with the correct explanations of the phenomena. The constant reiteration of the claim that genes determine organisms is a consequence of the ease with which major genetic changes can be induced in experiments and the large effects that those changes have on the experimental objects. Moreover, only those phenomena are considered that lend themselves to the method. Developmental geneticists ask questions about the differentiation of anterior and posterior ends of animals and the formation of major body segments in between because single major gene defects can be found that alter that process. They do not know how to ask why different individuals have heads and legs of different sizes and shapes, even individuals of different species. So they never ask (Lewontin 2000, pp. 98-99).

Lewontin attributes the entrenchment of genetic reductionism to the understanding of biological entities we have constructed using genetic-reductive methodologies and research programs that focus on the “perturbative” powers of individual genes at the expense of an addressing the complexly interactive nature of other elements in biological systems. The reason for altering this view of biological entities so as to foster an understanding of these interactions is clear.

There exists, and has existed for a long time, a large body of evidence that demonstrates that the ontogeny of an organism is the consequence of a

unique interaction between the genes it carries, the temporal sequence of external environments through which it passes during its life, and random events of molecular interactions within individual cells. It is these interactions that must be incorporated into any proper account of how an organism is formed (Lewontin 2000, pp.17-18).

The central feature of the developmental systems approach is a focus on the interactive nature of a variety of aspects of biological systems in the life-course of organisms. This focus on the life-course in its entirety (i.e., *ontogeny*) produces a perspective from which the ebb and flow of contingent causal cycles within these systems take center stage.

Providing an overview of DST remains something of a difficult task. While there is a relatively limited amount of scholarship on the topic, there are different interpretations of the central tenets. These differences can be characterized in large part by the discipline in which DST is being deployed. Evolutionary biologists seem to be concerned with the impact on accepted models of evolutionary history that might follow from a focus on developmental systems rather than genes (Robert, Hall, et al. 2001). Psychologists are concerned to spell out the key processes in development that affect the central nervous system, brains and minds. In both arenas, DST represents a shift away from thinking about genes in isolation from the organisms in which they exist. By moving toward fuller description of the function of the organism, scholars are developing a framework in which problems pertaining to organisms can be more fully understood through the interaction of multiple elements at many levels, rather than reduction to the singular level of genetic factors.

In a variety of publications Susan Oyama has proposed core concepts for developmental systems theory that are helpful for getting a handle on the approach (Oyama

2000; Oyama, Griffiths et al. 2001; Robert forthcoming). One key feature of DST involves the recognition that it is empirically incorrect to characterize the development of an organism from birth, to maturity, to senescence, and death as the unfolding a “program” located in the genes. There is no single program, and the relevant influences on development cannot be parsed into neat categories of “environment” and “genes.”¹⁰⁷ This is betrayed by the fact that all cells in the organism develop into remarkably different functional systems, despite having identical genetic material within the nucleus. Thus, development, differentiation, and the regulation of growth cast genes as but one of many resources operating in the construction of the traits and characters of the mature organism. The organism is constructed through interaction, not programmed for maturation (Oyama 2000).

The second main feature of DST centers on refurbishing our understanding of reproduction. Describing what takes place in reproduction (aside from the obvious bits) requires a full characterization of the material and circumstances of heredity. We need to step back from genetic studies and ask “what is transmitted from organisms to offspring?” While nuclear DNA is well-characterized, the cellular machinery with which DNA interacts in vivo is not. Even less well understood are a host of features that reproduce the environment at specific slices of time throughout the life of the offspring. Such features include maternal reproductive environment, nutrient flow, symbiotic micro-organisms, cellular gradients, parental care, etc. Hence, our understanding of “heredity” needs to be

¹⁰⁷ Burian, Gayon and Zallen (1988) document that with a few exceptions most French experimental biologists in the early 20th century recognized this aspect of organisms as fundamental. Research in causal embryology proceeded from the recognition that every cell in the organism contains the same genetic material, and therefore what requires explanation is not the presence of the genes through successive generations, but rather how cells differentiate into the complex arrangement of functioning organisms despite this genetic uniformity.

expanded to include these elements that constitute interacting agents in development. The answer to the question is, then, “what is transmitted between generations is not traits, or blueprints, or symbolic representations of traits, but developmental *means* (or *resources*, or *interactants*)” (Oyama 2000, p. 29).

