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BRAIN MECHANISMS AND THE DISEASE MODEL OF ADDICTION

Is it the whole story of the addicted self? A philosophical-skeptical perspective

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Introduction

Contemporary scientific and philosophical debates on addiction center on two models. In the brain disease model, drug addiction is a "chronic and relapsing brain disease that results from the prolonged effects of drugs on the brain" (Leshner 1997: 45). In contrast, the self or person model individuates addiction in folk psychological terms, as a kind of behavior marked by the repeated use of a drug of choice (DoC) and the difficulty involved in quitting (Satel and Lilienfeld 2013; Flanagan 2013a; Tekin *et al.* 2017). On the one hand, the brain disease model promotes cellular-level understanding, painting the addict as a biological organism. It takes seriously recent advances in brain imaging methods and the increased sophistication in the neuroscientific modeling of the brain's reward system—the mesolimbic dopamine system—and promotes pharmaceuticals to target and rebalance the chemical anomalies in it. On the other hand, the self model sees the addict as a person or intentional system (Dennett 1971) who acts according to beliefs and desires and responds to complex historical, environmental, and interpersonal aspects of life. It points to the personal-socio-cultural markers of addiction and promotes social, psychological, and behavioral intervention strategies, e.g., encouraging the addict to change the interpersonal environment that triggers and perpetuates addictive behavior, to receive intensive psychotherapy, to cultivate meditation, and so on.[1]

Although each model accepts the validity of the other, to a certain point, there is a *prima facie* tension between them. The brain disease model acknowledges the environmental, social, and cultural factors in the development of addiction, but its proponents often favor neuroscientific work in *lieu* of other research paradigms, e.g., social sciences and cognitive sciences. The self model acknowledges the importance of the cellular-level understanding of addiction, but its adherents warn of the dangers of neuro-enthusiasm (Satel and Lilienfeld 2013), i.e., the over-promotion of scientific and clinical approaches to target the person. In this chapter, I propose using Ken Schaffner's framework for thinking about reductionism in science (Schaffner 2013) to resolve the issue. In this framework, the brain disease model moves in the direction of what

Schaffner calls "sweeping reductionism," with the self model moving towards "creeping reductionism." Because of the virtues of creeping reductionism, I argue the self model has more promise for addiction research.

I start the chapter with a brief overview of Schaffner's framework for reduction in science, describing his notions of "sweeping" and "creeping" reductionism. I apply this framework to the brain disease and the self models, noting the epistemic advantages of committing to the latter. In my analysis, I draw on scientific research and clinical work on addiction; to this I add first-person accounts of those influenced by addiction, a seldom used but rich resource (Tekin 2016).

Reductionism in science and psychiatry

A common debate among scientists and philosophers is whether human sciences, such as psychology and psychiatry, involve phenomena distinct from those targeted in the physical sciences. According to reductionism, target phenomena in human sciences are only *prima facie* distinct from those in the physical sciences, lending themselves to explanation or even replacement by phenomena in the physical and chemical sciences. In this respect, mental disorders can be reduced to phenomena in physics and chemistry.

For Schaffner, reductionism exists on a spectrum. On the one extreme, human phenomena "are nothing but aggregates of physicochemical entities," a view he labels "sweeping reductionism" (Schaffner 2013: 1003). For "sweeping" reductionists, "there is a theory of everything" and "there is nothing but those basic elements—for example, a very powerful biological theory that explains all of psychology and psychiatry" (Schaffner 2013: 1003). At the other extreme is "creeping reductionism" (Schaffner 2013). A "creeping" reductionist will argue that the different models of the target phenomena—in this case addiction—provide fragmentary explanations that must be combined for a fuller understanding. In Schaffner's view, a full commitment to sweeping reductionism is impossible, except perhaps as a metaphysical claim. Furthermore, sweeping reductionism has little value in the biological and psychological sciences.

The brain disease model leans towards a sweeping reductionist commitment, if not fully embodying it, and this is cause for concern. Creeping reductionists, for Schaffner, do not typically commit to a nothing-but approach in scientific explanations. Rather, they embrace a pragmatic and pluralistic parallelism, working at several levels of aggregation and discourse at once. Insofar as the self model I discuss here proposes to work on addiction at different levels (ranging from cellular and molecular, to individual and social) simultaneously, it embodies creeping reductionism and is, thus, a scientifically and clinically resourceful approach, enabling cross-fertilization across different levels.

