Section 1 Introduction

Chapter 1

Introduction to the volume

Audrey R. Chapman

The misuse of alcohol and illicit drugs inflicts a major toll on individual users, their families, and the wider society. Addictive disorders contribute to excess morbidity and mortality and are economically costly. They also disproportionately affect people in the prime of life (Merikangas and Risch, 2003). The World Health Organization (WHO) divides the adverse effects of alcohol, opioids, and other psychoactive substances into four categories: chronic health effects (such as the toxic effect of alcohol in producing liver cirrhosis); the acute or short-term biological health effects of the substance (such as the effects of drug and alcohol overdose); the adverse social consequences of substance use (such as criminal activity to obtain access); and chronic social problems (such as the impact on family life) (WHO, 2004: 10–11). In addition, alcohol and drug consumption is associated with widespread psychosocial consequences, including violence, absenteeism in the workplace, and child neglect and abuse (WHO, 2011: 24). WHO estimates that alcohol ranks eighth among global risk factors for death and is the third leading global risk factor for disease and disability (WHO, 2011: 34). Of the ten leading risk factors of avoidable burden of ill-health, tobacco was fourth and alcohol fifth in 2000 (WHO, 2004: 16–17). Alcohol-related disability is a condition that affects more than 12% of the population in the United States at some point in their life. The majority of individuals with alcohol dependence (AD) – about three-quarters – never receive treatment (Heilig et al., 2011: 670–671).

Dependence on psychoactive substances has long been thought to have a biological basis, as suggested by observations of its prevalence in some families. The breaking of the genetic code in the 1960s and the inception of the Human Genome Project to sequence the human genome in 1990 have spurred efforts to identify the genetic basis of predispositions to drug and alcohol dependence. Given the high costs and difficulties in successfully treating addiction (Sellman, 2009), there has been interest in discovering more effective approaches to treatment. It has been thought that a better understanding of the genetic contribution of addiction could lead to more effective drugs to assist in cessation of drug use with fewer adverse side effects. Relatedly, it is assumed that genotyping could also better match patients to existing pharmacological treatments for addiction (Hall et al., 2002: 1482). This volume briefly describes such scientific research as well as current progress in identifying the genetic contributions to AD and other forms of addiction.

Like other behavioral genetics research, the manner in which genetics research associated with addiction is conducted, interpreted to the public, and then translated into clinical practice and policy initiatives raises important ethical, social, and legal issues. This volume

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has a dual focus: identifying the ethical issues and requirements related to carrying out genetically based research on addiction and specifying the ethical, legal, and public policy implications of the interpretation, translation, and application of this research. There are four sections in the volume. Section 1 consists of this introduction and two other chapters, one an overview of genetic research on AD and the other on the promises and risks for participants in studies. Section 2 addresses research issues, both human subject protection issues in genetically focused addiction research and issues related to seeking or accepting support for addiction research from industry. Section 3 explores ethical and policy issues in translating addiction research for public understanding and into public policy. The concluding chapter, which constitutes Section 4, uses the key issues raised in the volume and the recommendations made by the various chapter authors to develop guidelines for research and its policy applications.

Conceptualizing addiction

Criteria for addiction

To start at the beginning, what is addiction? According to one dictionary definition, addiction is the "compulsive need for the use of a habit-forming substance (like heroin, nicotine, or alcohol) characterized by tolerance and by well-defined physical symptoms upon withdrawal" (Merriam-Webster Dictionary). Addiction is often used interchangeably with the terms "substance dependence" or the "dependence syndrome." Although, as noted below, there is ongoing debate among philosophers, ethicists, public health specialists, scientists, and the general public about the conception of addiction, there is considerable consensus about the criteria for identifying someone who is addicted. As noted from the descriptions below, the two major medical classifications of dependence have considerable overlap. Both emphasize a strong desire or sense of compulsion to take the substance in question and difficulties in controlling the pattern of use and its termination, despite clear evidence of overtly harmful consequences.

