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Stigma and Addiction: Being and Becoming

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In his account of clinical neuroscience and subjectivity, Gillett (2009) provides three examples of neurological conditions that dramatically affect the lived experience of the individual: locked in syndrome, persistent vegetative state, and minimally conscious state. Yet for individuals with mental illnesses such as severe addiction, embodied subjectivity may have different implications for human identity. Because attitudes towards individuals with addiction are heavily moralized, being somebody somewhere with an addiction may result in both self- and other-labeling, and consequently, a development of a "spoiled" public identity.

Our response will briefly focus on the potential implications of neuroscience and human subjectivity in addiction. Using the so-called "brain disease" model of addiction as an illustrative example, we argue that the development of a stigmatizing identity of an 'addict' is gradual, and emerges through a complex process which includes, but is not limited to, the individual's narrative history, social lived experience, neuro-genetics, and a self-labeling process. Since identity emerges from, in part, and is preserved through relationships, a stigmatizing illness identity considerably affects the being-in-the-world-with-others for "the addict".

Stigma and Being-in-the-World-with-Others

Stigma is a universal phenomenon, observed in essentially all cultures (Goffman 1968), and even across species (Dugatkin, FitzGerald, and Lavoie 1994). According to Link and Phelan (2001), stigma occurs when "elements of labeling, stereotyping, separation, status loss, and discrimination occur together in a power situation that allows them" (337). Further, when a particular character trait is recognized as being socially significant, prevailing "folk psychology" categorizations associate these individuals with characteristics that may be considered socially undesirable. Therefore, stigma is a relational notion.

How we as humans think about other humans as moral agents depends, fundamentally, on what we understand a human being to be. How, whether, and by what means human beings can know about the subjective lives of other beings is ethically significant. This ethical question relies on the belief – or rather metaphysical assumption – that other human beings have minds (brains) at the very least comparatively similar to our own. However, problems arise in how human beings attempt to establish the nature of those other human brains. This has to do, in part, with how human beings understand the composition of the brains of other human beings – or rather, the neurobiology. Accordingly, neuroscience knowledge may be instrumental for providing insight into the *form* of other human brains (Farah 2008).

Due to advances in the general understanding of the human brain, causal neurobiological explanations of our mental lives are becoming more prevalent. Previously intangible mental qualities of the human brain have now begun to assume concrete features. Dumit (2003) calls the "set of acts that concerns our brains and our bodies deriving from received-facts of science and medicine the *objective-self*." (39). The objective-self is how we understand our brains and our bodies, as being composed of biological material. Given the general folk psychology conceptions regarding human bodies and brains in relation to objective, referential objects of

science and medicine, the objective-self is necessarily inaccurate. Nonetheless, objective-selves challenge generally accepted notions of normality and contribute to the understanding of what kind of brain we have in comparison to others (Dumit 2003).

In recent years, folk psychology perspectives on addiction – that addicted individuals freely and voluntarily use drugs, and the continued use despite negative consequences derives from a weak "will" or character – have faced opposition from robust empirical research that associates impaired brain regions, states, and processes with addictive behaviours (Volkow and Li 2004). In this view – which we hasten to add is incompletely understood in many important details – addiction is a complex process through which substances, the external environment, and our social relationships conspire to modify the molecular properties of cells in the brain in fundamental ways. These changes affect the operation of brain circuits; these, in turn, guide behaviour. Modern neuroscience has identified at least some of the cellular and molecular mechanisms involved in tolerance, neural resensitization, withdrawal and dependence, and these phenomena are largely convergent across various chemical modalities that are likely to lead to drug addiction, and 'behavioural addictions' such as gambling.

Both scientists and mental health advocates have long suggested that an increase in the lay public's understanding of mental illness' biological underpinnings may reduce discrimination and prejudice. However, as Gillett (2009) remarks in describing *The Adequacy Requirement*, "our ethical responses to any thing should pay due regard to its most significant features and not just focus on one aspect of it (e.g., a purely neurobiological description)" (XX). Consistent with this view, at least for now, is the fact that the rise of a genetic model of mental illness has not persuaded the public, nor increased tolerance (Schnittker 2008).

Attributing neurobiological factors to addiction has the potential to reduce stigma (both perceived and experienced), blame and responsibility, and provide more effective treatment options for society. However, attributing addiction to neurobiological causes may have unintended consequences on public orientations towards those living with an addiction. Adopting a view of addiction emerging from faulty brains may encourage an "us-them" distinction, as those deemed neurobiologically "abnormal" may be considered "diseased" (Phelan, Cruz-Rojas, and Reiff 2002). Indeed, Marie and Miles (2008) indicated that participants were less willing to engage in a social relationship with someone who was an alcohol abuser or substance dependent, as opposed to those diagnosed with either schizophrenia or depression. Viewing addiction as a chronic condition may also suggest something continuous and persistent, and unlikely curable (Phelan 2005). Other studies found that when participants were given biogenetic explanations, or endorsed brain disease explanations as causal, individuals with a mental illness were perceived as more dangerous, having a lack of self-control, unpredictable, and were deemed less responsible for their actions (Mehta and Farina 1997). These perceptions have far-reaching consequences for stigma and identity insofar as they relate to our assessments of the value of others.

Not only might such insights alter external views of addiction, but addicted individuals may thus perceive their brains – and by extension themselves – to be diseased if that is the perspective of the wider community. The individual who self-fashions herself an addict can recognize in her objective-self as having a different *kind* of brain, signifying a deviation from normal and thus even concluding that she is a different *kind* of human being. Therefore, a pure neurobiological explanation of our selves may raise doubts about our own sense of normalcy, affecting our self-labeling with respect to the meaning of our thoughts, our behaviours, and our relationships with others.

The mechanisms used to translate neuroscience research to the public, while on the one hand attempting to persuade and educate by neuroessentializing human subjectivity, may

inadvertently contribute to the beliefs that perpetuate stigmatizing attitudes. This is particularly the case with demoralizing attitudes towards individuals living with a serious addiction and culturally ingrained views on moral responsibility. However, many individuals living with severe mental illness desire a perspective "of the objective person that does not, in their view, prejudge them and condemn them to blame and guilt"(Dumit 2003, 45). Thus, a neurobiological model of addiction is necessary, but not sufficient in reducing the complex phenomenon of stigma. Future research can examine the intersection of a desire for a neurobiological model of addiction, and why stigmatizing attitudes towards individuals living with addictions persist.

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