The brain disease model of addiction

In the brain disease model, all drugs of abuse have common direct or indirect effects on a single pathway in the brain—the brain's reward system, i.e., the mesolimbic dopamine system. (Leshner 1997).[2] The mesolimbic dopamine pathway is individuated at the molecular, cellular, structural, and functional levels; it extends from the ventral tegmentum to the nucleus accumbens, with connections to such areas in the brain as the limbic system and the orbitofrontal cortex (Hyman 1996; Ortiz *et al.* 1995). It is associated with motivation, decision-making, and inhibitory control and is thought to be a key detector of rewarding stimuli, as it controls responses to natural rewards such as food, sex, and social interactions. According to the disease model, drugs interact with the mesolimbic pathway to produce addictive effects and "reset" the

brain's reward system by causing sharp increases in the release of dopamine (Volkow *et al.* 2016; Di Chiara 2002). Such increases elicit a reward signal that triggers associative learning, leading individuals to associate certain stimuli with the drug use, e.g., the environment of drug taking, persons with whom it has been taken, etc. These environmental cues may all trigger craving for and use of the drug. Such conditioned responses become deeply ingrained, often lasting long after use has stopped.

Though there is no scientific consensus on the matter, for some scientists the activation of the brain's reward system is significantly different from that associated with natural rewards, such as food, sex, and social relationships (Wise 2002).[3] Dopamine cells stop firing after repeated consumption of a "natural reward," satiating the drive to continue, but addictive drugs circumvent the natural satiation and continue to directly increase dopamine levels (Wise 2002). Accordingly, compulsive behaviors are more likely to emerge when people use drugs than when they pursue a natural reward (Volkow *et al.* 2016).

A well-known metaphor for the disease model is the broken switch (Leshner 1997). Drug use begins as a voluntary behavior, "but when that switch is thrown, the individual moves into the state of addiction, characterized by compulsive drug seeking and use" (Leshner 1997: 46). Put otherwise, addiction is a consequence of fundamental changes in brain function, and the goal of treatment must be either to reverse or to compensate for those brain changes, through pharmaceuticals or behavioral treatments (Leshner 1997). Elucidation of the biology underlying the metaphorical switch is seen as the key to the development of more effective treatments, particularly anti-addiction medications.

It would be unfair to characterize the brain disease model as fully embodying a sweeping reductionist framework according to which addiction is "nothing but" anomalies in the brain's dopamine system, as its proponents acknowledge the importance of social and environmental factors in its development. That said, however, the brain disease model leans towards a "sweeping reductionist" framework insofar as it promotes research into the neurobiology of addiction and does not offer an explicit framework for investigating the social and environmental factors contributing to addiction. A case in point is the National Institute of Mental Health (NIMH)'s Research and Domain Criteria (RDoC) initiative, i.e., its development of an alternative to the DSM-5 in guiding research for mental disorders. It does not fund research projects unless they explicitly analyze the neurobiology of mental disorder (Vaidyanathan 2016, personal communication). It encourages research programs that advance initiatives to find pharmaceuticals to target and rebalance the neurobiological breakdown in the brain. An exclusive focus on the neurobiology of addiction discourages alternative initiatives, including, for instance, research into the relationship between homelessness and addiction or intervention strategies involving housing programs for the homeless.

Critics have voiced a number of concerns with the brain disease model. One is that brain chemistry is plastic and responds to more than simply repeated drug use; a wide range of factors come into play, including changes in the physical and social environment. Arguably, therefore, anomalies in the brain's reward system that contribute to the development of addictive behavior can be corrected not only by anti-addiction medications but also by psychological and behavioral interventions (Satel and Lilienfeld 2013). Another, perhaps more important, limitation is the model's apparent lack of focus on the complexity and multi-aspectuality of the lives of individuals with addiction, despite the need to develop clinically effective strategies for recovery (Tekin *et al.* in press). This implies that the brain is the most important and useful level of analysis for understanding and treating addiction, even though drug use and abuse do not emerge in a vacuum independent of an individual's history, interpersonal relationships, socio-economic status, etc., and its successful treatment requires targeting and analyzing these dimensions. For

example, addictive behavior only affects people who have sufficient access to drugs to abuse, and this might be relevant to understand the demographics of the population with a drug abuse problem. Furthermore, the individual reasons people start drug use, e.g., to remedy shyness in social contexts, to cope with a dysfunctional relationship, to adopt the social expectations of a particular group, etc., are as important in understanding addiction as are the neurochemical underpinnings.