The *International Classification of Mental and Behavioral Disorders*, 10th revision, usually referred to as the ICD-10, was endorsed by the 43rd World Health Assembly in 1990 and came into use in 1994. The ICD-10 lists six criteria for substance dependence, some of which are measurable in biological terms whereas others are not. To be diagnosable as "dependent," three or more of the following must have been experienced or exhibited together at some time during the previous year:

1. strong desire or sense of compulsion to take the substance
2. difficulties in controlling substance-taking behavior in terms of its onset, termination, or levels of use
3. physiological withdrawal state when substance use has ceased or been reduced, as evidenced by the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms
4. evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses
5. progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects

6. persistance with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to heavy substance use, or drug-related impairment of cognitive functioning (WHO, 2004: 13).

The second major source of criteria for identifying substance dependence is the fourth edition of the *Diagnostic and Statistical Manual* (DSM-IV) of the American Psychiatric Association. According to the DSM-IV, substance dependence is "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":

1. tolerance, as defined by either of the following:
   a. a need for markedly increased amounts of the substance to achieve intoxication or desired effect
   b. markedly diminished effect with continued use of the same amount of the substance
2. withdrawal, as manifested by either of the following:
   a. the characteristic withdrawal syndrome for the substance
   b. the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
3. the substance is often taken in larger amounts or over a longer period than was intended
4. there is a persistent desire or unsuccessful efforts to cut down or control substance use
5. a great deal of time is spent in activities necessary to obtain the substance
6. important social, occupational, or recreational activities are given up or reduced because of substance use
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 (American Psychiatric Association, 1994).

Conceptions of addiction

There are currently two major approaches to conceptualizing addiction, both of which were developed primarily in reference to patterns of opioid drug misuse and not AD. Presumably the conception would apply to AD, as well as nicotine addiction. The traditional and popular understanding of addiction, sometimes labeled as the moral model, presents addiction as an issue of moral impropriety based on a choice that individuals voluntarily make and for which they should be held responsible. By contrast, the more recently developed medical model holds that addiction is primarily a psychiatric or brain disorder that requires treatment. Some researchers propose a third psychological approach. Chapter 13 by Toby Jayaratne, Alicia Giordimaina, and Amy Gaviglio in this volume, for example, discusses the tendency for some individuals with a propensity for AD to attempt to decrease the threat this poses to their self-esteem by employing genetic explanations as a psychological coping tactic. There are more- and less-nuanced proponents of each of these models, as well as a small number of analysts who take the position that addiction is both a disease and a moral condition (Cochrane, 2007). Conceptions of addiction have implications for how society should treat addicts and whether addicts are considered to have the capability to exercise rational or responsible agency – for example, to make an autonomous decision to participate in a genetic research study of addiction.

The dominant moral model of addiction holds that addicts knowingly and willingly choose to use drugs or alcohol without regard for the adverse consequences for themselves and others. According to this view, the choice that individuals make to use psychoactive substances springs from a weak will. Some adherents of this position recognize that in a minority of cases the decision to use harmful substances develops into an addictive pattern. Others believe that addiction is just an excuse for continuing to use drugs while avoiding responsibility for the consequences of doing so (Carter et al., 2009: 25). This perspective is sometimes also referred to as the "skeptical" view, because it discounts the relevance of biogenetic mechanisms and recent neuroscience research as well as the need for medical treatment for addiction. According to one proponent, "addiction is no more a treatable medical problem than is unemployment, lack of coping skills, or degraded communities and despairing lives…More treatment will not win our badly misguided war on drugs. It will only distract our attention from the real issues in addiction" (Peele, 1990). Many of those who subscribe to this approach to addiction contend that most addicts have the capacity to stop drug use on their own (Peele, 2004, cited in Carter et al., 2009: 25). Some of those who argue that addiction is best conceptualized as a moral condition rather than a compulsion requiring medical treatment base their views on the fact that drug seeking and drug taking involve a series of actions that require rational planning; they therefore draw the conclusion that addicts rationally decide to continue to use drugs. Others worry that medicalization might encourage drug use or lead addicted persons to fatalism about their condition (Hyman, 2007: 8–9). The common belief that drug use is a voluntary choice that results in significant personal and social harm has led most societies to adopt punitive laws to discourage drug use and to impose significant penalties for purchase and use of illegal substances or if addicts engage in harmful or illegal acts while under the influence of an addictive substance.

