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PEER REVIEWED SUBMISSION

Neglecting the Social System: Clinical Neuroimaging and the Biological Reductionism of Addiction

A Special Theme Issue Article

Daniel Z. Buchman B.A., M.S.W. (candidate),
Student in the Faculty of Social Work, University of Toronto
Concurrent Disorders Service at the Centre for Addiction and Mental Health
Toronto, Canada

ABSTRACT

A main strength of neuroimaging and neuroscience is its reductionist focus on the brain. A limitation is that it runs the possibility of ignoring larger social factors. The brain image may not necessarily indicate the brain's neuroplastic 'rewiring' over time from genomic, epigenetic, environmental and social conditions. These factors are all necessary to understand the diverse nature of our brains, especially complex concerns such as addiction. For addiction to emerge it requires an intersection of genetic, environmental and social influences. It is foreseeable to ignore this multi-factorial interaction in the clinical setting when interpreting predictive brain imaging scans. This paper argues that relying too heavily on clinical neuroimaging in the treatment of patients who present a vulnerability to addiction can lead to cases of biological reductionism ignoring the influence social systems have on brain responses.

Introduction:

The rapid growth of molecular genetics appears to be reducing the brain to its most basic biological level. These developments are supported by the increasing availability of powerful biotechnologies such as neuroimaging. For the first time neural structures associated with various systems and mechanisms involved in diseases such as addiction can be 'seen'. As a result, the 'normal' brain is going through a conceptual transformation. Prior to these technological developments, nearly everything known about the operations, events and functions of the 'normal' brain required inferential observation and the acquisition of tacit knowledge (Young, 2006).

The brain is the most complex and perplexing organ in the human body. Empirical research on brain mechanisms examines neurologically 'abnormal' brains – that is, those diagnosed with neurological or psychiatric injuries and diseases. The problem of interpreting, understanding, locating and conceptualizing the brain and its emergent mental properties is not a new phenomenon, yet it has continued to riddle scientists and philosophers for centuries. Psychologists, social workers, psychiatrists, nurses and other mental health support workers have similarly been attempting to achieve this task by applying therapeutic, psychotherapeutic and pharmacotherapeutic techniques and technologies in helping to alleviate suffering and improve the well-being of their clients.

Clinicians have been working with neurologically diverse clients for decades. This notion of neurodiversity is intriguing as it implies a standard or normalcy, or rather something that is divergent from the typical. Thus, the neurologically 'diverse' would be the identified 'other' – and in this case, the 'other' would be that of a psychiatric diagnosis. Historically this 'othering' has resulted in unfortunate stigma and discrimination against those with a mental illness or addiction. Yet the meaning of neurodiversity is incorrect as there are no two brains that are identical. While certain structures and corresponding mechanisms are common amongst most brains, the way each brain responds to various genetic and social pressures is unique due to the intrinsic brain mechanism known as neuroplasticity.

Neuroimaging research has encountered similar concerns. Studies in genomic neuroimaging tend to rely on averaged data of participants to determine the area of haemodynamic movement, and have had difficulty accounting for inter-subject variability (Canli, 2006). It is therefore more correct, then, to speak of diversity within our brains or the diverse nature of our brains. For that reason, the neuroimage of the typical brain is not so typical at all.

It is without question that fMRI, CT, SPECT, PET and other novel imaging approaches are and will be integrated into the clinical setting (Klitzman, 2006). Presently, brain scans can already help confirm a diagnosis where the behavioural etiology and standardized or suspected diagnostic criterion has been fulfilled (Glannon, 2006). Nonetheless, these advancements raise several concerns, specifically related to potential social and ethical implications of endophenotype interpretation and predictive neuroimaging.

 (ISSN: 1916-2405)

While the strength of neuroimaging and neuroscience is its reductionist focus on the brain, its limitation is that it runs the possibility of ignoring larger social factors. The brain image may not necessarily indicate the brain's neuroplastic 'rewiring' over time from genomic, epigenetic, environmental and social conditions. These factors are all necessary to understand the diverse nature of human brains, especially complex concerns such as addiction which require an intersection of biological and social systems. It is foreseeable to ignore this key interaction in the clinical setting when interpreting predictive brain imaging scans.

This paper argues that relying too heavily on clinical neuroimaging in the treatment of patients who present a vulnerability to addiction can lead to cases of biological reductionism ignoring the influence social systems has on brain responses. What may present as a 'neurotypical' brain may only become 'atypical' given certain environmental and social conditions. Brain plasticity does not occur within a biological vacuum.

To examine these implications I will begin by providing a brief background to endophenotypes. Second, I discuss social and ethical issues concerning the clinical interpretation of predictive neuroimaging (endophenotypes) in addiction. Finally, I examine addiction neuroscience's position on addiction as a compulsive behaviour in relation to biological and social systems.

