Neuroscience Research on the Addictions: A Prospectus for Future Ethical and Policy Analysis

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Abstract

The increasing evidence that many addictive phenomena have a genetic and neurobiological basis promises improvements in societal responses to addiction that raise important ethical and social policy issues. One of the major potential benefits of such research is improved treatment of drug addiction, but in order to do the research required to realize this promise, it will be necessary to address ethical doubts raised about the capacity of addicted persons to give free and informed consent to participate in studies that involve the administration of drugs of dependence. Neuroscience research on addiction promises to transform the long running debate between moral and medical models of addiction by providing a detailed causal explanation of addiction in terms of brain processes. We must avoid causal models of addiction being misinterpreted as supporting simple-minded social policies, e.g., that we identify the minority of the community that is genetically and biologically vulnerable to addiction and hence can neglect social policy options for reducing addiction, including drug control policies. Causal accounts of addiction supplied by neuroscience and genetic research may also be seen to warrant the use of pharmacotherapies and drug vaccines under legal coercion. Neuroscientists also need to anticipate the ethical issues that may arise if the knowledge that they produce delivers interventions that enhance human cognitive and other capacities. Advances in neuroimaging that enable us to identify “addicts” or predict future risk of addiction will raise concerns about invasion of privacy, third-party use of neuroimaging data, the powers of courts to coerce defendants to undergo such tests, and consumer protection against the overinterpretation of test results. Given the strong public and media interest in the results of their research, neuroscientists and geneticists have a moral obligation, and a professional interest, to minimize popular misunderstandings of their work in the media that may rebound to its detriment.
1. Introduction

Addiction to alcohol, tobacco, and illicit drugs are common problems among adults in many developed societies. In the United States (Anthony & Helzer, 1991; Helzer, Burnam, & McEvoy, 1991; Kessler et al., 1994) and Australia (Andrews, Henderson, & Hall, 2001) in any year around 25% of adults are dependent on tobacco, 7% are dependent on alcohol, and 2% are dependent on illicit drugs. Addiction to tobacco contributes substantially to the global burden of disease via increased premature deaths and years of life lived with disability (Ezzati, Lopez, Rodgers, Vander Hoorn, & Murray, 2002; Murray & Lopez, 1996). Addiction to alcohol and other drugs causes substantial suffering to persons afflicted by addiction and their behavior adversely effects their families and others in the community through motor vehicle accidents, violence, assault and crime, and impaired work performance and parenting (Collins & Lapsley, 2002; National Academy of Science, 1996).

Neuroscience and genetic research promise to improve our understanding of addiction to nicotine, alcohol, and other drugs and thereby increase our ability to treat those afflicted by addiction and possibly our capacity to prevent addictive disorders (Adler, 1995; Cami & Farre, 2003; Independent Working Group on Brain and Mind Disorders, 2003; Leshner, 1997; National Academy of Science, 1996). There is good evidence from twin and adoption studies, for example, that there is a substantial genetic contribution to vulnerability to addiction to alcohol, nicotine, and other drugs (Ball & Collier, 2002; Hall, Madden, & Lynskey, 2002; True et al., 1999). There are also promising candidate genes that may explain this vulnerability (Ball & Collier, 2002; Tyndale, 2003), although as yet few of these have been consistently replicated and many of the associations are modest (Tyndale, 2003).

Neuroscience research on addiction indicates that it is increasingly likely that the basis for the genetic vulnerability to addiction reflects variations in metabolism of drugs of dependence and in neurotransmitter function in key brain regions where psychoactive drugs act (Cami & Farre, 2003; Leshner, 1997; National Academy of Science, 1996). The chronic use of psychoactive drugs produces long lasting changes in brain systems that may underlie the rewarding effects of drugs, the development of tolerance and the experience of withdrawal symptoms, and the high rate of relapse to drug use after abstinence (Adler, 1995). Neuroscience research is beginning to provide detailed understanding of brain processes involved in many addictive phenomena and accordingly promises to provide more effective pharmacological therapies for addiction that target these processes (Adler, 1995; Maldonado, 2003).