There are conceptions of addiction that share many of the premises of the moral model while not explicitly presenting addiction as a moral issue. Bennet Foddy and Julian Savulescu (2007) offer a self-labeled reductive account that characterizes addiction as pleasure-seeking, individually based action that is rationally decided by its user. In their account, addictive desires differ from other desires for pleasure more in degree than in kind: they are especially strong; they occur in a particular context that triggers anticipation; and they are socially unacceptable because they threaten the welfare of the individual or challenge social norms. Foddy and Savulescu also compare addiction to substances with physical dependency syndromes and the addiction to other biological sources of pleasure such as sugar, sex, eating, or water. Like advocates of the moral model, they reject the view of addiction as a disease. For them pleasure is a healthy, necessary part of an individual's life. When it becomes excessive and out of control it may be considered to be a poor choice, but not a disease. They argue that very few addicts suffer brain damage that impairs their judgment, and for the most part, the changes in an addict's brain are comparable to those of a normal person when they engage in any normal rewarding activity (Foddy and Savulescu, 2010: 6). They relegate the concept of addiction to being nothing more than "an illiberal term invented to describe those who seek pleasure in a way that expresses our social disapproval" (Foddy and Savulescu, 2010: 20). Instead their "liberal account" of addiction advocates that the pleasure of addiction can be conceptualized as a legitimate human good and can be part of an autonomous, and even rational, life plan (Foddy and Savulescu, 2010: 19–20).

By contrast, the medical or disease model of addiction, informed by neuroscience research and brain-imaging studies, presents an addict's drug-seeking behavior as the direct result of changes in the structure and function of the brain caused by chronic substance

use. Neurobiological research, particularly brain scans, suggests that chronic substance use can produce long-term disruptions of neurocognitive circuits involved in motivation and attention, decision making, and the ability to inhibit impulses. These alterations then increase cravings, impair appreciation of the consequences of substance use, and make it more difficult to resist urges to use the substance in question (Carter et al., 2009: 25–26). Some proponents of the medical model explicitly conceptualize addiction as a brain disease. The "chronic and relapsing brain disease" model of addiction put forward by Alan Leshner (1997) uses evidence that prolonged substance use causes pervasive and long-term changes in brain function to explain why an addicted person is vulnerable to relapse even after protracted periods of abstinence.

Acknowledging that the science is still in its early stages, Steven Hyman (2007) offers a nuanced and qualified interpretation of the neurobiology of addiction. He proposes that addictive drugs tap into and, in vulnerable individuals, usurp the potent neurotransmitter dopamine system in the brain that regulates rewards. The neural circuits "over learn" from excessive and distorted dopamine signals. This usurpation of the dopamine system makes drugs salient to the addict at the expense of other, more adaptive, goals. The result is a brain in which drug cues powerfully activate drug seeking and create craving if use is delayed, thus undermining the addict's ability to avoid seeking and using. Nevertheless Hyman cautions that this model does not reduce afflicted individuals to "zombies" who are permanently controlled by external cues or devoid of other goals. He also suggests that despite likely multiple relapses, addicts can regain a good measure of control over their drug taking.

Recognition of the important contribution of neuroscience does not necessarily lead to a reductive neuro-essentialist conception of addiction. Many proponents of this perspective acknowledge the importance of social, environmental, and cultural factors as well. For example, an approach that includes a neuroscientific component, but also goes beyond it, termed a "biopsychosocial systems model," proposes that psychological and sociological factors complement and are in dynamic interplay with neurobiological and genetic factors (Buchman et al., 2010: 37).