In this emerging framework for understanding the organism from the perspective of development, genes lose their mythical status as the causal force to which all features of an organism are traced. In place of this arrangement, genes are one set of many resources that interact in complex, often cyclical, pathways that altogether comprise the biological system that produces the mature organism with all its characters and traits. On this view, to focus on a trait or character of an adult organism and its pattern of appearance through generations will provide no information about the organism that reproduces the trait.

Another tenet of developmental systems theory is the requirement for specifying the levels of analysis at which the interacting factors operate. The goal here is to identify and characterize entire systems of feedback and feedforward information through multiple levels of the organism, and through different temporal sequences. The resulting picture of the development of the organism includes elements from levels as large as geographic obstacles to those as minute as molecules. Unpacking all the levels in between requires that we take seriously the potential for causal influence, in *both* directions, through and across all these levels. What is required is a “shift from single to multiple scales” (Oyama 2000, p. 3ff).

This brief review of concepts emerging from DST highlights four valuable recognitions that we may be able to take from this focus on development and deploy in the study of human alcohol problems. First, DST promotes dispensing with a focus on singular *traits* and reorienting our analysis to consider *systems*. Second, by expanding our view of what is involved in heredity we can uncover multiple *interactants* that together collude in the

construction of the organism. Third, DST promotes a shift from single to *multiple levels* of phenomena that can be considered relevant to the construction of the organism. Finally, we have the recognition that causal influence operates in non-linear pathways in *multiple directions* through and across these levels.

4. Developmental Systems Theory and Alcoholism

Applying these concepts from DST to the study of alcoholism offers the promise of highlighting the importance of some features of the phenomena that may lead to the discovery of systems that play significant roles in the development of alcohol-related behaviors over the life-course of individuals and groups. The clearest benefit from applying DST to alcoholism will be a shift from research aiming to elucidate the “predisposing factors” for the condition, to a focus on the systematic interrelations among factors that interact to produce the condition over the life course. The focus on the life course, and therefore longitudinal studies, is another clear ramification of adopting tools from DST.

By undercutting the reductionist paradigm with respect to genes, DST clears out a space in which we can evaluate many components that are relevant interactants within any single condition (rather than assuming that genes are responsible and ignoring non-genetic factors that could perfectly-well be involved in the construction of the problem). So, on this approach, genes still may turn out to offer the best explanation of any single disorder (such as one of the 35 different subsets of alcoholism plucked out by the DSM-IV diagnostic criteria), but only after we have evaluated all the other factors (many not-yet-understood) that participate in the development under investigation. Absent an accounting of the other cycles of developmental systems that may be involved, we can never claim to have anything more than a correlation whose cause may be a third factor (or system of factors).

For example, let us examine the causal model deployed as part of the frugivory hypothesis discussed in the previous chapter. Suppose we have good evidence suggesting that in the hominid lineage leading to modern human there was significant selection based on ability to derive nutritive value from ethyl alcohol. Suppose further that as a result of such selective forces modern populations possess a variety of gene alleles that, given a certain suite of cellular machinery and a cue to begin transcription, code for production of two enzymes, alcohol dehydrogenase (ADH) and acetaldehyde dehydrogenase (ALDH). These two enzymes participate in the metabolism of the ethyl alcohol molecule. First, ethanol is broken down by ADH, and the aldehyde that is a byproduct of this reaction is then broken down by ALDH. The products of these first two reactions then enter the Krebs cycle (also called the citric acid cycle) through which after a number of molecular interactions they produce ATP, a molecule used as an energy source in a variety of reactions throughout the value. In this way, ADH and ALDH serve as something akin to gatekeepers for the production of energy from ethyl alcohol. With slow-acting variants of the ADH enzyme, the metabolism of alcohol takes place slowly, so that alcohol has a greater chance to diffuse throughout all the tissues of the body,¹⁰⁸ including the brain, and induce intoxication. Slow metabolism of the ALDH enzyme allows aldehyde to accrue in the body, which produces an uncomfortable toxic response that includes flushing and burning sensations in the skin. Conversely, fast acting variants of both these enzymes produces an organism that can consume considerable quantities of alcohol, metabolize it quickly, and derive little psychotropic alteration as a result.

¹⁰⁸ This is why testing the air flowing from a drinker's lungs (i.e., "breathalyzer tests") can provide a measure of intoxication. Alcohol permeates the lungs at the same rate that it permeates through other tissues of the body, including the brain.