The promise of neuroscience and genetic research for understanding addiction also raises major ethical and social issues (Independent Working Group on Brain and Mind Disorders, 2003; Safire, 2002). These can be considered under two broad headings: (1) ethical issues that arise in carrying out neuroscience and genetic research on addiction; and (2) the broader social and ethical implications of the potential uses of neuroscience and genetic knowledge (e.g., for therapeutic, preventive, and enhancement purposes) and their impacts on public understanding of and policies towards addiction (Roskies, 2002). In this paper, we outline the major ethical and social issues that will require more systematic and detailed analysis by neuroscience researchers, ethicists, policy makers, and the broader community.
2. Ethical issues in human neuroscience research on the addictions

Human neuroscience experiments often involve laboratory studies of the effects of chronic drug exposure on current brain function or the acute effects of exposure to drugs, drug analogues, or drug-related cues (e.g., injecting equipment) on behavior and brain function (Adler, 1995). Such studies increasingly use brain imaging technologies (such as PET, SPECT, and fMRI) (Fu & McGuire, 1999; Gilman, 1998) to study the acute effects of drugs and the neurobiological consequences of chronic drug use (e.g., Kling et al., 2000; Martin-Söelch et al., 2001; Sell et al., 1999).

Since the Nuremberg trials of German medical researchers after World War II, an international consensus has developed that biomedical research on humans (Brody, 1998; Jonsen, 1998) requires independent ethical review of the risks and benefits of proposed research, free and informed consent from research participants, and protection of privacy and confidentiality of information (Brody, 1998). Research involving persons who are cognitively or physically impaired requires special ethical consideration (Brody, 1998) because such “vulnerable persons” may not be capable of providing informed consent. That is, they may not be able to (1) understand the rationale behind a clinical trial, (2) understand exactly what is required of them and why, and (3) give their free and informed consent to participate in the study (National Bioethics Advisory Commission, 1999).

Concern about research on vulnerable persons has been most pronounced in experimental and clinical studies of persons with schizophrenia (Roberts & Roberts, 1999; Shamoo, 1998). Critics of such studies (e.g., Hall, 1999) have advocated stringent standards for obtaining informed consent for cognitively impaired persons, including independent review by Institutional Review Boards whose members include patients or patient advocates (Hall, 1999). Some researchers have criticized these types of protection for being cumbersome, overly paternalistic, and denying the mentally ill the right to make decisions on their own behalf (Carpenter, Schooler, & Kane, 1997). They argue that these restrictions will prevent important research being done into the causes and treatment of these disorders (Roberts & Roberts, 1999).

2.1. Are drug-dependent people vulnerable persons?

Until recently, the view among addiction researchers has been that drug-dependent people are able to give free and informed consent to participate in research studies and clinical trials so long as they are not intoxicated or suffering acute withdrawal symptoms at the time that they give consent (e.g., Adler, 1995; Gorelick, Pickens, & Benkovsky, 1999). It has been recommended that the severity of withdrawal symptoms should be assessed when screening potential research participants and before obtaining informed consent to participate in a study (Adler, 1995; Gorelick et al., 1999). This view has recently been contested by Charland (2002) and Cohen (2002). These authors have taken the defining characteristics of drug dependence in DSM-IV—especially the loss of control over drug use—to mean that people who are drug dependent lack the capacity to give free and informed consent to participate in research studies in which they may be given their drug of dependence.
Cohen (2002) argues that “the nature and pathology of untreated substance dependence make the condition inherently incompatible with a rational, internally uncoerced and informed consent on the part of those volunteering to receive addictive drugs in a nontherapeutic research setting” (p 74). This “may no longer obtain,” he argues, after addicts have undergone treatment. According to Cohen, it is only ethical to give drugs of dependence in experimental studies to abstinent addicts and possibly those who are in treatment. Even though research suggests that alcohol- and drug-dependent people who participate in research are helped rather than harmed (e.g., Dolinsky & Babor, 1997; Gorelick et al., 1999), we anticipate that ethics committees will not approve studies that administer drugs of dependence to abstinent addicts. If ethics review committees accept Cohen’s argument, the outcome could be that no experimental research will be undertaken in which drug-dependent people receive their drug of dependence.