Like the moral model, the medical model has implications for how society approaches and deals with addicted individuals. Many proponents hope it will decrease the stigma associated with addiction and will incline society to treat addicts more humanely. Other advocates believe that treating addiction more as a disease than a moral failing could encourage greater societal investment into medical research into addiction and the development of more effective medical interventions (Carter et al., 2009: 25–26). However, the very possibility that societies could move in this direction makes some analysts reluctant to replace the moral model with a neurobiological perspective – both for the benefit of the addict and the protection of society.

The acceptance of the view of addiction as a disease could also have unintended negative consequences. Some worry that if addiction is viewed as primarily a genetic or brain disease it will contribute to negative perceptions of substance-use problems, much as it has in the case of mental illness (Buchman et al., 2010: 37). An uncritical acceptance of the brain disease model of addiction could encourage an overemphasis on pharmacological strategies to try to cure addiction rather than social-policy measures to reduce use of alcohol and drugs, which are more likely to be effective. In some circumstances it might also be interpreted as a warrant for the coercive treatment of addicts (Carter and Hall, 2007: 16).

An issue underlying much of this debate on the nature of addiction is the extent to which an addicted individual is in control of his or her actions, and concomitantly, the extent to

which he or she should be held accountable by society. Specifically, how severely does addiction compromise the autonomous agency of the user and what are the implications? As Gideon Yaffe notes, there is both a legitimate moral and legal basis for distinguishing among: (1) those who pursue illegal or immoral courses of action freely; (2) those who do wrong out of compulsion – that is, unfreely; and (3) those who do wrong as a result of transitory powerful impulses (Yaffe, 2001: 179). The question is into which category addicts should be placed and whether the characterization applies to all addicts. This is a complex issue that has attracted much philosophical discussion too complex to recount adequately here.

To provide a simplified characterization, at one end of the spectrum there are those philosophers, psychologists, and medical doctors who believe that the autonomy or the capacity for self-determination of addicts is severely impaired. As noted, the two major medical classifications of dependence on psychoactive substances, one compiled by the WHO and the other by the American Psychiatric Association, cite a strong desire or sense of compulsion as one of the characteristics of addiction. Compulsion, which compromises the voluntary nature of choice, is one clinical defining feature of addiction that is usually taken to compromise decision-making capacity. Intoxication and withdrawal, which compromise the ability to comprehend choices, are two others (Charland, 2002: 40–41). Luis Charland argues that "the brain of a heroin addict has almost literally been hijacked by the drug" (Charland, 2002: 43). Although he acknowledges that the decisional impairments in heroin addiction fluctuate, he argues that their brain mechanisms and systems that govern evaluation have been disrupted and reoriented, thus entrenching the damage to their decision-making capacity (Charland, 2002: 43).

Charland's characterization, which comes in an article discussing whether heroin addicts are able to give consent to participating in clinical trials of heroin replacement therapy, is countered by other characterizations of addiction. Neil Levy argues that although addicts have impaired autonomy, the evidence available demonstrates that their actual behavior is sensitive to moderate incentives, both positive and negative in nature – for example, price increases in the drugs consumed – indicating they are not subject to irresistible desires. Levy argues that autonomy comes in degrees: it is not an all-or-nothing phenomenon. Addicts are subject to oscillations in preferences and suffer from diminished autonomy, but they are still capable of choice and are able to resist taking the substance to which they are addicted at least some of the time (Levy, 2011). Although Steven Hyman acknowledges that addiction impairs the capacity to make decisions about drug use, he, like Levy, maintains that this "loss of control is not complete or simple" (Hyman, 2007: 8). Similarly, Adrian Carter and Wayne Hall stress that "the fact that individuals with an addiction retain some control over their decisions about drug use and that the impulse to use drugs is resistible must be stated clearly" (Carter and Hall, 2007: 16).