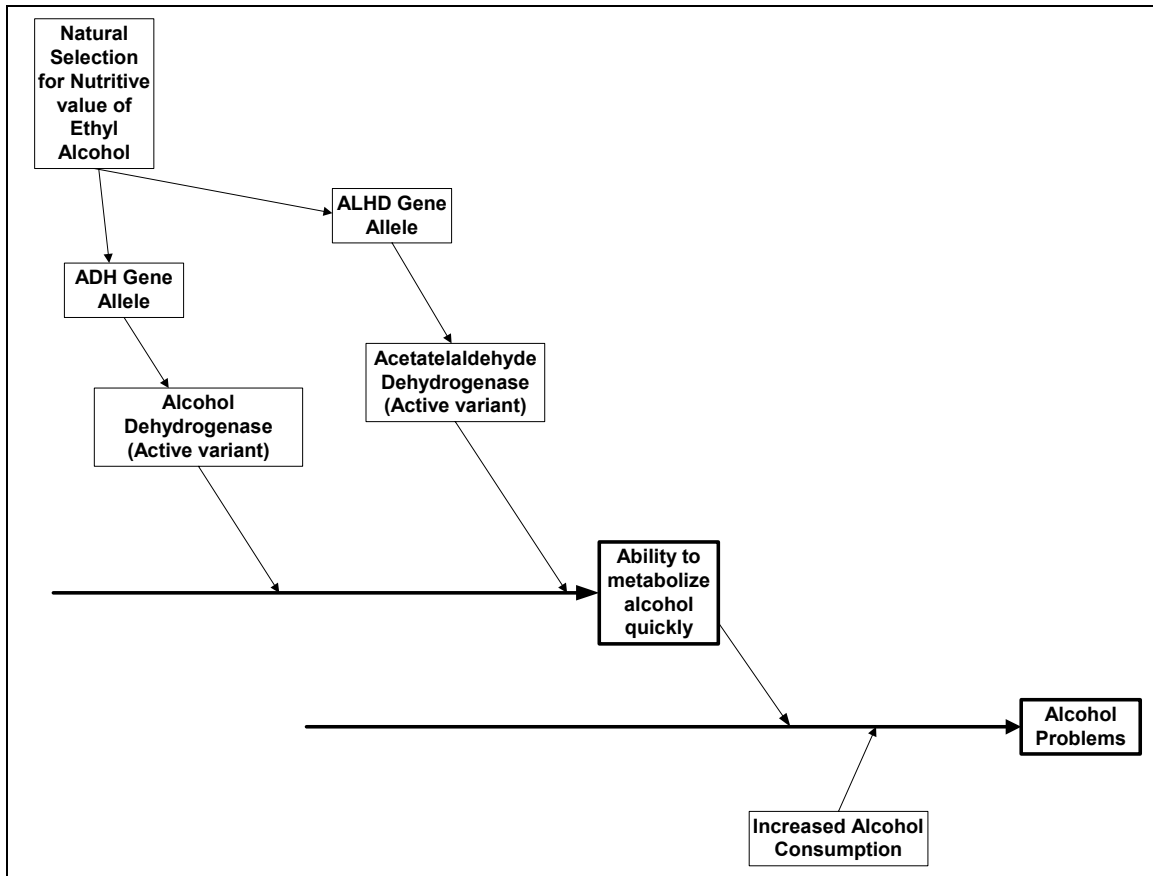


Figure 4.1. One Possible Causal Pathway for Alcohol Problems, based on the ADH and ALDH enzyme activity path deployed as a premise in the Frugivory Hypothesis.

For the last assumption in our example, let us suppose that distributed throughout the population are a number of variant gene alleles that produce enzymes whose rate of activity differs significantly. The frugivory hypothesis on the evolutionary aspects of modern human alcoholism proposes that the differential rates of alcohol metabolism across the population should be considered a genetic factor that predisposes certain people to alcoholism. In figure 4.1 I have constructed a diagram of this causal influence from natural selection, through gene alleles, to alcohol metabolism rates that illustrates the proposed causal model. The two horizontal arrows are main effects, and the smaller arrows represent

causal factors that can figure into empirical investigations and subsequent quantification of the contribution they offer for each effect.

Note that in this model the arrows move in one direction, from genes through to a main effect of a property of the whole organism, viz., the ability to metabolize alcohol quickly. Notice also that the main effect that concerns us is not this metabolic state, but rather its collusion in the production of alcohol problems, which is the real phenomenon we are limited to measuring by the DSM-IV criteria. This is the best we can do when dealing with a genetic reductionist model of alcoholism.