Charland (2002) has argued that heroin addicts are unable to give free and informed consent to participate in heroin prescription trials. Heroin addicts, he argues on the testimony of one former heroin addict, are incapable of saying “no” to the offer of free heroin because of their addiction. Untreated heroin addicts offered their drug of dependence are “vulnerable subjects” who cannot serve as experimental subjects in such studies, or they can only do so if consent is given on their behalf by others. If accepted, these views would prevent addicts from participating in clinical research from which they stand to benefit.

2.2. Ethical issues in genetic research on addictive disorders

Ethical and social policy issues raised by genetic research to identify heritable traits require special attention for the following reasons: (1) the predictive nature of genetic information has the potential to adversely affect people’s lives; (2) genetic information carries implications not just for individuals but also families; and (3) genetic information has the potential to be used to stigmatize and victimize (Australian Law Reform Commission, National Health and Medical Research Council, and Australian Health Ethics Committee, 2003). These issues are especially pertinent when researching stigmatized health problems such as addictive disorders.

Genetic linkage studies of psychiatric disorders present a number of ethical challenges. Challenges faced during the ascertainment process include protecting privacy and ensuring that informed consent is given to the collection of information (Alexander, Lerer, & Baron, 1992). One of the main difficulties faced by researchers is engaging potential study participants without compromising their privacy or that of other participating family members (Alexander et al., 1992). The predictive nature of genetic information makes this a very salient concern in the study of addiction and other psychiatric disorders where misinformation about the causes of the disorders and their appropriate treatment may lead to unjustified attributions of blame and guilt, which adversely affect relationships between family members (National Health and Medical Research Council, 2000; Parker, 2002).

Genetic studies of depressive disorders need to ensure that subjects voluntarily participate in research that does not directly benefit them. Persons suffering from bipolar disorders can be competent to make such a decision but when they are suffering from these disorders they
may feel vulnerable and pressured, or they may be apathetic about being involved. It has been suggested that people who suffer from episodic disorders should only be asked to participate in studies when they are stable (Nuffield Council on Bioethics, 1998; Parker, 2002).

Individual genetic information collected in research studies should probably not be fed back to research participants (Nuffield Council on Bioethics, 1998), although they should be given feedback on the overall study results. This is for several reasons: (1) the significance of the probabilistic information is often uncertain and this is difficult to explain in the absence of expert genetic counseling; and (2) participants who are informed of the genetic test results may be required to disclose them to third parties, such as insurers. Making it a rule that genetic information not be disclosed to research participants obviates the need for counseling about genetic information of uncertain significance and protects participants from being required to disclose genetic test results to third parties (Nuffield Council on Bioethics, 1998).

3. Social and ethical implications of neuroscience research on addiction

3.1. Governing ideas of addiction

There has been a long running debate between moral and medical models of addiction (Gerstein & Harwood, 1990; Leshner, 1997). A moral model of addiction sees it as largely a voluntary behavior in which people choose to engage, and “addiction” as an excuse for bad behavior that allows drug users to take drugs without assuming responsibility for their conduct (Szasz, 1997). A medical model of addiction, by contrast, recognizes that while many people use drugs without losing control over their use, a minority develops an addiction that requires treatment if the sufferer is to become and remain abstinent (e.g., Leshner, 1997). The neurosciences improve upon older medical models of addiction by promising to provide a causal explanation of addiction in terms of detailed changes in brain processes (Mauron, 2003). One influential thesis is that addiction is a “brain disease” that results from the flick of a metaphorical switch in the brain that occurs as a result of chronic drug abuse (Leshner, 1997). This perspective undermines the simple view that addiction is wholly a matter of individual choice and hence that drug users are best dealt with by punishment and imprisonment.