In short, the personal, social, and cultural context of addiction is as equally important as the neurobiology of addiction, and the brain disease model does not easily lend itself to the study of the former. Despite the general recognition of the importance of these factors, the conceptual and empirical frameworks for studying addiction continue to target the brain mechanisms, at the expense of a conceptual and empirical framework that benefits from person-level understanding, using resources, say, from cognitive psychology, sociology, anthropology, etc. A plethora of treatment strategies that go beyond drug-related interventions thus remain under-explored. Scientific psychiatry may be missing valuable opportunities to study the complex factors that contribute to the development of addiction and other mental disorders.

The self model of addiction

As noted above, the self model considers addiction a person-level phenomenon; it represents addiction in reference to an individual's personal history, socio-economic status, race, gender, and other identity-constituting factors, her interpersonal relationships, and her conception of herself, in addition to her biological make-up. It differs from the brain disease model in a fundamental way: the target of inquiry is the self or the person or the intentional system (Dennett 1971), not just the brain. In this respect, the self model is "holistic" and aims to explain addiction in reference to the full complexity of an individual. In contrast, the brain disease model embodies "smallism" (Wilson 1999), over-emphasizing the building blocks of human bodies, such as neurons, and underestimating more integrated and complex phenomena, such as addiction.

The self model takes the self, or the person, to be a dynamic, complex, relational configuration with a more or less integrated cluster of person-level properties (Tekin *et al.* in press; Neisser 1988; Jopling 2000; Thagard 2014; Bechtel 2008; Tekin 2014, 2015). Under what I previously called the multitudinous self view (Tekin 2014), the self is individuated and empirically tractable—using the resources provided by cognitive, developmental, social, and biological psychology—through five different but complementary dimensions: (1) the ecological aspect of the self, or the embodied self in the physical world, which perceives, acts, and interacts with the physical environment; (2) the interpersonal aspect of the self, or the self embedded in the social world, which constitutes and is constituted by inter-subjective relationships; (3) the temporally extended aspect of self, or the self in time, grounded in memories of the past and anticipation of the future; (4) the private aspect of the self, which is exposed to experiences available only to the first person and not to others; (5) the conceptual aspect of the self, which represents the self to that individual by drawing on her properties or characteristics and the social and cultural context to which she belongs.[4] The different aspects of the self connect the individual to herself and to the physical, social, and cultural environment in which she is situated. A condition such as addiction affects each dimension of the self, and the more-or-less integrated unity of all dimensions.

Because each aspect is experienced from the first-person point of view and can also be tracked from third-person points of view, the model can represent addiction phenomenologically, empirically, and conceptually. To clarify, let me start with the ecological dimension of the multitudinous self. It represents the individual's embodiment in the physical world, including

her biological features, genetic make-up, and the constraints of her body. In this sense, the ecological dimension is specified by the body, the physical conditions of a particular environment, and the active perceptual exploration of and response to these conditions. This particular aspect of the multitudinous self tracks addictive behavior in a number of ways. First, there is something going on in the body—in the central nervous system, brain cells, brain's reward system, hormones, genes, etc.—of an individual when she becomes addicted to a certain drug. As discussed in the previous section, recent work on the brain's mesolimbic dopamine system, on which the brain disease model is grounded, indicates correlations between addictive behavior and dopamine activity (Nestler 2013). We cannot dismiss such findings; nor should we wish to, and future research should shed more light on the connections. Second, the symptoms of withdrawal can be traced through the ecological dimension. The addict's hands shake. She gets anxious, restless, and irritable. She wakes up in the middle of the night with nausea or wanting the DoC. In a worst-case scenario, she has delirium and hallucinations. This continues until she takes the DoC.