Regardless of perspectives about the nature of addiction, most ethicists, even those who acknowledge at least a partial impairment of decision-making capacity, still argue that addicts should be held responsible. A (US) National Bioethics Advisory Commission Report concluded that the disease of addiction is not an excuse for behavior *per se*, because drug-dependent individuals are not always devoid of rational decision-making capacity (National Bioethics Advisory Commission, 1998: 8). Similarly, Stephen Morse points out that although an addict's rationality is often severely compromised at the time of drug seeking and using, it is not compromised at all times for most addicts. Therefore he or she is capable of and responsible for taking steps when not in a strongly driven state to prevent the maladaptive behavior that the addict knows will result when the craving returns (Morse, 2007: 13). Steven

Hyman cautions that some apparently voluntary behaviors of addicts may not be as freely planned and executed as they first appear (Hyman, 2007: 8), but he nonetheless still believes that it may be wise for societies to err on the side of holding addicted individuals responsible for their behavior – but "with a view to the rehabilitation of the addicted person and protection of society rather than moral opprobrium" (Hyman, 2007: 10). Likewise, Thomas Cochrane argues that fully replacing the moral model with a neurobiological perspective would be counterproductive because some demonstrations of moral judgment actually work to control addictive behavior. He goes on to say that "Even proof that addicts lack *all* control would not obviate the need for a moral stance on the part of others, as long as it can be shown that such a moral stance alters the addictive behavior" (Cochrane, 2007: 25).

Further complicating this whole issue, empirical studies of dependence symptoms indicate that the severity of dependence varies along a continuum from light to moderate and then severe. The cutoff point or threshold for addiction or dependence is somewhat arbitrary. Many people who use drugs and alcohol experience problems but do not meet criteria for dependence. To engage in genetic research it is important to have a good measure of the phenotype, but current diagnostic criteria for dependence and/or substance use are often highly correlated with a variety of other possible causes and consequences, including personality traits, demographic characteristics, and psychopathology. The complicated nature of addiction makes it unlikely that single causes and simple diagnostic criteria are likely to provide clear guidance on how best to define and diagnose the phenotype (T. Babor, personal communication, 2011).

Types of genetic research on addiction

The increasing evidence that addiction to alcohol and opioid substances has a genetic contribution has given rise to research to improve our understanding of addiction and thereby to be able to more effectively treat those afflicted and possibly improve our ability to prevent at least some addictive disorders. Genetic research on addiction seeks to identify the genes associated with a predisposition or vulnerability toward dependence and addiction. Qualitative family-based research designed to examine patterns of inheritance has been a cornerstone of this research. There are several types of family studies. Classical twin studies evaluate genetic inheritance by comparing data on a trait under study from identical/monozygotic and fraternal/dizygotic twin pairs. Additive genetic influences are shared 100% between members of monozygotic twin pairs, whereas dizygotic twin pairs on average share 50% of their genes, the same degree of genetic similarity as non-twin siblings. Adoption studies of biologically related people reared apart in presumably different environments help to separate genetic and environmental influences on variation in vulnerability to substance disorders. Some researchers have also pooled data from the various types of family studies to conduct a range of meta-analyses (Baker, 2004: 42–45).

Family, twin, and adoption studies provide robust evidence for a significant, but not exclusive, genetic contribution to the development of substance use and dependence. Environmental factors and individual experiences play an important role in shaping use patterns and dependence. Twin studies strongly indicate the existence of genetic risk factors for multiple aspects of smoking and AD, including initiation, continuation, amount consumed, and cessation (WHO, 2004: 151–152). Depending on the diagnostic criteria used, heritability estimates of AD range from 52 to 63% (WHO, 2004: 132). Heritability of opioid dependence is estimated to be even higher, at almost 70% (WHO, 2004: 136). However, the various

types of family designs, with the exception perhaps of adoption studies, cannot identify the relative contribution of genetic and environmental factors (Agrawal and Lynskey, 2008). Nor can they identify which genes or chromosomes are involved.