In figure 4.2 I have arranged a more robust model of alcohol use and abuse in which the same enzymes figure alongside other interactants, in a more complex system of effects. Note that this model employs the assumption of a system with three major subsets as outlined in the previous chapter (alcoholic, human, organism). The center main effect “Experience and Reflection on Alcohol Use” has been added, and is meant to capture the self-reflective aspects of individual drinking behavior that earlier I called “drinking career.” For the rough purposes of this example, these figure as levels at which analyses of the phenomena need to be mined for interactants. Note also that one portion of the diagram has been cordoned off with a temporal label “ T_N ” that serves to indicate the specific time during ontogeny during which this portion of the system is functioning. All components of the system should be amenable to this sort of analysis, and ultimately this diagram could serve to represent only one time-slice in a longitudinal evaluation of the influence of these interactants.

Finally, note that in figure 4.2 one crucial tenet of DST is not adequately represented. The reasons for this is that it would very difficult to do this in two-dimensions. What’s missing are the arrows that could represent multi-direction influences. For example, the

factor in the lower right “Cultural context for alcohol use” should be connected also to “age at exposure” as well as “experience and reflection” and any number of other interactants.¹⁰⁹

Without these interrelations, the model remains encapsulated in a linear causal framework.

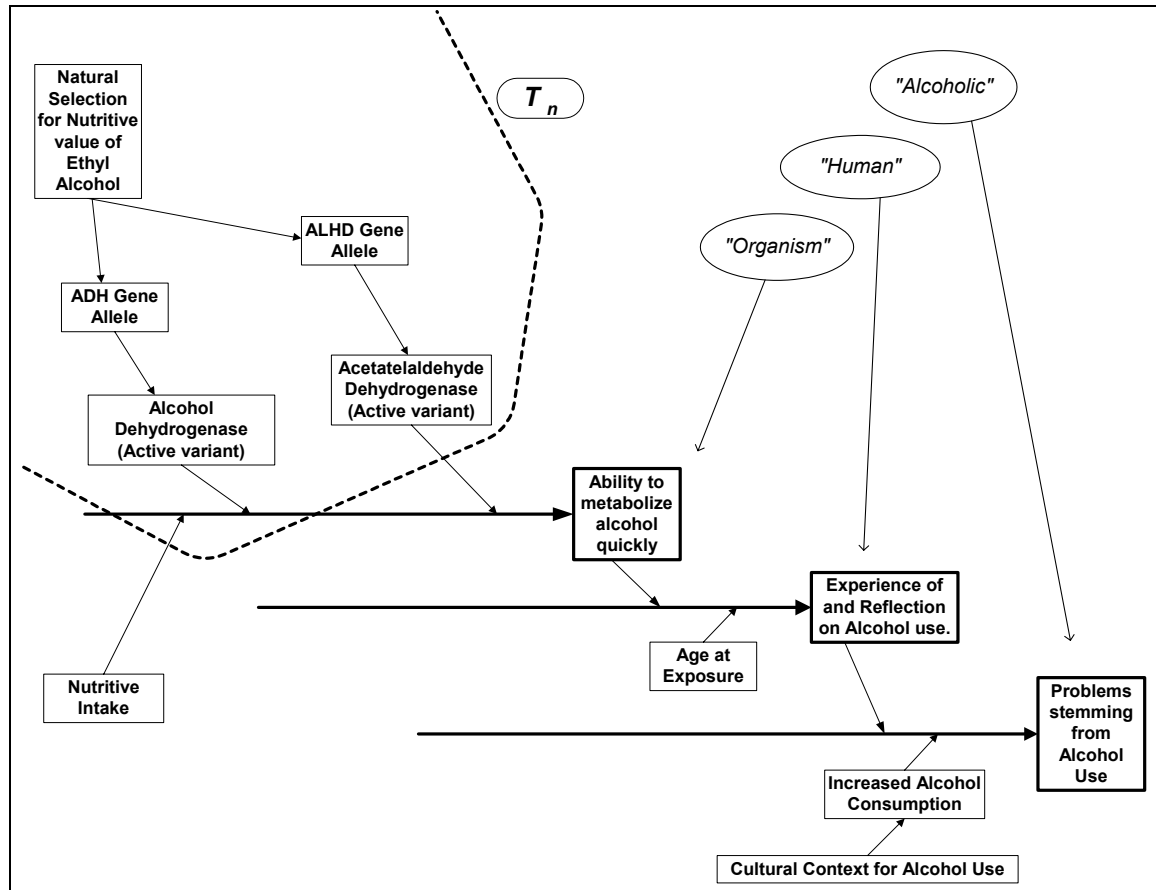


Figure 4.2. Causal pathways from ADH and ALDH to alcohol problems, seen as three interrelated systems named “alcoholic” “human” and “organism.”

