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Framing Nicotine Addiction as a "Disease of the Brain": Social and Ethical Consequences*

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Abstract

Objectives—In this article, we seek to better understand how a genomic vision of addiction may influence drug prevention and treatment. Though *social* influences on substance use and abuse (e.g., peer and family influence, socioeconomic status) are well documented, biomedical intervention is becoming increasingly technoscientific in nature. We wish to elucidate how emphasizing biological influences on substance use may lead to a vision of addiction as a phenomenon isolated within our bodies and neurochemistry, not lived daily within a complex social web of relationships and a particular political economy, including the tobacco industry, which aggressively markets products known to cause harm.

Methods—We explore the emerging view of addiction as a "disease of the brain" in open-ended interviews with 86 stakeholders from the fields of nicotine research and tobacco control. Interview data were analyzed using standard qualitative techniques.

Results—Most stakeholders hold a medicalized view of addiction. Though environmental variables are understood to be a primary cause of smoking initiation, the speed and strength with which addiction occurs is understood to be a largely biological process. Though stakeholders believe that an increased focus on addiction as a disease of the brain is not likely to lead to widespread unrealistic expectations for cessation therapies, they remain concerned that it may reinforce teenagers' expectations that quitting is not difficult. Finally, stakeholder responses indicate that genetic and neuroscientific research is unlikely to increase or decrease stigmatization, but will be used by interest groups to buttress their existing views of the stigma associated with smoking.

Conclusion—We argue that the main potential harms of focusing on biological etiology stem from a concept of addiction that is disassociated from social context. Focusing on genetic testing and brain scans may lead one to overemphasize pharmaceutical "magic bullet cures" and underemphasize, and underfund, more traditional therapies and public health prevention strategies that have proven to be effective. Genetic research on addiction may fundamentally change our

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conception of deviance and our identities, and may thus transform our susceptibility to substance use into something isolated in our biology, not embedded in a biosocial context.

In November 2010, the Director of the National Institutes of Health (NIH), Francis Collins, announced the creation of a taskforce to help move all substance use, abuse, and addiction research programs within the NIH into a single, new institute (Collins, 2010). This transition would force a merger of the National Institute on Drug Abuse (NIDA), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and the drug-related research portfolios from other institutes such as the National Cancer Institute (NCI) (Seffrin et al., 2010). Supporters justify the new institute's creation by highlighting an emerging body of scientific findings: recent research in neuroscience suggests that a single mechanism underlies compulsive behavior and addiction, regardless of the particular substance or drug of abuse. Similar brain reward pathways are thought to be involved in the development and maintenance of addiction, raising the possibility of common risk factors and the hope for novel prevention and treatment strategies across substances. In addition, supporters argue that many would be better served by a single institute combining all substance use, including patients with comorbid drug use, as well as the general population, which would benefit from comprehensive early prevention efforts (Scientific Management Review Board, 2010). Collins stated that this new institute "makes scientific sense and would enhance NIH's efforts to address the substance abuse and addiction problems that take such a terrible toll on our society" (Collins, 2010).

However, the merger remains contentious; others express concern that the merger will result in less funding for drug or substance-specific research portfolios focused on prevention, as well as decreased support for psychological and social research. One concern is that an overly reductionist vision of addiction is fueling the merger in ways that may narrow the research focus, minimizing approaches that situate addiction within a complex biopsychosocial context. One coalition of tobacco prevention groups complained: "We believe that the proposed reorganization could also narrow the focus of tobacco-related research to issues related to addiction when such research, however significant, is only one of several areas in which innovation research is necessary to expand the evidence base" (Seffrin et al., 2010:2–3). Similarly, Caryn Lerman, President of the Society for Research on Nicotine and Tobacco, advised NIH director Francis Collins that if "tobacco use research is to be moved from other NIH institutes to a new Institute, retain *tobacco policy and product research* within NCI, consistent with the relevance of this subset of the portfolio to medical consequences of drug use" (Lerman, 2010:3, emphasis in original). In other words, there is a concern that addiction's etiology will be reduced to biology alone, to a "disease of the brain." No decisions have been announced regarding this possible merger.

Framing addiction as a disease of the brain with common mechanisms is one driving force behind the proposed merger, which is of special concern since NIDA claims that it alone "supports more than 85 percent of the world's research on the health aspects of drug abuse and addiction" (National Institute on Drug Abuse, 2004). Although NIDA funds prevention and environmental influences on drug initiation, maintenance, and cessation, the bulk of NIDA research funding ($519.497 million) goes to "Basic and Clinical Neuroscience," which includes work to "expand the understanding of the neurobiological, genetic/epigenetic, and behavioral factors underlying drug abuse and addiction" (National Institute on Drug Abuse, 2011). Another $116.017 million goes to "Pharmacotherapies and Medical Consequences" research, which is responsible for "medications development aimed at helping people recover from drug abuse and addiction and sustain abstinence" (National Institute on Drug Abuse, 2011). By contrast, research in "Epidemiology, Services and Prevention Research," which seeks to "promote integrated approaches to understand and address the interactions between individuals and environments that contribute to the

continuum of drug abuse-related problems," receives less funding ($260.465 million). Cutting-edge research that seeks to "elucidate the nature of the addictive process; to determine the potential use of new therapies for substance abuse, both pharmacological and psychosocial; and to decipher the long-term consequences of drug abuse on brain development, maturation, function, and structure, and on other organ systems," funded through the intramural research program," receives $90.368 million (National Institute on Drug Abuse, 2011).NIAAA similarly privileges biomedical research (Midanik, 2004). Clearly, though prevention and gene- and brain-environment interaction is important to these funding institutions, genetic and neuroscience research are prioritized, suggesting the discursive power of reductionist, biological views of addiction.