Technological advances spurred by the Human Genome Project have made molecular approaches more readily available to investigate regions of DNA that may be involved in the susceptibility to AD and other forms of addiction. Linkage analysis, which examines genetic samples to try to identify the correlation of a trait and genetic markers among related individuals who have the phenotype in question (e.g., AD), has been an important tool for identifying the approximate chromosomal region in which some of the major genes contributing to the trait are located. Another technique, association studies, focuses on a single gene that has already been isolated, referred to as the candidate gene, to identify whether variation in this gene's alleles (alternate forms of the gene) might be statistically associated with variations in its expression by comparing people with and without the phenotype. The development of microarray analysis has accelerated the process by enabling scientists to examine thousands of genes simultaneously (Baker, 2004: 45–49; WHO, 2004: 127–128).

It should be emphasized that we are still a long way from identifying the individual genetic differences that contribute to the development of any form of substance dependence. Despite good evidence that genes contribute to addiction susceptibility, the results of qualitative family studies and molecular approaches to addiction disorders have been fairly modest thus far. The lack of commonly occurring susceptibility alleles that strongly predict addiction risk has been a major challenge to this research. The complexity of unraveling the genetic contributions to AD and other addictions precludes any likelihood that genetic research can contribute to predictive genetic screening or pharmacogenetic testing to inform treatment selection of addictive disorders in the near future. After reviewing the scientific evidence, the next chapter in this volume, contributed by Rebecca Mathews, Adrian Carter, and Wayne Hall, concludes that genetic testing is not ready for use to predict AD liability, especially for population screening, but shows that the evidence linking genetic variants with differential responses to treatment appears to be more robust for some population groups.

The complexity of the task is a major challenge to the application of genetics in the field of addiction. Contrary to the popular view of human genetics, which assumes a simple or direct relationship between a mutation or a variant form of a single gene and the development of a specific disorder, single gene or Mendelian disorders, such as Huntington's chorea, are very rare. Predisposition toward alcohol and/or drug dependence is a complex disorder, and like other complex disorders it appears to be shaped by multiple alleles (variant forms of a gene), each contributing a small effect, that dynamically interact with each other and with environmental factors. Gene/environmental interactions are key to determining outcomes. As a recent WHO review of evidence on genetic vulnerability to substance dependence explains, "while individual genetic differences contribute to the development of substance dependence, genetic factors are but one contributor to the complex interplay of physiological, social, cultural and personal factors that are involved" (WHO, 2004: 125).

There are several implications of this understanding of genetic heterogeneity. Multiple risk alleles in different combinations can contribute to genetic risk in individual cases. It is unlikely, therefore, that everyone with a particular "risk gene" for substance use or dependence will become dependent. Conversely, some of those who become dependent may not carry a specific genetic risk factor being researched (WHO, 2004: 125). Or to put the matter another way, patients diagnosed with a clinical condition labeled as alcohol dependency or

another form of addiction presenting with similar symptoms can arrive at this phenotype through very different trajectories of genetic risk factors and exposure to environmental risk factors (Heilig et al., 2011: 671).

Ethical issues in conducting and translating genetic research on addiction

Like other areas of behavioral genetics, research on addiction touches on sensitive questions about the determinants of human behavior, the balance between freedom and determinism, and the extent and ways in which we share our genetic identity with other members of our family and our broader social community. The research raises ethical issues that fall under two broad categories: the ethical issues that arise in conducting the genetic research on addiction; and the broader social and ethical implications of interpreting the research and translating it into prevention and treatment programs and social policy. The decision of the directors of the Human Genome Project, funded by the National Institutes of Health, to devote 3–5% of their total research budget to ethical, legal, and social issues related to the science attests to the significance of these issues. It is hoped that this volume will contribute to the sensitization of genetics researchers to the ethical requirements of this research and will help to inform policymakers to be cautious in interpreting and applying the research findings.

Ethical issues in human genetic research on addiction

There is an international consensus that biomedical research should conform to a series of foundational ethical principles. Informed consent to protect a subject's right to make an autonomous choice is arguably the most important of these. The informed consent process requires that potential subjects be accurately informed of the purpose, methods, risks, benefits, and alternatives to the research; that they understand this information and be able to apply it to their own situation; and also that they make a voluntary and uncoerced decision as to whether to participate in the research (Emanuel et al., 2000). Genetic research on addiction pushes the limits of the protection typically accorded by informed consent when it seeks to obtain consent from addicted individuals, who may have reduced decision-making capacity or competence. Given this concern and the complexity of understanding the implications of genetic research, it is important that genetic research on addiction take special precautions to assess whether the requirements for informed consent can be met.