The case of alcoholism brilliantly illustrates the usefulness of the concept of an “interactant.” As defined by DST, the *interactant* is an element that plays a role in

¹⁰⁹ Age of exposure and age at onset have been examined as significant variables figuring in the progression of alcohol-related problems, but to date these factors have not been explored in conjunction with a model of alcohol’s role in the development of the organism as a whole. See, e.g., Chou and Pickering (1992); Gonzalez (1991); Dawson (2000). Some research relevant to this issue is appearing in the context of investigations into fetal alcohol syndrome.

development, expression of hereditary information, expression of environmental information, etc. What is different about the interactant (from, say, a gene-for, or an evolutionary module) is that in order to flesh out the concept its role in ontogeny has to be assessed throughout the life-course, and at all “levels” of phenomena within the system that comprises the organism. An interactant may be something we formerly treated as a feature of “the environment.” Interactants can be inside or outside the skin.

Alcohol is an excellent example of an interactant—it exists both inside the flesh after ingestion, where it participates in a wide array of phenomena, and outside the flesh, where it participates in systems of cultural heritage, the social milieu, and exchange systems.

As mentioned above, interactants participate in many different systems in the organism. What does this mean? With human alcoholism, the alcohol-as-interactant participates in many systems, and its participation is a function of the history of the organism and its previous interactions with alcohol, as well as a function of the developmental stage of the organism. The developmental effects of exposure to alcoholism have begun to emerge from studies of the “fetal alcohol syndrome,” that is, the result of exposure to large amounts of alcohol in the prenatal environment. But these studies have so far failed to trace out the (admittedly vast) potential developmental consequences of such early exposure throughout ontogeny. Another result of considering alcohol-as-interactant may be a fruitful focus on data regarding certain variables already gathered from traditional studies. For example, the variables “age at first drink” and “age at exposure” used in a number of traditional clinical and sociological studies, can be included for analysis in a framework that also considers developmental feedback systems, interaction between metabolic function and nutrition, endocrine system disruption, and so on. The net effect of evaluating such diverse but related phenomena may be a developing picture that more adequately situates alcohol as an

interactive element within a larger system characterized by many interleaving feedback loops, from the level of molecule on up to psychology.

The difference between these simple diagrams serves to illustrate two things: the inadequate simplicity of the attempt to reduce complex features of biological systems to genetic factors, and the need for better techniques with which to characterize and model the truly non-linear interrelation of interactants in developmental systems. Note that the above schematic representations of causal pathways only take into account one set of gene alleles, and three “levels” of the problem, that may participate in the construction of a metabolic state. For any given “trait” of the organism that we wish to study, there will probably be hundreds of relevant elements, nestled within many levels and all interconnected in bi-directional causal pathways. What I have provided in figure 5.2 represents only an example of one step in this analysis of hundreds, and since the nature of the system is not amenable to analysis by components, any model that fails to address the full complement of relevant elements will not capture the dynamic mechanisms we seek. Further, these models built on the basis of considerations from developmental systems theory need to be extended so as to handle information about social context that places it on par with biological or genetic information. To put it differently,

Individual biological limitations understood from viewing individuals as isolated entities in a vacuum are not individual limitations for individuals embedded in society. It is not that the whole is more than the sum of its parts. It is that the properties of the parts cannot be understood except in their context in the whole. Parts do not have individual properties in some isolated sense, but only in the context in which they are found. The theory of human nature that searches for that nature in the products of genes in

individuals and the limitations of individuals caused by those genes, or in the properties of an external world that are fixed and that cannot be altered except in a destructive way, misses the whole point (Lewontin 1992, p. 122).

The deployment of DST for increasing our understanding of human problems has great promise. Every effort needs to be made to place all of the parts of complex systems within the context of their operation during the sequence of life. Until such time that techniques for modeling such systems can be realized, it is futile to focus our effort on further application of genetic reductionist analyses of alcoholism.