The manifestation of addiction in the ecological dimension of the self is not only accessible through a first-person perspective (the addict herself) but also through the third-person perspective (e.g., scientists who study her behavior, brain, hormones, genes, interpersonal relationship, childhood, socio-economic class, etc., and clinical practitioners, including doctors, nurses, social workers, etc.). The addict experiences the craving; her partner observes her restlessness, anger and frustration in the absence of the DoC; her psychotherapist notices she is more tense and quieter than usual; scientists gather evidence about the level of the DoC in her blood; social workers try to help her get connected to communities where she can find fulfillment and flourish. Knowledge of the ecological aspect of the self can and does facilitate a number of effective (albeit limited) interventions. For example, through cognitive behavior therapy, an addict may learn self-monitoring to recognize cravings early, identify situations putting her at risk, develop strategies for coping with cravings—sometimes with the support of medications—and avoid high-risk situations (Carroll and Onken 2005).

Now consider the intersubjective aspect of the self, the part handling the "species-specific signals of emotional rapport and communication between the self and other people" (Neisser 1988: 387; Bechtel 2008). Through intersubjectivity, an individual begins the interpersonal relationships of care and concern through which her identity is formed, then enriched or impoverished, depending on the level and the kind of care she receives. This aspect of the self tracks addictive behavior patterns in multiple ways. First, forms of addictive behavior in the consumption of the DoC progress in a particular kind of social environment. For example, in a lifestyle Flanagan calls "the male life of public and gregarious heavy drinking," "social drinking" is widely encouraged and becomes the context through which individuals socialize in their professional lives to talk "business" (Flanagan 2013b: 870). Over time, drinking becomes the sole reason to take part in these events, not business. Second, the kind and the quality of an individual's interpersonal relationships are major factors in the development of addictive behavior. Individuals with addiction often have a complex history of family relationships. Being subjected to physical or sexual abuse as a child is strongly linked to addiction (Marcenko *et al.* 2000; Langeland *et al.* 2002). Sometimes people are in relationships they do not want to be in, and intoxication becomes an easy escape. It ultimately becomes a problem on its own, however, harming not only the self, but others (Graham 2013).

Tracking the intersubjective dimension of the self may provide explanations of why a person becomes and stays addicted. At the same time, it may facilitate the development of effective interventions. If a particular lifestyle is enabling addiction, as in the "male life of public and gregarious heavy drinking," interventions could include helping the individual change his social

environment. Other forms of interventions include helping him get rid of a relationship causing distress or develop more effective coping strategies.

The temporally extended aspect of the self also tracks addiction. This aspect individuates the person in time; she is shaped by her experiences and memories of the past and her anticipation of the future. The development of addiction over time may take many different trajectories, but there are some commonalities. A drug is sampled first, often with no intention of making it a regular activity. There is no fear of the harm that may ensue. It is found to be enjoyable, invites further consumption, and becomes increasingly frequent. At some point, the individual may notice the behavior is harmful and contradicts her vision of her future self. She may recognize the necessity of not using the DoC to realize her goals, and this may help her stop the addictive behavior.

Understanding the temporal dimension of the self may facilitate an understanding of how and why people get addicted, e.g., what kind of developmental environment contributes to the development of addictive behavior, and why they have difficulty keeping their promises to themselves and others, e.g., growing up in families where people have not kept promises. The study of this aspect of the self may facilitate the development of effective interventions. The ability to recognize temporal extendedness is already used as a resource in cognitive behavior therapy when patients are taught stimulus control strategies, for example. In this type of therapy, addicts learn to avoid situations associated with drug use and to spend more time in activities incompatible with drug use. They learn to practice "urge control" by recognizing and changing the thoughts, feelings, and plans that lead to drug use. Patients' past attempts and failures are used as a benchmark to customize therapy (Azrin *et al.* 1994).