Important social consequences arise from the way neuroscience and genetic research is subtly, and at times not so subtly, constructing a new understanding of addiction as a disease of the brain or as a "genetic" or biological disorder. In this article, we use nicotine addiction as a case study through which to understand the changing nature of our perceptions of addiction and to anticipate how new frameworks will be integrated into existing public health and clinical programs at the federal, state, and local levels. Indeed, the issues and concerns we raise are not limited to the study of one phenomenon, nicotine addiction, but are relevant to a range of complex behaviors that involve substance use for which researchers seek to locate genetic underpinnings. Tobacco serves as an illustrative case because the medicalization of smoking/tobacco addiction has occurred more recently than for other substances, for example, alcoholism. It is also an arena where both social programs (e.g., clean air laws and higher taxes) and pharmaceutical interventions (e.g., nicotine replacement therapies) have been successful.

In this article we draw on in-depth interviews with 86 experts in tobacco control, nicotine addiction research, or smoking cessation to examine how genetic research on addiction (and addiction more broadly) shapes the meaning ascribed to smoking and thus affects the scope and structure of tobacco control policy. This article is organized around four key questions raised by critical analyses of this body of research. First, will genetic research change how we understand addiction? We argue that most stakeholders hold a medicalized view of addiction, though a cultural shift that would locate addiction solely within the body, especially with regard to initiation, has not occurred. However, biology is widely understood to be a factor in the strength and speed with which one becomes addicted after initiation. Second, will increased focus on addiction as a disease of the brain lead to unrealistic expectations for treatment? Our data suggests that this is not likely, but for teenagers, it may reinforce expectations that quitting smoking is not difficult. Third, will it change the way people understand cessation, cessation options, and their personal responsibility for or ability to quit smoking? We argue that this research will change what people understand to be the most effective cessation techniques, with increased emphasis on pharmaceuticals and decreased emphasis on "willpower" and modification of behaviors and environments. Finally, will it increase or decrease stigmatization of smoking? We predict that it is unlikely this research will either increase or decrease smoking stigma, but that individuals will utilize the research to buttress their existing view of stigmatization and smoking. Because of the complex nature of smoking and addiction, we join the voices of those from a wide variety of disciplines studying addiction who are calling for an interdisciplinary understanding of addiction as a biosocial or biocultural phenomenon (Acker, 2010; Courtwright, 2010; Keane and Hamill, 2010; Kushner, 2010; Vrecko, 2010; Windle, 2010).

Background

For more than two centuries in the United States there has been conflict over how to understand the excessive use of consciousness-altering drugs or substances, which has been

understood variously as a sin, crime, bad habit, moral weakness, disease, and, most recently, as a disease of the brain (Dackis and O'Brien, 2005). Tobacco use has followed alcohol and other drugs in a conceptual shift from being viewed as a habit to, in many scientific and medical circles, a disease (cf. Kessler et al., 1997; Kmietowicz, 2000). Smoking, with roots in spiritual and medicinal ceremonies in native New World cultures, has long been considered a "habit," possibly even beneficial to one's health and capable of stimulating virility and vigor and staving off syphilis (Robicsek, 2004). In contrast to alcoholism, which physicians Benjamin Rush and Thomas Trotter defined as a "disease" in the late 1700s (Conrad and Schneider, 1980), for much of the 20th century smoking was considered socially acceptable. Massive advertisement and propaganda campaigns by the tobacco industry (Brandt, 2007), coupled with the addictive nature of nicotine and its widespread use, blunted moral objections to smokers and smoking (Courtwright, 2001). In the past 30 years, however, smoking has become both more stigmatized and considered an addiction in need of intervention (Chapple et al., 2004; Goldstein, 1991; Hines, 1996; Markle and Troyer, 1979). The perception that smokers can and should be treated as patients has risen in large part from overwhelming data implicating cigarette smoke in serious health problems like emphysema, heart disease, and various cancers (Welshman, 2004). Like other drugs of abuse, smoking has been codified as a clinical problem by the American Psychiatric Association (American Psychiatric Association, 2000).

Current scientific views of addiction, including nicotine addiction, are consistent with the standard "medicalization" framework where problems and behaviors "become defined and treated as medical problems" (Conrad, 1992:209). Medicalization has moved drug use and abuse from being understood as a "choice" to one in which the addict is understood as suffering from a "disease" (Conrad and Schneider, 1980; Keane, 2002). There has been another turn in the past 20 years to what Clarke and colleagues (2003) label "biomedicalization." Biomedicalization encompasses the medicalization of traits, behaviors, and bodily processes, but with special attention toward the "increasingly technoscientific" nature of medicine, the economic drive behind such new technologies and drugs, and the transformation of bodies in such a way that creates new "individual and collective technoscientific identities" (Clarke et al., 2003:163).