A neurochemical model of addiction has a number of as yet unrealized advantages over moral models of addiction. It makes possible a more humane, less punitive response to addiction by raising the prospect of increased funding for addiction treatment, reducing the need for imprisonment as the first line treatment for addiction, and reducing the stigmatization of those who are addicted to drugs. It is for these reasons a view that appeals to some people who are addicted to drugs and to some of their families. A “disease” that can be “seen” in the many-hued splendor of a PET scan carries more conviction than one justified by the possibly exculatory self-reports of individuals who claim to be unable to control their drug use. Medical models of addiction may also, however, be interpreted as favoring simple-minded social policies. For example, the idea that addiction is a categorical disease entity lends itself to a seductive simplification in the case of alcohol, namely, that if we identify the minority of
people who are genetically vulnerable to alcohol dependence, then the rest of the population can use alcohol with impunity (Hall & Sannibale, 1996). This view ignores the adverse public health effects of alcohol intoxication, the dimensional nature of symptoms of dependence, and the evidence that multiple genes are involved in susceptibility to alcohol and drug dependence. It can possibly lead to addicts to abdicate responsibility for their behavior (Nelkin & Lindee, 1996).

The challenge for addiction neuroscience researchers is to give biology its due without depicting addicts as automatons driven by the state of receptors in their midbrains (Valenstein, 1998). Addiction needs to be seen, in part, as the result of choices that are not always wisely made by young people who operate with a short time perspective, a sense of personal invulnerability and skepticism towards elders’ advice about the risks of drug use. Loss of control over drug use is also a matter of degree, with most dependent drug users retaining some capacity to reduce their drug use or to become abstinent. Pharmacological drug treatments need to be seen as prostheses for an impaired will, a kind of Ulyssian self-binding against temptation, rather than the sine qua non of addiction treatment. Pharmacological treatment needs to be seen as the beginning of the process of recovery and reintegration of the drug-dependent person into the community. We still need to attend to a broader range of social policies in seeking to prevent drug use by youth (Spooner & Hall, 2002).

3.2. Coerced treatment of addiction

The most likely benefit of neuroscience and genetic research on addiction is improved treatment of drug-dependent persons. This might happen in a number of ways. First, a better understanding of the genetic and neuroscience bases of drug dependence may lead to more effective drugs to assist in cessation of drug use (Leshner, 1997; National Academy of Science, 1996; Walton, Johnstone, Munafo, Neville, & Griffiths, 2001). These may include drugs that act on key neural reward pathways or affect drug metabolism. Such drugs may have fewer adverse side effects than existing ones. Second, genotyping may better match patients to existing pharmacological treatments for addiction, such as bupropion and nicotine replacement in the case of smoking (Munafo, Johnstone, Murphy, & Walton, 2001; Walton et al., 2001).

There is a less welcome possibility that may arise from the development of more effective pharmacological and immunological treatments for addiction. If addicts are seen to be suffering from a brain disease that robs them of their autonomy and impairs their capacity to consent to treatment, then, some will argue, we should treat them under legal coercion. This could in principle be “for their own good,” although in practice coerced treatment has most often been advocated for drug-dependent people who commit criminal offences.

3.2.1. The case for coerced treatment

Legally coerced drug treatment for persons charged with or convicted of an offence to which their drug dependence has contributed is usually provided as an alternative to incarceration under the threat of imprisonment if the person fails to comply with treatment.
One of its major justifications is that treating offenders’ drug dependence will reduce the likelihood of their reoffending (Gerstein & Harwood, 1990; Inciardi & McBride, 1991). The advent of HIV/AIDS has provided an additional argument for treating rather than imprisoning drug-dependent offenders (Dolan, Wodak, Hall, Gaughwin, & Rae, 1996). The correctional and public health arguments for drug treatment under coercion are reinforced by the economic argument that it is less costly to treat offenders who are drug dependent in the community than it is to imprison them (Gerstein & Harwood, 1990).

3.2.2. Ethical issues in coerced treatment

Some authors reject any form of treatment under coercion for any form of drug dependence. Szasz (1997), for example, denies that drug dependence exists, arguing that all drug use is voluntary. According to him, the law should not prohibit adults from using any drug, and any drug user who commits a criminal offence should be punished, with no excuses by reason of drug dependence. The punitive consequences of Szasz’s heroic libertarianism have more public support than the case for legalizing the use of illegal drugs. Others, such as Newman (1974), accept that dependence exists but oppose compulsory drug treatment on the grounds that it does not work. If treatment under coercion were ineffective (as Newman claims), then there would be no ethical justification for providing it. Of course, even if treatment under coercion is effective, it does not follow that it should be provided. The community may, for example, place a higher value on punishing offenders (Hall, 1997).