Endophenotypes

Irving Gottesman and James Shields (1972, 1973) introduced endophenotypes in the early 1970s with regard to schizophrenia. The original intention of endophenotypes was to quantify biological, cognitive, or behavioral markers identified prior to the onset of schizophrenia (Young, 2006). Presently, endophenotypes apply to the entire spectrum of brain disorders and are commonly associated with evidence derived from neuroanatomical, neuropsychological, neurophysiological, endrocrinological, cognitive and biochemical research located along genotype-clinical phenotype pathways (Gottesman & Gould, 2003). Such research aims to detect mechanisms as opposed to markers. While a wide range of technologies exist in empirical research, presently neuroimaging provides the most precise depiction of brain structures and processes (Young, 2006).

Despite the incredible potential of endophenotypes, they are not the "only key needed to unlock the underlying biological mechanisms" (Glannon, 2003, p.280). More importantly, endophenotypes are restricted within the boundaries of the cultural, social and anthropological systems and norms from which they were interpreted (Illes & Racine, 2005). Though functional neuroimaging can associate a clinical phenotype to brain structures and regions (the endophenotype), the technology gives no inherent significance to the question of why the 'highlighted' parts are related (Young, 2006). In view of that, the endophenotype is correlative, rather than causative.

Addiction, Predictors, and the Clinical Setting

Addiction, like other phenomena, emerges from the interacting levels of biological, chemical, physical, technological and social systems (Bunge, 1979, 2003, 2006). Substance use, misuse and dependence occur along a continuum, with addiction resting at its distal end.

In what follows, the term client is used to refer to any individual using health care services, and clinician refers to the broad spectrum of service providers, ranging anywhere from addiction counselors to psychiatrists to outreach workers.

Human beings have a varied response to the behavioral and physiological effects of drugs, because the effects of the substances depend on intricate psychological, environmental and pharmacological interactions (Crombag & Robinson, 2004). Various markers, identifiers and candidate genes have been identified which indicate an individual's risk of developing a substance use disorder. For example, an increased EEG beta power in males is a likely predictor of purported vulnerability to alcoholism (Rangaswamy, Porjesz, Chorlian, Wang, Jones, Kuperman, et al, 2006) and a reduced P300 amplitude suggests both a vulnerability to alcoholism (Carlson, Iacono, & McGue, 2002), and an increased risk for developing a substance use disorder (Carlson, McLarnon, Iacono, 2007). A more recent biological focus has been on other vulnerability markers such as the D2 dopamine receptor allele (Young, Lawford, Nutting, & Noble, 2004).

Individuals of lower socioeconomic status (SES), marginalized, or disadvantaged, suffer disproportionately from addiction, and these factors are equally as important for research and consideration. For instance, lower SES has frequently been implicated in predicting substance use among youth (Frisher, Crome, Macleod, Bloor, & Hickman, 2007). Furthermore, a study by Noble, McCandliss, and Farah (2007) looked at the relationship between low SES and brain development in children. The authors found disparities in neurocognitive profiles in children of middle and low socioeconomic status, suggesting a new brain-based lens in viewing wider problems associated with poverty.

These discussions lend themselves to questions of, given environmental situations, how neurotypical any one brain really is. This has far-reaching implications on the way clinicians and individuals interpret addiction-related endophenotypes, and how to approach it clinically. For instance, both clinicians and clients may find it problematic to interpret an endophenotype that suggests a genetic predisposition or vulnerability for a condition such as alcoholism. One possibility is that clients may interpret such a brain scan result as a death sentence or an "unaltered fate" (Klitzman, 2006). This will be an important time for the clinician to engage with the client, as knowing one has a certain condition impacts individuals in various ways, as in adopting a "sick role" (Parsons, 1951). An alternate scenario would see an identified vulnerability offer beneficial insight regarding problematic symptoms and socially undesirable behaviours, such as persistent excessive drinking (Klitzman, 2006). Therefore, the interpretation can go two ways: either the client perceives herself responsible to herself and others for her drinking and acts accordingly, or the client

absolves herself of responsibility, as she is not culpable for something genetically pre-determined. What is further troubling is that the client may believe their brain to be abnormal, despite the fact that in some situations a mere change in environment may instigate a cessation of use – indicating an effect of neuroplasticity – as exemplified in the well-known example of Vietnam War Veterans and opioid use.

Addiction Neuroscience, Environmental Risk, and Neuroethics

Craving is widely considered as the cardinal feature of addiction. This has implications clinically, as interpreting brain images of craving in the absence of the social or environmental contexts ignores a huge component of the craving mechanism. Addiction neuroscience's position with regard to drug craving or a person with an addiction's 'compulsive' behaviour is that it is an aspect of a larger brain disease. The debate persists in the literature regarding this issue, focusing specifically on drug craving and consumption with respect to voluntary control (Hyman, 2007).