It is within the context of widespread biomedicalization that the "NIDA paradigm"—the idea that addiction is a "disease of the brain"—has arisen. In this paradigm, addiction is the result of heavy drug use that changes the structure and function of the brain, making cessation difficult (Courtwright, 2010; Leshner, 1997). The NIDA paradigm is supported by prominent biological theories of addiction that implicate the "brain reward" dopamine, serotonin, and glutamate systems (including associated genes, neurotransmitters, receptors, transporters, and enzyme targets) (Li, Mao, and Wei, 2008; Nestler, 2005; Schnoll, Johnson, and Lerman, 2007), and memory-related processes (Uhl et al., 2008). Further, many of these systems are also implicated in a variety of diseases and disorders, such as schizophrenia, which is considered a risk factor for smoking (O'Daly et al., 2005).

The NIDA paradigm does not preclude and, in fact, seems to include, genetic predispositions to addiction; NIDA publications cite the estimate that 40 to 60 percent of a person's vulnerability to addiction is due to genetic factors (National Institute on Drug Abuse, 2007:8). Further, NIDA funds research that seeks to identify genetic predispositions that were initially identified through classic approaches such as twin studies: genome-wide association studies (GWAS) (Bierut et al., 2007) and recent meta-analyses of GWAS (Thorgeirsson et al., 2010) are part of the body of literature that utilizes the most recent techniques for examining possible genetic influences on smoking initiation, maintenance, and cessation (Amos, Spitz, and Cinciripini, 2010). Smoking thus parallels a common understanding of alcoholism: certain people are vulnerable, and once an individual begins

smoking, those with certain genetic variants are at higher risk to drink or smoke heavily and have extreme difficulty quitting (Amos, Spitz, and Cinciripini, 2010).

Smoking research examines not just the effect of smoking and drug use on higher brain functions or genetic susceptibility to addiction *after* initiation, but also the effect of genes on higher brain function as it relates to susceptibility to *initiate* drug use. Recent studies indicate that genes in the chromosomal region that code for brain-derived neurotropic factor (BDNF) are associated with smoking initiation (Tobacco and Genetics Consortium, 2010). Similarly, some NIDA-funded researchers argue that genetically-based personality traits like risk taking and impulsivity may predispose certain individuals to experiment with drugs, including tobacco (Kreek et al., 2005). Neurogenetic research claims that people may become addicted to drugs for reasons that include genetic predispositions, personality predilections, and risks from a variety of comorbid disorders and diseases such as schizophrenia (Courtwright, 2010; Keane and Hamill, 2010; Kushner, 2010).

Taken together, this research, including, but not limited to, the NIDA paradigm, implies that the potential to become addicted is an inherent part of our bodies—addiction rests within us waiting to be triggered (Schull, 2003) and everyone is at risk. Individuals become addicted not just to the drug, but to neurotransmitters like dopamine and serotonin released in the brain (Keane and Hamill, 2010; Schull, 2003). Even for those without genetic or other risk factors, because of the power of the reward system of the brain, these artificial substances purportedly “flip a switch” in the brain such that, once activated, drug use moves from being voluntary to involuntary and “beyond one’s control” (Brower, 2006), especially as continued heavy use changes an individual’s brain structure and function.

Imaging studies, including positron emission tomography (PET), magnetic resonance imaging (MRI), and functional MRI (fMRI) (Illes, Kirschen, and Gabrieli, 2003; Li and Sinha, 2008; Volkow et al., 2007) have intensified understandings of addiction as a brain disease in profound ways because of the power of brain images to make objective claims not only about our brains, but also about who we are (Dumit, 2004). As Wayne Hall has noted, “a ‘disease’ that can be ‘seen’ in the many-hued splendour of a PET scan carries more conviction than … self-reports of addicts who claim they are unable to control their drug use” (Hall, Carter, and Morley, 2003:867). Because brain images are fluid signifiers that are easily (if at times inaccurately) made meaningful by society and laypersons (McCabe and Castel, 2007), they can serve different meanings simultaneously (Dumit, 2004). As Campbell states, “the social power of this tool is lodged in its potential to change public perceptions—to effectively change the ‘truths’ upon which members of the public proceed on the path to recovery” (Campbell, 2010:100). As a consequence, independent of researchers’ intentions and prior to any clinical applications, neuroimaging research on addiction is creating novel relationships between brain images, “brain types,” and individual perceptions of self and disease that have implications for societal and clinical responses to nicotine addiction as well as for the subjectivity of smokers.

A conception of addiction as a disease of the brain also has potential legal implications, though these are more relevant to illicit drugs and alcohol than to nicotine. Legal responsibility is distinct from the public or lay understandings of responsibility discussed earlier, and the courts have consistently ruled that addicts are responsible for illegal behavior (Powell v. Texas, 1968; Traynor v. Turnage, 1988; United States v. Moore, 1973). However, the disease model of addiction has influenced sentencing—addicts are still legally responsible for their actions, but the consequences of a guilty verdict may be mediated by their addiction. For example, the rise of drug courts presents a significant deviation from traditional adjudication, in which nosologic categories were irrelevant (Nolan, 2002). Drug courts offer an alternative that provides a space for law and therapy to be “a fully

collaborative enterprise" (Nolan, 2002:1726). In drug courts, addiction is understood as a disease and not the result of poor moral choices; this model provides the basis for the courts to "coerce" treatment and replace prison with heavy surveillance and treatment (Tiger, 2011).