The private aspect of the self traces the individual's conscious awareness of felt experiences—what William James takes to be uniquely ours (James 1890). The private aspect of the self is not phenomenologically available to anyone else (e.g., feelings of pain or disappointment) but, with the help of language, it can be communicated (Bechtel 2008: 260). Addiction is traceable in the private aspect of the self, including the felt experience of being addicted, cravings for the DoC, the distress of not using it when consumption is delayed, and regrets about consuming it despite resolves to the contrary. It is extremely difficult for others, e.g., caregivers, clinicians, etc., to appreciate the complexity of the various phenomena experienced by the addict. However, linguistic representations of these experiences, say, when the individual is describing what it is like verbally or in writing, provide substantive information, and these data can be used for explanations and interventions. Some say the memoirs of addiction are helpful, not only to the experts wanting to fathom addiction, but also to the addicts themselves (Flanagan 2013a, 2013b). They argue the private dimension of the self offers excellent resources to scientifically investigate addiction. As we better understand this aspect of the self, we will better understand what addiction is and what successful interventions look like.

Last but not least, we have the conceptual aspect of the self. Self-concepts selectively represent the self to the self. They are the products of the dynamic interaction of the other four aspects of the self with the external social and cultural environment. Self-concepts include ideas about our physical bodies (ecological aspect), interpersonal experiences (intersubjective aspect), the kinds of things we have done in the past and are likely to do in the future (temporally extended aspect), and the quality and meaning of our thoughts and feelings (private aspect) (Jopling 1997, 2000; Neisser 1988; Bechtel 2008). For instance, an individual's self-concept as a "responsible person" is the product of the intersubjective aspect of her selfhood and of the norms of responsibility in the culture of which she is a part. Self-regarding feelings and attitudes, such as self-confidence, security, self-esteem, self-respect, and social trust, emerge as she develops self-concepts and as the different dimensions of the self interact with her social and cultural world. Self-concepts are informed by the other four aspects of the multitudinous self and by the

individual's unique embodied experiences in the world (Neisser 1988; Jopling 1997; Bechtel 2008; Tekin 2011). In turn, self-concepts inform and shape the other aspects of the self.

Self-concepts are also informed by pathologies to which the individual is subjected. For example, an addict may develop "self-regarding reactive attitudes of bewilderment, disappointment, and shame" about her addiction (Flanagan 2013a: 6). In what Flanagan calls the twin normative failure model, a fundamental aspect of addiction is a conflict in the addict's self-concepts (Tekin *et al*. in press). She suffers from twin normative failures, in that she recognizes (1) she can't successfully moderate or quit the DoC on her own because she fails to "execute normal powers of effective rational agency," and her excess consumption of the drug of (DoC) leads to (2) a failure to "live up to the hopes, expectations, standards, and ideals she has for a good life for herself" (Flanagan 2013a: 1). In the first kind of failure, she conceptualizes herself, correctly, as unable to control her drug abuse. She wants to quit, resolves to take steps that will help her quit, yet fails to do so. In the second kind of failure, she conceptualizes herself as a person with multiple roles: e.g., professor, friend, daughter, activist. She understands herself as a person through these multiple roles, and she has standards for how to play these roles. She has a vision, for instance, of herself as a good scholar, or a dedicated human rights activist, or a responsible colleague, etc. However, due to her addiction, she is unable to live up to her standards, ideals or aspirations, or to the norms of the multiple roles she occupies. The successful negotiation of these roles is part of what it will mean for her to be the person she aims to be. In her self-narrative, the addict realizes the meaning, worth, and success of her life depend on her not using, but she continues to use. Given the two failures, addiction undermines both the rationality of the addict's life and its goodness. The addict is not the person she could or should be; nor is she the person she wants to be.

Self-concepts are also action-guiding (Tekin 2014, 2015). They inform how individuals behave and can motivate them to change. In the context of addiction, the formation or alteration of self-concepts will influence future actions. Hopelessness in the face of repeated relapses and self-concepts such as being weak-willed may diminish an addict's ability to quit the addictive behavior. Alternatively, she may express conflict and heightened distress because of a strong resolution to quit drinking, especially if she is unable to do so. From this conflict, she may be able to redefine her self and enumerate new behavioral goals. She may take a step towards change by altering a self-concept and stabilizing a new behavior pattern. Or perceiving herself as someone who needs help, she may reach out to the communities of other individuals with addiction. The success of Alcoholics Anonymous programs partially owes to this.