A consensus view on drug treatment under coercion prepared for the World Health Organization (Porter, Arif, & Curran, 1986) concluded that such treatment was legally and ethically justified only if (1) the rights of the individuals were protected by “due process” (in accordance with human rights principles), and (2) if effective and humane treatment was provided. In the absence of due process, coerced treatment could become de facto imprisonment without judicial oversight. In the absence of humane and effective treatment, coerced drug treatment could become a cost-cutting exercise to reduce prison overcrowding.

The uncertain benefits of coerced treatment have led some proponents to argue that offenders should be allowed two “constrained choices” (Fox, 1992). The first constrained choice would be whether they participate in drug treatment or not. If they declined to be treated, they would be dealt with by the criminal justice system in the same way as anyone charged with their offence. The second constrained choice would be given to those who agreed to participate in drug treatment: this would be a choice of the type of treatment that they received. There is some empirical support for these recommendations in that there is better evidence for the effectiveness of coerced treatment that requires some “voluntary interest” by the offender (Gerstein & Harwood, 1990).

If pharmacological treatments are used under legal coercion, their safety, effectiveness, and cost-effectiveness should be rigorously evaluated (National Research Council, 2001). We also need to ensure that due process is observed and that effective and humane treatment is provided to drug-dependent offenders.
3.3. Preventing addiction

Drug control policies aim to prevent addiction by reducing the availability of drugs. This can be accomplished either by banning their use (in the case of cannabis, heroin, and cocaine) or by imposing high taxes (e.g., on alcohol and tobacco) and restricting minors’ access (World Health Organization, 1998). These policies affect the whole community, not just those who are drug dependent or at risk of becoming drug dependent. One might argue that on the grounds of efficiency and equity that drug control measures should be focused on those at highest risk of becoming drug dependent by virtue of their genetic risk. In other medical settings, this has been called “predictive genetic testing” (Evans, Skrzynia, & Burke, 2001). There are a number of problems with this proposal.

First, predictive testing is most defensible when we screen for disorders in which a single gene confers a high risk of developing a serious disorder for which safe and effective interventions exist (Holtzman & Marteau, 2000). When multiple genes predispose to common diseases, there are genes by environment interactions, with the result that a person with these genes has an increased risk of developing the disease but the probability of their doing is often still quite small (Evans et al., 2001). In general, the more genes that are involved in disease susceptibility, the less useful information about their genotype is to most individuals (Hall et al., 2002).

Second, given the low prevalence of high-risk combinations of susceptibility genes, a very large number of individuals would need to be screened to identify those with these genes. This may be expensive and difficult to justify on public health grounds (Vineis, Schulte, & McMichael, 2001).

Third, screening is only justifiable if there is an effective intervention to prevent the disorder in those who possess susceptibility genes (Evans et al., 2001). No such interventions exist. The development of an effective drug vaccine would provide more incentive for screening but it would also raise other ethical issues (e.g., about the right of parents to vaccinate their children). It would also raise serious questions of public policy, e.g., would it be more practicable to screen and vaccinate or simply to have universal drug vaccination? Who would pay the costs of either type of program? How likely is it that such a program would be publicly funded in the face of opposition from tobacco manufacturers?

Fourth, there is a possibility that predictive genetic testing may also have perverse and unintended effects (Holtzman & Marteau, 2000; Marteau & Croyle, 1998). For example, what effects would testing adolescents for susceptibility to drug dependence have on their preparedness to try drugs? What effects would it have on health insurance and on the social stigmatization of those who are at risk?