These new biological connections are products of novel scientific approaches and provide insights that may help in the development of new cessation technologies; however, it is critical to contextualize them within the sociology of science, which details the intimate relationship between scientific research and its sociocultural context (Haraway, 1988; Harding, 1986; Latour, 2004; Longino, 2002; Sprague, 2005). But as Kushner points out, "the fact that science, like everything else, is socially constructed in no way diminishes its explanatory power" any more than it limits the value of social or historical analyses (Kushner, 2006:138). Although some social analyses of addiction have eschewed the work of biologists (cf. Levine, 1978; Moore, 1992), the definitive language used by the biological model also often excludes the important social, cultural, and structural influences on drug use and abuse. Because of the social and economic power of biomedical research and its centrality as an explanatory frame accounting for socially undesirable behavior, focusing on the biological may in fact broadly influence both stakeholder and public opinion about addiction in ways that yield unintentional consequences for addiction prevention, treatment, and research.

Methods

To discern how stakeholders concerned with nicotine addiction understand neurogenetic research, we interviewed 86 professionals from around the United States (20 scientists, 25 tobacco prevention specialists, 19 clinicians who specialize in nicotine addiction and smoking, 11 representatives of pharmaceutical companies with tobacco cessation products on the market or in development, and 11 health payers) (see Table 1). Because of the diverse nature of tobacco use as a health problem, it was essential to include stakeholders across a wide variety of professions: those developing and marketing new therapies, those treating patients seeking help for addiction, those creating prevention programs or formulating public policy, and those who make decisions about what products will be insured by health payers. Interviews with key informants provide an account of their understanding of genetic research and suggest how they might act in the face of emerging genetic findings, thus providing critical insight into the potential impact of genetic research on policy, clinical practice, and prevention (Patton, 1987). Experts were queried on a variety of topics, including: the etiology addiction; the potential effects of new technologies on public health campaigns; the stigmatization of smokers; and smoker and public conceptions of smoking, addiction, and smoking cessation (see Table 2).

Interviews lasted from 30 to 75 minutes and were recorded and transcribed. Interview transcripts were coded according to common themes generated by the responses (cf. Clarke, 2005). We sought to identify ranges of responses to specific questions, but also allowed for new themes to emerge from the data (Corbin and Strauss, 2008). Data from these themes were distilled into memos, where more cohesive descriptions and analysis of the text were developed. We made every effort to include the range of responses, including marginal opinions (or "counter themes") in our analysis (Clarke, 2005).

Results

Will Genetic Research Change How We Understand Addiction?

When asked what addiction was, about half of all stakeholders (almost all preventionists and health payers, half of clinicians and scientists, but only one pharmaceutical employee)

described addiction in a way that placed it squarely within a medicalized realm, but that did not necessarily indicate etiology. These stakeholders defined addiction by either deferring to the current version of the *Diagnostic and Statistical Manual* (*DSM*) (American Psychiatric Association, 2000), or utilizing concepts including compulsive use, "needing the drug to feel well," loss of control, "bad decision making," craving, inability or difficulty quitting, increased tolerance, or use that interferes with a productive lifestyle. These results may be consistent with either environmental variables or biological ones; etiology is not clear. Examples include:

> An addiction is something that once you start doing it, the behavior or the drug or whatever it is, that you find it hard to stop, despite it having negative impacts on your health or how you feel. (clinician practicing on the East Coast)

> Nicotine addiction … also leads to other diseases, … but I think in and to itself, it's a disease." (tobacco cessation specialist in a state public health department in the Pacific Northwest)

Our interviews show that addiction is widely understood as an uncontrollable behavior, with individuals unable to quit on their own. These statements are consistent with the fact that diagnosis for substance-use disorders remains focused on behavior; although there is increasing use of pharmaceutical interventions for addiction (e.g., nicotine replacement therapy and non-nicotine pharmaceuticals like bupropion and varenicline), treatment programs continue to focus on behavior modification in order to facilitate smoking cessation.

Probes about addiction etiology yielded a stakeholder view of addiction that is consistent with the conception that the potential for addiction exists in our bodies prior to drug use, waiting to be triggered, especially for those with certain genetic profiles. Most stakeholders believed the environmental factors (e.g., peers, family, stress) to be the primary causal factor for smoking initiation, but the speed and severity with which one became dependent on tobacco was determined by biology—once you initiate smoking, it flips a biological "switch" that perpetuates the behavior. A typical comment was:

> I think for initiation, stress and peer pressure [are most important]. For maintenance, once you start, then I think that's when the genetics kick in. (pharmaceutical employee: director for development of a new cessation aid)

Only three stakeholders in our sample of 86 believed addiction was solely the result of social variables, which indicates a fairly high degree of medicalization in this sample of stakeholders.

Almost everyone in our sample understood addiction as a disease or problem largely consistent with *DSM* diagnoses. However, stakeholders also identified environmental, social, and cultural variables underlying drug use and abuse, which indicates that they do not hold a view of addiction that is exclusively consistent with "biomedicalization." Even so, most of our sample believed that the degree and speed with which one becomes addicted is governed by one's genes.

Will Genetic Research Lead to Unrealistic Expectations for Treatments?