Because of their plastic nature, self-concepts offer great opportunities for successful clinical interventions. Clinicians may work towards helping the addict develop more positive and resourceful self-concepts, strengthening her self-esteem, as well as her self-control capacities, and help her flourish by stopping her use of the DoC. By working with her self-concepts, clinicians can motivate her to think, act, and behave in certain ways, expanding her possibilities for action (Tekin 2010, 2011, 2014, 2015; Jopling 1997).

The model of the self, as described above, fits easily into a creeping reductionist framework, with myriad advantages for psychiatry. Such a model could enhance, for instance, etiological psychiatric research using an interventionist account of causality (Woodward 2003), as recently applied by Kenneth Kendler (2014). In the interventionist account, factors that are "difference makers" are causal in the development of a phenomenon, for example, addiction. As one goal of psychiatric research is to unpack the risk factors, i.e., the difference makers (Kendler 2014), researchers into mental disorders may be well advised to take a closer look at the different aspects of the self. With the interventionist model of causality, we can identify a wide range of etiological factors spawning the ecological, interpersonal, temporal, private and conceptual dimensions of a phenomenon. If, for instance, empirical data show variations in the socio-economic status

of the community are directly associated with variations in the risk of addiction, we can develop interventions, e.g., increasing social capital in neighborhoods through community interventions. Another example may be studying addictive behavior through the ecological aspect of the self using neuroscientific methods (e.g., the anomalies in the mesolimbic reward system) to see if, say, pharmaceutical interventions targeting the dopamine system are the difference makers. As the above suggests, we can identify risk factors and, ultimately, reduce rates of addiction by studying the multiple dimensions of the self, each of which may specify multiple risk factors.

The self model's creeping reductionist framework also embraces pragmatic goals of psychiatry. Researchers and clinicians have diverse interests: sometimes the goal of research is to reveal the disease pathways at a molecular level; at other times, the goal is to motivate behavioral and habitual changes in a short period in a clinical setting. But different aspects of the multitudinous self model can be used for these various purposes by identifying the different research targets.

Another virtue of the self model is that it transcends the deep debate about the relative values of explanations in psychiatry grounded in the biological sciences versus those in the psychological and social sciences. The multitudinous self model, because it encourages research from cellular and molecular levels to cognitive, personal, and social levels, promises substantial scientific progress in clarifying the etiology of psychiatric illness by working at diverse levels and dimensions. It promotes multiple complementary research programs in psychiatry. Finally, with the help of the multitudinous self model, we can reach scientific insights and develop a better capacity to make specific interventions, without having to make larger assumptions about the relative superiority of biological, psychological, or epidemiological variables.

Conclusion

The self model offers a rich way to understand and address addiction, largely because of its creeping reductionist framework. Creeping reductionism is open to pluralistic explanations, including the effect on addiction of the various aspects of the self, and encourages the development of a diverse portfolio of intervention strategies. It tames unfettered (and unwise) optimism about the use of pharmaceuticals to treat addiction, embraces pluralistic approaches, and refuses to privilege one explanation over another. It does not deny the efficacy of pharmacological treatment methods, but it accepts that, while one individual may benefit from a strictly pharmacological treatment, another may flourish with person-centered psychotherapy. In brief, the notion of creeping reductionism is a conceptually productive way of making sense of addiction, with great practical import.

Notes

1 Note that there are different subspecies of the brain disease models and the self models, each offering a version of the models discussed in this chapter; for brevity, I discuss only these two.
2 Nora Volkow, current head of the National Institute of Drug Abuse (NIDA), and George Koob, head of the National Institute of Alcohol Abuse and Alcoholism (NIAAA), are more cautious than Alan Leshner, who was the head of NIDA in 1997. Their arguments are more nuanced; they now write about the "brain disease model of addiction." They refrain from stating directly that addiction is a brain disease and note the biological and social factors involved in addiction (Volkow *et al.* 2016).
3 See Foddy (2011) to see some objections to the argument that the activation of the brain's reward system is significantly different from that associated with natural rewards, such as food, sex, and social relationships.
4 For a detailed development of this model, see Tekin (2014); Tekin, Flanagan, Graham (in press); Tekin (forthcoming).

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