Drug craving has been studied extensively in neuroimaging research implicating both biological (Everitt, 1997; Kilts et al, 2001; Lubman, Yücel, Pantelis, 2004; Volkow et al, 2005; Wexler, Gottschalk, & Fulbright, 2001) and environmental factors (Lee, Lim, Wiederhold, & Graham, 2005; Pickens et al, 1991). Yet interpreting a drug craving brain scan as foundationally biological is troubling. First, cravings are largely cue-elicited and triggered by environmental stimuli (Childress et al, 1999; Grant, London, & Newlin, 1996; Loewenstein, 2000). Second, continued exposure to environmental triggers instigates a perpetual cycle of cravings. Therefore, not only is it more likely that cravings lead to increased drug consumption, but also that they precipitate an engagement in a series of behaviours that facilitate this process (Levy, 2007). Thus, a complete removal or an infrequent encounter of environmental-related triggers will do a great deal to decrease or heavily control use patterns.

Reducing addiction exclusively to biological levels could also encourage ethically problematic uses of powerful new biotechnologies as preventative measures. An example of such a technology is the cocaine vaccine (Martell, Mitchell, Poling, Gonsai, & Kosten, 2005). Coercive vaccination programs against the euphoric properties of substances may be necessary for individuals whom neuroimaging defines to be genetically and/or socially at risk, such as aboriginal or First Nation's people. But, these approaches – promoted by the seductive appeal of biological reductionism – will likely further marginalize these groups, restrict access to health care and other resources, help gain public justification for the unethical use of biotechnologies and act as a replacement to more sensible social, economic and drug policies from which all of society would stand to benefit (Carter & Hall, 2007).

If clinicians choose to implement addiction treatments – whether pharmacologic or psychotherapeutic – on a predictive brain scan without giving any acknowledgement to the intersecting biological and social systems, not only could iatrogenisis or biolooping (Hacking, 1999) occur, but unnecessary harm could be inflicted on the client. The harm in this situation extends to the false creation of a substance use disorder that might never have emerged, and the labeling of a brain as abnormal. The ethical implications here are profound. Even if the clinician recognizes the system intersections and their relation to addiction, the possible implications of putting into practice these approaches, or even standard addiction techniques such as motivational interviewing (Miller & Rollnick, 2002), should still be evaluated against the potential risks of withholding information from client's who are at an increased risk of substance use disorder (Glannon, 2006).

Of course, the science isn't perfect as the predictive accuracy of a pre-symptomatic substance use disorder is satisfactory at best. While the correlation may be strong between endophenotypes and future substance abuse disorder, this does not necessarily indicate a causal relationship. Having reduced P300 waves, experiencing symptoms of depression, or being male and having a familial history of alcohol use, will not directly lead to alcoholism. The likelihood that the event will happen is increased, true, but again, this is not the entire story. Therefore, at the clinical level, the increasing value placed on biological levels in individuals at an increased risk of developing an addiction has significant implications neuroethically.

Conclusions

As the technologies of neuroimaging continue to improve, it is becoming easier to not only understand the general design of the brain, but also the localization of the processes that contribute to our mental activities. These developments give new insight into the development and selection of treatments, based on a shared understanding of biological and environmental factors, by tailoring them specifically to meet the needs of the individual client. Advancements in neuroimaging research in addictions have not only helped to enhance the knowledge base, but can also affect the lives of those who suffer from the perils of addiction.

Progression in addiction neuroscience may foster a naïve enthusiasm for an exclusively biological approach. The path of addiction and depiction of the addicted brain are not without social and environmental contributors. The interaction of social, environmental and biological systems account for the emergence of mental activity, such that it's phenotypic expression varies depending on the neurologically diverse makeup of that individual.

What may have originated as a normal brain, given an adverse environment and intrinsic neuroplasticity, may not be so normal after all. Therefore, what we are inclined to call normal has to be seen in a different light. Addiction is a process that has evolved over time – an interaction taking place between neurological maturation and genetic endowment within one's environment.

Nonetheless, the technology is still young. As predictive neuroimaging rapidly becomes more precise, its applications have yet to be established (Glannon, 2006). Clinically acknowledging social and environmental risk factors in addition to biological ones has the potential to reduce potential iatrogenisis and harm. Discussing all relevant factors with clients can help prevent distress and reduce stigmatization of a possible addiction by educating those who may feel neurologically abnormal as a result. How a client

digests this information, and the way in which a clinician presents it, will considerably affect the therapeutic process and the nature of the therapeutic relationship (Glannon, 2006).

Ultimately, if incorporated clinically, predictive neuroimaging has the potential to identify endophenotypes, highlighting those brains which present an increased vulnerability to addiction. These findings could initiate pharmacological and psychosocial interventions to aid the client and their families in preventing or helping to control present drug misuse. Yet clinical interpretation of these scans in the absence of environmental factors could lead to oppressive and coercive practices, as well as exercises in discrimination and harm, especially among marginalized populations. Further philosophical and scientific inquiry in the area of the ethics of vulnerability should be explored.

Competing Interests: *None*

Acknowledgements: *None*

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Key Words: *neuroethics, addiction, substance use, endophenotypes, environmental influence*

Address for Correspondence:

c/o The Faculty of Social Work, University of Toronto, 246 Bloor Street West, Toronto, ON, Canada M5S 1A1

e-mail: *Daniel.buchman@utoronto.ca*