Addiction biology research increasingly relies on technological processes to probe etiology, including a variety of studies utilizing genetics, brain receptors, and new imaging technology. For example, in May 2006, the U.S. Food and Drug Administration (FDA) approved varenicline as a smoking cessation drug. Varenicline, marketed by Pfizer as Chantix, represents a new class of drugs whose development was possible only with increased understanding of nicotine's effects on the brain. Varenicline mimics nicotine's

ability to bind to specific ion channels in the central nervous system, which both moderates craving and lessens the effect of nicotine if one smokes (Yarnell, 2005).Another potential therapy is a nicotine vaccine (NicVAX, by Nabi Biopharmaceuticals), currently in FDA Phase III clinical trials. The nicotine vaccine induces the body to produce antibodies that bind to nicotine and prevent it from acting on receptors in the brain (Hall, 2005). These pharmaceuticals are two among many: Spectra Intelligence reports that there are nearly 35 drug candidates in various stages of development and testing for treatment of nicotine, alcohol, and narcotic dependence (cited in Thayer, 2006). These numbers illustrate the economic potential of these drugs for the pharmaceutical industry: in 2005, nicotine addiction therapies alone totaled $1.5 billion in sales (Thayer, 2006).

Although neurogenetic research is likely to lead to better smoking cessation therapies and targeted treatment, it may also lead to unrealistic ideas about the possibility for “magic bullet” treatment interventions that suggest anyone could quit smoking with minimal difficulty (Hall, Carter, and Morley, 2003). Although expanding options to help people quit smoking is useful and needed, even varenicline’s manufacturer acknowledges that in clinical trials over half of those who use this drug to quit smoking will return to smoking within 12 weeks, which demonstrates that even highly promising treatments are not magic bullet cures (Pfizer Inc., 2011). Further, varenicline’s side effects, which potentially include “changes in behavior, hostility, agitation, depressed mood, suicidal thoughts or actions” (Pfizer Inc., 2010), raise questions regarding the side effects of these promising treatments.

Interviewees pointed to their concern about possible unrealistic expectations regarding the power of new drugs to “cure” addiction. Many stakeholders, for example, were concerned that scientific and public focus on the biological bases of addiction and new drugs to treat addiction, like the nicotine vaccine, would lead both clinicians and smokers to believe one could just prescribe or receive a pharmaceutical “fix” for addiction. Roughly half the clinicians and some of the prevention workers were concerned that the public (or smokers) would perceive such new pharmaceutical therapies as magic bullet cures, and thus leave them with a false sense of safety that they could quit smoking at any time without difficulty.

> [People might think that the vaccine is] a be all, end all, which is what they always think new drugs are. … it’s like, “Okay, I don’t have to change my behavior because there’s a magic fix at the end of the rainbow.” (prevention worker: program manager for a state health department in the South)

> There’s no silver bullet to treat nicotine dependence. I think we need a retinue of agents. One could be potentially in quotes, a “vaccine,” some kind of antagonist that would block specific receptor sites. But, having said that, we have stuff that works. We know comprehensive, population-based campaigns are highly effective. We know the current treatment of nicotine dependency is effective, despite what some folks would say. (prevention worker: teaching and researching at a public health department in the Northeast)

These quotations reflect a recurrent theme in the interviews: the biomedical model of addiction fails to provide all the tools necessary to address addiction—a fact acknowledged by neuroscientists and geneticists. Although genetic researchers accept that there is scientific support for both a neurobiological and environmental/social basis for addiction (Biglan et al., 1995; Graham et al., 2006), the certainty implied by biomedical language used to describe addiction as a disease of the brain obscures and minimizes the many unknowns about the relationship between specific genes and nicotine addiction. Economic forces also provide strong motivation for the pharmaceutical industry and the tobacco industry to emphasize the biological and genetic nature of addiction, promote some drugs as acceptable to use and some as not, and to dictate what therapies are appropriate for addiction

(Campbell, 2010; Gundle, Dingel, and Koenig, 2010; Kushner, 2010; Rasmussen, 2010). Social support and behavioral therapies, though often underplayed in these biological frameworks, are nevertheless critical to success (Acker, 2010; Benowitz, 2008).

Will Genetic Research Change the Way People Understand Cessation, Cessation Options, and Their Personal Responsibility for Quitting?

Given that an individual's expectations can influence his or her experience, physiology, and behaviors such that the individual's expectations are confirmed (Barskey et al., 2002; Kirsch, 1985), knowledge of a genetic risk for addiction may predispose the individual to be more likely to smoke or less likely to quit smoking. Thus a critical part of anticipating the impact of neurogenetic research on existing medical and health practices will involve paying careful attention to issues stemming from the social *significance* and *meanings* attributed to neurogenetic information, and further recognizing that these meanings may vary significantly across social groups. In other words, those who feel "fated" to be addicted to nicotine may see no need to abstain. Stakeholders, in general, felt that overfocusing on a biomedical model of addiction may lead to a fatalistic attitude in some smokers. A typical comment was:

> One thing that might happen is that, just like the obesity stories, that folks will sort of default and say, "Well, it's genetic, I can't help it." Or, "I have trouble quitting," or, "I can't quit." Or, you know, "I was born with it." I mean, there are all kinds of ramifications that spill from things. (clinician: from the Midwest)

Despite these concerns, the most common response across all stakeholders was that genetics would make no overall impact on behavior at a population level, either because people would respond in diverse ways to genetic knowledge or, especially in the case of teenagers, because a predisposition would not be as pertinent to their choices as peer or other environmental influences.