3.4. Neuroscience and enhancement

Enhancement is the use of medical interventions, such as drugs and prostheses, to enhance human performance or capability (Parens, 1998, 2002) making people “better than well” (Kramer, 1993). Some critics argue that existing pharmacological treatments are already being used in this way. Fukuyama (2002), for example, argues that the selective serotonin
reuptake inhibitors (SSRIs), which are used to treat depression, are being used by people who are not depressed to modulate mood and change personality. He claims the same is true with the use of methylphenidate, which is used to treat attention-deficit hyperactivity disorder. According to Fukuyama and others (e.g., Elliott, 2003), this drug is being used to improve attention in children and adults who do not have a diagnosable disorder. Other critics predict that future treatments for cognitive impairment and memory loss in Alzheimer’s disease will be used to enhance memory and cognition in adults who do not have these disorders in analogous ways to the way in which Viagra is being used to improve sexual performance in males who do not suffer from erectile impotence (Hall, 2003).

Critics of enhancement express concerns that can be usefully classified into two broad categories: concerns about the harms that may be experienced by those who use the enhancement technologies; and concerns about the adverse social impacts of the widespread promotion and societal embrace of enhancement technologies (Farah, 2002).

There are good reasons to be concerned about the possible harms that people who use enhancements might experience. The common occurrence of adverse reactions to many therapeutic drugs provides abundant empirical evidence of harm from medical interventions. These harms are usually outweighed by the relief afforded from the symptoms of disease and disability. The trade-off between adverse effects and the less certain benefits of enhancement may be less clear, however.

One can argue that addiction is a harm that arises from a type of enhancement: the use of mood altering drugs to improve mood and well-being. It arguably represents a pharmacological misappropriation of brain mechanisms selected by evolution to reward behavior that is required for individual and species survival, such as eating, drinking, and copulating (Hall, 2002; Hill & Newlin, 2002). Some generalize the evolutionary argument, arguing that all technologically achieved gains in human performance necessarily involve trade-offs between human abilities selected by our ancestral environment. If, for example, we optimize some abilities, this is likely to be at a cost in overall performance (Farah, 2002). Those who defend enhancement concede that there may be adverse side effects but argue that the use of enhancement technologies should be monitored for adverse effects so that potential users can be advised of their risks (Caplan, 2002; Stock, 2002).

Two concerns have been expressed about the societal implications of widespread use of enhancement technologies. First and foremost is the concern that marked social inequities in access to enhancement technologies will amplify existing social inequities (Farah, 2002; Fukuyama, 2002; Parens, 2002). Those who defend enhancement argue that this is more a criticism of existing social hierarchies than a compelling objection to enhancement (Caplan, 2002, 2003). This objection could be used, for example, to justify bans on private education. It could in any case be easily overcome by making all forms of enhancement freely available to all at low cost, e.g., by putting enhancing drugs in the water supply or publicly subsidizing the use of enhancement technologies, as many developed societies do with medical treatments and with what are arguably forms of medical enhancement, such as IVF and contraception (Parens, 2002). A second concern about the social impacts of enhancement technologies is that their widespread use will raise the standards for what counts as normalcy (Farah, 2002; Parens, 2002). This would, these critics suggest, force an arms race in the use of enhancement
technologies in which everyone would be coerced into using enhancement technologies as a way of “keeping up with the Joneses” who already use them. Such a trend would increase discrimination against the disabled and people with medical conditions who declined to be enhanced (Parens, 2002). The rejoinder of those who defend enhancement is that the logical implication of the critics’ argument is that those who do not want to be enhanced should be able to coercively prevent those who do from being enhanced (Caplan, 2002). Caplan (2002) argues that this policy is not followed elsewhere in society, e.g., in policies towards private education, academic coaching, or cosmetic surgery.

The history of psychopharmacology reveals inconsistent attitudes towards psychopharmacological enhancement (Healy, 2001). In the late 1960s, a “counterculture” arose in many developed societies that celebrated antipsychiatrists, such as R. D. Laing, who combined an enthusiasm for the mind-expanding effects of cannabis and LSD with hostility to the use of psychotropic drugs to treat schizophrenia.