Concern with vulnerability, understood in terms of the ability to give or withhold informed consent or otherwise be taken advantage of in research, has been central to the development of the Common Rule, the portion of the Code of Federal Regulations that governs much of the human research conducted in the United States. The Common Rule restricts the research that may be conducted on a number of groups – which do not include persons suffering from addiction *per se*, but also notes that others may also be vulnerable. It also requires that research protocols include protections for those who might be vulnerable but does not specify what those should be. In recent years the association of vulnerability with membership in a specific group, such as children or prisoners, has been supplemented or in some cases reconceptualized to apply to the characteristics of individual persons or the factors or conditions that may render individuals vulnerable in a specific research setting (Iltis, 2009). The potential vulnerability of subjects in research on the genetics of addiction suggests the need for appropriate protections to be designed.

Obligations to protect the privacy and confidentiality of the research data collected constitute another ethical challenge for genetic research on addiction. The right to privacy and confidentiality has special salience for genetic research for several reasons. Genetic information may be seen by individuals as central to their personal identities in ways that other medical information is not. This reflects the genetic essentialism conveyed by images and narratives found in popular culture and the media that equates human beings with their genes. Some analysts even suggest that DNA functions in many respects as a secular equivalent of the medieval Christian conception of the immortal soul (Nelkin and Lindee, 1995: 2). In addition, genetic information carries implications not just for individuals but for their families as well. Therefore the release of that information can adversely affect relationships among family members. Also the predictive nature of genetic information has the potential to adversely affect people's lives. For example, it may foster a sense of determinism that causes depression or reduces the inclination to take precautionary measures. Yet another factor is that genetic information has the potential to be used for discriminatory purposes by employers and insurance companies. Like some other areas of behavioral genetics research, a known predisposition to addiction is also likely to be a stigmatizing health condition. Protection of the confidentiality of genetic data is more complex than for other forms of medical information, because genetic data are intrinsically identifiable – that is, traceable back to the individual – and cannot be easily de-identified. The development of genomic databases and biobanks that store large amounts of genetic data and make them available to researchers, although central to the advancement of biomedical research, complicates protection of the confidentiality of research participants.

Ethical issues in translating and applying genetic research

The need to guard against genetic research being misunderstood or misused is underscored by the early history of genetic research. In the first half of the twentieth century human genetics as a program of research was intertwined with the early eugenics movement, which sought to improve the physical, mental, and behavioral qualities of the human race through selective breeding. As a result, belief in the heritability of addiction translated into negative eugenic programs to prevent the reproduction of those persons considered to be genetically defective. This latter category often had more to do with cultural beliefs and prejudices at the time than with scientific findings.

Charles Davenport, the founding director in 1909 of Cold Spring Harbor Laboratory, a facility that played an important role in early genetics research, was also a leading figure in the American eugenics movement. Davenport argued that patterns of human heritability acting through physiological and anatomical mechanisms were evident in a wide range of mental deficiencies. The mental deficiencies he identified and sought to eliminate included alcoholism as well as insanity, epilepsy, pauperism, criminality, and feeblemindedness – a catchall used for a wide range of mental problems (Kevles, 1995: 46). Davenport's interest in fostering the development of good human stock led him to advocate for a selective immigration policy that would deny entry to individuals and families with what he viewed as a poor hereditary history. He also supported the introduction of state-enforced sterilization to prevent the reproduction of the genetically defective (Kevles, 1995: 47).

Several states enacted components of the eugenics movement's program into public policies. In 1907, Indiana became the first state to adopt a law mandating compulsory sterilization of the mentally deficient. Eventually 30 US states passed such laws. Until the