> [Genetic information] won't make a difference—they're going to do whatever they want to do. There are some proportions of people, and we don't know how many, who will actually be made more motivated not to smoke by that information. And there are some people who may in fact adopt a fatalistic approach: "Oh, well, I've got the gene. Nothing I do matters." (scientist: head of a genetics lab, southern university)

> Teenagers are not able to make informed decisions about anything that has long-term consequences. We *know* that. So why give them the information when there's very little chance that it's actually going to help their behavior in a positive way? That there's a chance that it could well alter it in a negative way. (prevention specialist: director of a midwestern state's tobacco control program, emphasis in original)

Empirical investigations are beginning to explore how people will respond to genetic information about individual risks for tobacco dependence or smoking-related disease. Studies asking people to respond to hypothetical genetic information in the form of vignettes (Sanderson and Michie, 2007; Sanderson and Wardle, 2005; Wright, Weinman, and Marteau, 2003; Wright et al., 2006) and those providing individuals with actual genetic test results regarding risk for smoking-related disease (Bize et al., 2009; Carpenter et al., 2007; Hishida et al., 2010; McBride et al., 2002; Sanderson et al., 2008, 2009, 2010) indicate that genetic tests may increase people's motivation to quit smoking. The latter studies, however, do not indicate an actual increase in long-term quit rates. Although many of the stakeholders we interviewed feared a fatalistic reaction among smokers to the genetics of addiction, there is little evidence of increased fatalism among these studies' participants. This finding may have resulted from selection bias: those who choose to take part in these studies are eager to

receive genetic information and, because motivation to quit increased *regardless of the result* of the genetic test, these smokers may already be more motivated to quit and use the genetic results as additional motivation (Sanderson et al., 2008; Sanderson and Wardle, 2005). Though the worst-case fear of widespread fatalism appears to be largely unfounded, if stakeholders *believe* that fatalism is a concern, they may resist integration of genetic information into tobacco prevention and treatment programs. These studies found mixed results for other concerns stemming from genetic tests, including depression (Sanderson et al., 2008; Sanderson and Wardle, 2005) or complacency in the face of negative genetic tests (Sanderson and Michie, 2007; Wright et al., 2006).

Another consistent finding in these empirical studies is that patients who received positive genetic results are more likely to use pharmaceutical aids and, in one case, less likely to rely on willpower to quit smoking (Carpenter et al., 2007; Marteau and Weinman, 2006; Wright, Weinman, and Marteau, 2003). This finding is consistent with other studies investigating individuals' responses to genetic tests, which indicate that receiving positive genetic tests make people less likely to attempt lifestyle or environmental changes to improve health and more likely to rely on pharmaceuticals (Marteau et al., 2004).

This latter finding indicates that genetic tests are changing how we think about treatment options and, because biomedical models are increasingly central to how we view ourselves, new medical and genetic tests and treatments will inevitably change how we think about our bodies and express our identities (Rose, 2007). Within this larger historical context, genetic susceptibility operates as a new third option between normal and pathological; genomic medicine suggests a concept of symptom-less diseases and makes "hidden" traits central to our "diagnostic and therapeutic hopes" (Rose, 2007:84). Within this framework, our potential to become addicted becomes a trait in itself, independent of drug use (Schull, 2003). This conception of addiction fundamentally shifts addiction from compulsive drug use to something latent inside of us, detectable, independent of the presence or absence of substances in the environment or of any negative social or constitutional effect of our drug use. When addiction is thus embedded in the body, doctors become detectives who seek to understand the "truth" of addiction (Keane, 2002). The risk of these new conceptualizations of addiction lies in trying to "identify, treat, manage, or administer those individuals, groups, or localities where risk is seen to be high" (Rose, 2007:70). Disconnecting drug use from its cultural context drastically oversimplifies its causes and obscures the uneasy way we distinguish those who are "addicted" from those who are "not addicted" (Keane and Hamill, 2010; Kushner, 2010). The potential for individual fatalism obscures a more fundamental and basic risk about our perception of drug use as a disease of the brain instead of as a biosocial phenomenon.

Harm, Stigma, and Ethics: Will a Biological Understanding of Smoking Increase or Decrease Stigmatization?

A major focus of many ethical analyses of the application of genetic knowledge is the potential for stigmatization and discrimination (Nelkin and Lindee, 1995). Many ethicists argue that the possibility of harm from disclosure of genetic information is augmented by the fact that researchers have reported correlations between the genes associated with nicotine addiction and addiction to other substances like alcohol (Dani and Harris, 2005; Lê et al., 2006), other behaviors like gambling (Petry and Oncken, 2002), and personality traits like risk taking (Kreek et al., 2005), as well as various mental states and diagnoses like schizophrenia (Martin and Freedman, 2007). As a result, genetic tests to determine an individual's susceptibility to nicotine addiction or to tailor smoking cessation treatment could reveal collateral information about other susceptibilities. The potential for stigmatization may increase given the perceived association of smoking with other behaviors and the degree to which any one behavior is stigmatized (Goffman, 1963).

For about a quarter of stakeholders, the idea that genetic testing could help target those at "high risk" for nicotine addiction with education messages, prevention programs, and treatment outweighed the possibility of harm.

> [T]he basic idea is that a lot of diseases, including nicotine addiction, are heritable, and just knowing that there's some family history can be informative. But if there's a genetic test that can specifically identify someone's risk, that's even better …. Because there might be targeted preventative measures and targeted interventions. (physician/scientist: clinical researcher at a large midwestern university)

However, many stakeholders articulated problems that may arise from trying to quantify risks for complex behaviors, including the difficulty of assessing "risk" and the possibility of risk identification leading to privacy concerns, discrimination by employers or health insurance companies, or increased stigma for those deemed susceptible.