While philosophers of science have been fascinated for centuries by the mechanics of conceptual change in science, it seems odd that there has been so little attention aimed at the many assumptions that have resisted change. The gene-myth of alcoholism is one such assumption whose picking apart is wholly due.

5. Conclusion

My reasons for putting the case of alcoholism on display in this dissertation are suggested in the main title of this work: “Heredity and the Human Condition.” It is my belief that many of the current inhabitants of the United States carry around what they take to be an informed opinion concerning the role of heredity in shaping who they are, how they act, what diseases might befall them, and how they may meet their demise—significant aspects of the human condition. I take it on faith (and from a smattering of cover stories on such periodicals as *Time* and *Newsweek*¹¹⁰) that heredity is afforded a great role in this

¹¹⁰ Three interesting covers of *Time* magazine come to mind. 11 January 1999: “The Future of Medicine: how genetic engineering will change us in the next century.” Here the image depicts a double helix spiraling upward and morphing into the serpent on the sword symbol long associated with medical practice. The image alone serves as an example of the rhetoric

landscape of forces that affect one's course through life. Part of my aim in putting together the preceding assortment of historical snapshots and philosophical analysis is to make it apparent that such a view is not in keeping with what we know about humans as organisms, nor about humans as humans. For while there are certainly a suite of diseases and conditions that follow from by the presence or absence of a single DNA nucleotide sequence (e.g., Tay-Sachs disease or cystic fibrosis), these are but the simplest and most straightforward cases to explain. Here the aberration at the level of DNA sequence produces clearly traceable cascade of phenomena that ultimately affect the structure (and therefore function) of a particular protein. Here the explanatory model of genetic reductionism succeeds. However, this model of explanation simply does not have purchase over features of the human condition that involve interactions of many genes, and much other biological material, through particular sequences of environmental scenarios. As but one aspect of the human condition, alcoholism provides a potentially rich case for working through the intricacies of the involvement of hereditary forces (biological and otherwise) in the development of human behaviors, attitudes, self-evaluations, and social roles. Alcoholism is particularly interesting because, unlike human "characteristics" such as I.Q. that rest fundamentally on social constructions, it involves an identifiable relationship between humans and the ethyl alcohol molecule. Although I am not at present aware of a

whereby medical matters are reduced to genetic problems. 13 September 1999: "The I.Q. Gene?" In this cover we see a baby holding a molecular model of the double helix. Finally, a cover story from 5 May 1997 addresses the role of genetics in addiction: "Scientists are Discovering the Chemical Secret to How We Get Addicted...and how we may get cured." Listed along the side bar as additions are "Sex, Drugs, Drinking, Smoking." Featuring as the article on the "chemical secret" underlying drinking problems is a report on the work by Blum and Noble on the Dopamine reward system and the genetic allele responsible for the dopamine D2 receptor. Although the role of this genetic factor has been widely discredited since then (Sander, Ladehoff et al. 1999), no mention of the fact has been made on the cover of *Time* magazine.

framework in which much sense can be made of the role of both heredity and intoxicating substances in the human condition, I am hopeful that by articulating the levels at which forces mix to produce alcoholism we may someday achieve a better understanding of how and why humans develop in the ways that they do. It may be a somewhat lofty goal, but since we are obviously not making headway with the medicalization of human deviance, nor with the genetic reductionist approach to human behavior, it is something worth striving for.

In this dissertation I have collected evidence, analysis and argument to show that our current predilection for genetic explanations of alcoholism is flawed. Despite fundamental obstacles to articulating what the term “alcoholism” means, genetic and evolutionary models of it have proliferated. In the second chapter I presented an overview of the heterogeneous nature of etiological theories on alcoholism’s cause. To be sure, there has been considerable refinement in the proposed pathways for the development of alcoholism. The progression from over one hundred candidate explanations in the first half of the century to emerging consensus on the use of the Diagnostic and Statistical Manual (DSM) criteria underscores the conceptual refinement that has taken place. In conjunction with this refinement researchers have developed methods ranging from family-based studies to genetic marker analyses that generate evidence supporting a genetic model of alcoholism’s cause. However, the DSM criteria employed in the course of gathering such evidence suffer from lack of refinement. With 35 distinct ways in which a subject could meet these criteria, they make a poor foundation on which to build arguments for correlation with biological or genetic material.