3.5. Neuroscience, prediction, and privacy

Advances in neuroimaging technologies raise the possibility of “reading people’s minds” by using these methods to ascertain the truthfulness of what defendants or suspects tell the police (Farah, 2002; Foster, Wolpe, & Caplan, 2003; Ross, 2003). This is more of an aspiration than reality at present, although some entrepreneurs claim that electrophysiological methods can be used to tell if a person is telling the truth (Foster et al., 2003). Future improvements in neuroimaging may, even if imperfectly, disclose facts about a person that they may prefer to keep private (Ross, 2003). Farah (2002) has pointed out, for example, that a substantial minority of drug-dependent persons show a characteristic pattern of brain activation in response to drug cues that could enable them to be identified as drug dependent (Farah, 2002).

Neuroscience investigations may also provide information that proves to be predictive of disease risk in the same way as genes for Mendelian disorders like Huntington’s disease (Foster et al., 2003; Greely, 2002). Characteristic patterns of brain activity in childhood and adolescence, for example, may predict increased risks of addiction later in adult life. This possibility raises the same ethical issues (e.g., privacy and discrimination) that are raised by testing for alleles that predict an increased risk of serious neurological disease (Greely, 2002). These potential developments raise important ethical issues, particularly whether persons should be compelled to undergo such tests by the courts, insurance companies, or employers. Similar issues will arise with the potential use by insurers and employers of neuroimaging tests undertaken in the course of medical treatment. The claims of entrepreneurs promoting these technologies to the public (e.g., as tests of marital fidelity) raise the need for consumer protection against the overinterpretation of equivocal test results and bogus claims (Caplan, 2002; Farah, 2002).

3.6. Neuroscience and the media

Public interest in scientific findings and the political imperative for scientists to justify public funding have increased pressure on scientists to report their research findings in the
leading scientists have historically accepted a social responsibility to educate the public. In the 19th century, Michael Faraday and Thomas Huxley gave public lectures on science and wrote about science for the popular press (Blakemore, 2002). In our own day, Carl Sagan, Stephen Hawking, Richard Dawkins, Colin Blakemore, Steven J. Gould, and James Watson have written for the general public. They have accepted that they have a moral obligation to ensure that the public is well informed about science (Blakemore, 2002).

Given the public interest in neuroscience research, potential misunderstandings may rebound to the detriment of neuroscience and genetics. Neuroscientists and geneticists arguably have a moral responsibility to be proactive in their dealings with the media (Blakemore, 2002). They need to ensure that accurate information is released to the media and that their publications include prominent disclaimers that anticipate and correct predictable misinterpretations of their findings. Geneticists need to be aware that journalists often operate with a neo-Mendelian folk genetics in which an “addiction” gene means that if one has the gene one is highly likely to become an addict while its absence is strongly protective. Funding bodies and universities need to encourage scientists to undertake media training and recognize good media work as a legitimate activity for scientists to engage in (Blakemore, 2002).

4. Summary

The potential benefits of an improved understanding of the neuroscience bases of addiction are improved treatment and possibly the prevention of drug addiction. The type of research required to realize these promises will pose ethical issues about the capacity of addicted persons to give free and informed consent to participate in studies that involve the administration of drugs of dependence.

Neuroscience research on addiction may transform the long-running debate between moral and medical models of addiction by providing a detailed causal explanation of addiction in terms of brain processes. The claim that addiction is a “brain disease” that results from chronic drug abuse undermines the moral view that addiction is wholly a matter of individual choice and hence that drug users are best dealt with by punishment and imprisonment. Medical models of addiction may also be misinterpreted as supporting simple-minded social policies. It may, for example, lead to the seductive simplification that if we identify the minority that is genetically and biologically vulnerable to alcohol dependence, then the rest of the population can use alcohol with impunity. It can also lead to a preoccupation with individual explanations of behavior to the neglect of social policies for reducing addiction. Neuroscience research may raise other issues. The use of pharmacotherapies and drug vaccines to treat addiction under legal coercion is likely to be contentious, as will the potential use of drugs and prostheses to enhance human performance. Advances in neuroimaging may
enable us to “read brains” and predict future disease risk. These possibilities raise concerns about invasion of privacy, third-party use of neuroimaging data, the powers of courts to coerce defendants to undergo such tests, and consumer protection. Neuroscientists arguably have a moral obligation to ensure that the media gets the science right. They certainly have a professional interest in minimizing misunderstandings of their work in the media that may rebound to the detriment of neuroscience and genetics.

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