> [A test] can be used as a weapon, and not as a good thing. … if we did identify it early, would it mean you wouldn't have access to health insurance, or you would pay more for health insurance, or, or those kinds of things? (prevention specialist: from a state department of health, Pacific Northwest)

Other theoretical concerns raised by social scientists, however, were not raised by these stakeholders, including problems of living a life of "risk" devoted to disease surveillance (Koenig and Stockdale, 2000) or the psychological impact of the construction of new categories of "potentially" ill people (Rose, 2007). For instance, although stakeholders understood that some people may become fatalistic about their ability to quit smoking, no stakeholder mentioned that people may become depressed about learning about increased susceptibilities, which initial research indicates is a possibility (Sanderson and Wardle, 2005).

In addition, racial differences in smoking rates and findings of genetic differences in drug metabolism must be contextualized within the historical context wherein drugs associated with marginalized racial groups have been more likely to fuel social concerns and criminalization (Acker, 2010; Courtwright, 2001). The biomedicalization of addiction could therefore promote racial discrimination and stereotyping (Dingel and Koenig, 2008). For example, African Americans are less likely to be prescribed pain medication than white counterparts with the same ailments (Anderson et al., 2004; Tamayo-Sarver et al., 2003), and regulatory surveillance of prescription drug use reduces legitimate use of legal drugs in African Americans (Pearson et al., 2006). Further, a reductionist framework that links smoking with genes promotes a biological view of human difference, ignoring a long history of racial oppression and targeted advertisement by the tobacco industry (Barbeau et al., 2005; Jain, 2003).

Stakeholders' opinions varied on whether the medicalization of addiction would decrease or increase the stigma associated with drug abuse. Roughly a third of prevention workers, scientists, and clinicians thought that a genetic understanding of nicotine addiction would increase stigma.

> When [genetic information] converges with the denormalization policies of the public health community, that could lead then to ultra-denormalization of people who have the genetic risk factors, and I'm concerned about that. (scientist: researcher from a large, nonprofit organization)

> Even if you have all the genes to prove that, yes, this group, or a particular race/ethnicity, or during this age group, or this gender, … is more likely to have [a genetic predisposition] … it is very important for us to know what is the percent [risk] from the genetic, versus what is the percent [risk] from the environmental

> factors, and what these environmental factors are affecting or influencing … There was a lot of stigma related to the Hantavirus. And it is something not needed. And the Native American did not need to be, once again, highlighted as, "this is an illness of the Native American." And we do that too often. (prevention worker: state health department in the Southwest)

Alternately, the recognition of a genetic contribution to stigmatized conditions may result in destigmatization by shifting responsibility for the trait or disorder away from individuals' choices or will, and onto their genetic makeup, over which they have no control (Lubman, Yucel, and Pantelis, 2004; Stein, 1999). Locating causal biological factors has brought funds and research to and alleviated stigma from disorders like epilepsy and depression (Jilek-Aall et al., 1997; Schreiber and Hartrick, 2002). In fact, about a third of our interviewees thought that new genetic research would destigmatize smoking.

> When the understanding is transferred from character and morality to physiology, it tends to affect a stigma. We saw that with HIV, where initially, most of the focus was on "what the people did," if you will, and less focus on the course of the disease. It was once prevalent with epilepsy, where people with epilepsy were severely stigmatized, and the understanding that this was just a neurological disorder helped destigmatize [it]. (scientist: vice-president of research at a private health research and data analysis company)

However, individuals in our sample also recognized possible benefits from stigmatization. Not simply victim blaming, stigmatization is also an efficient and beneficial process for changing social norms when the stigmatized behavior is clearly unacceptable, or poses important public health risks (Kim and Shanahan, 2003).

> I guess this is a little bit anecdotal, but a lot of people come into my clinic and they want to quit [smoking] because their friends hate it. You know, I think the stigma is earned and well-deserved, and I think if that's the truth, that's fine. If it works, great. (scientist: researcher at a cancer center in a large, midwestern university)

Despite some ethicists' warnings about increased stigma as a harm, our interviews suggest that different interest groups will use this knowledge for their own agenda. As such, a genetic etiology likely will both reinforce and diminish the stigma associated with smoking depending on the context and motivation of the stakeholders involved. Therefore, analyses of the social consequences of genetic explanations of smoking must also take into consideration the existing sociopolitical dynamic promoting the stigmatization of smoking as an integral (and successful) component of North American smoking control policies (Bayer and Stuber, 2006; Kagan and Nelson, 2001).

Conclusion

With rapid changes in both genetic and imaging technology fueling research into the biology of substance use, drug addiction is increasingly understood and portrayed as a disease of the brain. The biomedicalization of addiction has positive implications, including the potential for novel therapies, an increased understanding of drugs' effects on the brain, and an increased willingness of doctors and health-care providers to ensure that smokers have adequate medical assistance to quit. However, the ethical questions and social concerns raised by this conceptual shift are numerous.

Focusing predominately on the biology of nicotine addiction may draw our attention away from social and contextual variables, which in turn may undermine the logic behind current public health and tobacco control efforts to curb smoking. These efforts have their genesis in the 1964 *Surgeon General's Report on Smoking and Health*, which was the first official

statement linking cigarette smoking to cancer and cardiovascular disease (U.S. Public Health Service, 1964). This report had an almost immediate effect on policy and, by many accounts, the anti-smoking campaigns that followed this report have been major public health successes (Jacobson and Zapawa, 2001; Zhang et al., 2006).