In an effort to understand why hereditary, genetic, and evolutionary models of alcoholism have retained their prized status since the development of Mendelian analysis of heredity, I pondered the depths of cultural roots of views on heredity and alcoholism in

chapter 2. From the works of Plato through to the twisted utopic visions of the eugenics movement, the desire to attribute alcohol problems to a basis in heredity persist. The gene-myth for alcoholism has deep roots, and therefore any effort to unseat it must address these roots and their bearing on present issues. While a full accounting of this historical continuity has not been attempted here, I have sketched a few connections that further historical investigations can flesh out more fully. I have only scratched the surface of the link between the development of both genetic science and hereditary approaches to alcoholism within the context of the eugenics movement. The history of this movement has been explored through a considerable amount of scholarship, but the degree to which modern models of alcohol-related problems continue to remanufacture the arguments of eugenisists like George. Archdall Reid suggests that further investigations are required.

The first and second chapters have served to highlight the persistence of problems defining alcoholism. This definitional heterogeneity, coupled with the fact that human cultural uses of alcohol differ widely, renders modern social-scientific measurement and prediction of alcohol related outcomes deeply problematic. Furthermore, attempts to deploy data gathered from these contexts in the service of genetic reductions will only multiply the odds that correlations between genes and behaviors are spurious or the result of a third cause.

The aim of the third chapter was to articulate the sort of biological phenomena that could very well influence the appearance of correlations between genetic material and alcohol-related behaviors or problems. In this respect there are at least three sources of difficulty for genetic explanations. First, any adequate picture of the role of genes in the development of a character or trait must address the presence of “norms of reaction.” Without specifying the complex curvilinear patterns that are the substance of the interaction

of genetic material and environmental variables, explanations that connect genetic material with complex end states explain nothing at all. Second, as an explanatory element the mere presence of genetic material in an organism cannot stand on its own. A genetic explanation requires a whole host of biological elements and environmental contingencies whose role in the complex chain from DNA to protein products must be specified. On close examination of the particulars of the gene function in biological systems it is readily apparent that language involving “genes for” traits represents a tragic oversimplification. At the most fundamental level, the problems presented by function and development render the meaning of the term “gene” problematic. Third, with human beings these problems multiply with the addition of multiple levels of analysis and the possibility of feedback loops among them. This feature is evident at the macro-sociological level described by Ian Hacking, in what he calls the “looping effect” of human labels and kind designations. It is my contention that a robust model of human alcohol problems will need to address this problem of levels directly. Furthermore, we are required by the problem of levels to reassess patterns of heredity in light of biological elements at levels other than DNA. For this purpose I presented one type of parasitic infection that serves as a good example of how other biological elements can mimic or mask hereditary patterns.

The frugivory hypothesis, standing as the most recent deployment of the gene-myth in the attempt to explain alcoholism, provides a good example of the structure of genetic and evolutionary models. By looking closely at this hypothesis at the end of the last chapter I have put on display the gaps that require both empirical, but more importantly, conceptual substantiation. These conceptual difficulties stem from a mismatch between what we know about the role of genetic material in the development of organisms and the linear “bottom-up” model on which the frugivory hypothesis rests. The problems that belie oversimplified

models of “genes for” alcoholism stem from the sheer complexity of gene-to-product relationships and the problem of feedback through multiple levels. Without addressing complexity and non-linear, non-additive causal pathways *within* the organism, mere analysis of covariation between genetic material and diagnoses of alcoholism are inadequate.

In this fourth and final chapter I have presented an overview of what needs to be done in order to overcome a century of genetic reductions of alcoholism. While there is a promising shift in alcohol scholarship toward a focus on the “drinking career” and the analysis of alcohol-related problems in longitudinal perspective, I maintain that another avenue warrants attention. In my brief description of Developmental Systems Theory I pointed out how it may enhance our understanding the human organism throughout ontogeny, and consequently inform future explanatory models of alcohol problems. The focus on development and the systems that collude to enact it allows for the construction of a non-reductive model that may be able to accommodate the definitional problems of alcoholism that I earlier called the “problem of heterogeneity.”

Also, by focusing on the systematic interrelation of interactants in biological systems, DST offers promise as a strategy for overcoming the “problem of levels.” Since this problem threatens contemporary theories on the role of biological (and genetic) mechanisms in human alcoholism, DST appears to be a starting point from which to develop and refine our measures of alcohol-related behavior. By taking this as a fresh starting point, we may be able to develop a better way of understanding human alcoholism, and by this means perhaps flesh out more broadly an approach to the study of the human condition.

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