Given resource constraints, decisions about how to invest resources—local, state, federal, and private—are highly political and subject to political framing. Based on empirical evidence, many would argue that the most cost-effective, as well as purely efficacious, programs to prevent or treat nicotine addiction are in the realm of the social: smoke-free ordinances and increased cigarette taxes (Chaloupka, Wakefield, and Czart, 2001; Jacobson and Zapawa, 2001; Meyers, Neuberger, and He, 2009; Zhang et al., 2006). From a public health perspective, applications deriving from biologically-based applications are not cost effective (Hall, Gartner, and Carter, 2008). The counterargument suggests that public health programs have "run their course" and pharmaceutical innovations, possibly individually tailored with genetic tests, may help with the approximately 20 percent of smokers who continue in the face of aggressive public health campaigns (Warner and Mendez, 2010). Even so, some scholars have argued that nicotine addiction may be one area where traditional public health measures are more effective than genetic-based therapies, particularly since studies have shown that the social transmission of smoking is at least as important as its heritable aspects (Carlsten and Burke, 2006; Merikangas and Risch, 2003). Genetic research and testing could shift resources and interventions away from upstream social, political, and economic causes of smoking behavior to downstream clinical interventions, making local and state governments less willing to invest in programs not consistent with the popular (genetic) theories of addiction. Given that some successful public health programs have already been de-funded (Givel and Glantz, 2000; LaPelle, Zapka, and Ockene, 2006), and state budgets are increasingly tight and contentious, de-funding proven public health programs may occur more often.

Genetic and neuroscience research is but one more chapter in a long history of changing definitions and understanding of addiction. Most stakeholders in our sample subscribe to a medicalized vision of addiction that is generally consistent with *DSM* diagnoses. When asked about addiction etiology, most stakeholders believe social variables are paramount for smoking initiation, but understood biology to be more important for predicting the rapidity and severity with which an individual becomes addicted. This latter framework is consistent with a large body of research examining both genetic predispositions for smoking and pharmaceutical therapies for smoking cessation. Shifting our focus to the biological, individual aspects of drug addiction may increase teenagers' beliefs that quitting is not difficult with the appropriate pharmaceutical aids, and create a widespread shift toward pharmaceutical therapies and away from considering an individual's commitment to quitting, including the role of social and environmental variables. Our changing understanding of addiction is unlikely to either increase or decrease stigma in a straightforward way, but will be incorporated into existing frameworks of prevention and treatment in unique ways. Describing addiction as a disease of the brain and proposing institutionalizing this concept through a reorganization of our federal funding of addiction research promotes a simplistic vision of substance use, one that does not allow for the integration of neuroscientific and genetic approaches with a robust account of the social dimensions of addiction.

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TABLE 1

Description of Sample and Recruitment

Title	Description	Sampling and Recruitment	Number of Interviews
Scientists	Psychiatrists (n = 13), neuroscientists (n = 4), geneticists (n = 1), and other researchers (n = 2) who investigate neurogenetic links to nicotine addiction	Identified through scientific publications, professional meetings, and National Institutes of Health (NIH) grant awards, participation in Transdisciplinary Tobacco Use Research Centers (TTURCs), and by "snowball" sampling	20
Clinicians	Clinicians and researchers involved in smoking cessation, tobacco management, and the treatment and prevention of tobacco-related disease	Sampled from each of the 10 regions outlined by the Department of Health and Human Services (HHS), with two participants from each region (U.S. Department of Health and Human Services, n.d.)	19
Tobacco prevention specialists	Policymakers in the public and private spheres, including federal (n = 2), state (n = 19), and local (n = 2) health policy officials in tobacco control and health prevention; public health educators involved in developing and administering nationwide tobacco prevention programs (n = 2)	Sampled from the 10 geographic regions defined by the HHS (U.S. Department of Health and Human Services, n.d.) as well as federal sources; two stakeholders from each HHS region 1, 3, 5, 6, 7, 8, 10; three from each HHS region 9, four from HHS region 4, one each from HHS regions 3 and 2; two from federal sources	25
Health payers	Individuals involved in making coverage decisions for large health-care providers	Identified using Internet resources and company media contacts; participants had ties with medical assistance programs or worked for one of six large health-care companies; state Medicaid program in HHS Region 1 (n = 1); state-level private health-care companies in Region 9 (n = 4); state-level private health-care companies in Region 5 (n = 1); people involved at national level (n = 5)	11
Pharmaceutical employees	Bench scientists in research and development (n = 5), clinicians and others running trials (n = 4), communication and advertising specialist (n = 1), and drug representative (n = 1)	Recruited through personal contacts; represent 6 different pharmaceutical companies that market or are developing tobacco cessation products	11

NOTE: This table also appears in Dingel et al. (2011).

TABLE 2

Relevant Questions from the Interview Guide

Relevant Questions from the Interview Guide
Assuming that you could predict risk, what do you think would be the impact on individuals if you told them that they had a genetic variance that increased their susceptibility (or affected their tendency) to smoke?
Do you think this information could alter individuals' understanding of their smoking behavior or health?
Is it your impression that current tobacco control programs have used stigma as a way of reducing smoking? What do you think about this approach? Do you think that the recognition of a genetic component to smoking would affect the stigma associated with smoking?
Some pharmaceutical companies are developing vaccines that can be used to prevent and treat nicotine addiction. One blocks the effects of nicotine by stopping it from entering the brain.
Do you see any potential benefits or drawbacks?
Do you think it would influence people's judgment of addiction? (For example, less stigmatized?)