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Addiction Models and the Challenge of Having Impact

Meyer D. Glantz, Ph.D.

National Institute on Drug Abuse, NIH, 6001 Executive Blvd Room 5185, Bethesda, MD 20892, United States

Addiction continues to be one of the most serious and intractable problems facing society with alcohol use being the third leading preventable cause of death in the United States (Stahre. 2014). The article by Bickel, Crabbe, and Sher (2019) features presentations of two major approaches to addiction by prominent proponents John Crabbe and Warren Bickel with discussion by noted researcher Kenneth Sher. The article provides an opportunity to examine how these approaches model addiction in terms of substance use disorder (SUD) criteria and to consider their potential for impact on alcohol and drug use disorders and interventions in the nonacademic world.

A Rodent Model of Addiction

In addressing the utility of rodent models of addiction, Crabbe describes the reductionist animal model approach to understanding a complex human behavior by first manipulating rodents to simulate distinct key behaviors of addiction and then synthesizing the inferred fundamental principles into a heuristic model. In keeping with the article theme, Crabbe discusses the extent to which rodent models can elucidate each of the diagnostic criteria of DSM-5 (American Psychiatric Association, 2013) SUD diagnoses. The relevance of laboratory research to these criteria ranges from the well-established animal models of tolerance and withdrawal, to the growing animal research on use despite concurrent punishment, to the possibility of rodent research on aspects of persistent use and the choice of substance use over alternative behaviors, and finally to the likely impossible to relate to criteria requiring self-reflection such as using more than intended and desire but failure to quit.

Rodent models of addiction have come a long way since Falk's 1966 paper on "Schedule-Induced Polydipsia." Animal models have made important contributions to the study of substance use and addiction through their explication of relevant learning/behavior principles but important questions remain. What new advances can these behavior models offer and are their further contributions inherently limited due to the fundamental nature of current animal models which do not feature cognitive behaviors? What can be gained from additional observations of alcohol consummatory behavior by non-self-aware animals and to what extent can they inform us further about complex human behavior, dysfunction, and

mglantz@nida.nih.gov.

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intervention that intrinsically requires self-awareness and a cognitive-linguistic based intelligence? Rodent models have characterized some behavioral aspects of human addiction but they have not identified new aspects and they are unlikely to be able to especially due to the cognitive differences between rats and people. For those who might question whether this is a limitation, consider whether addiction research with fauna having even lower self-awareness and intelligence would be as informative as rodent research. Devineni and Heberlein (2010) have proposed that ethanol preference behaviors in Drosophila (fruit flies) model several critical characteristics of human alcohol addiction-like behavior. This may seem like an exercise in reductio ad absurdum but it is doubtful that much can be learned about human addiction from a Drosophila model of alcohol use based on normal fruit fly behavior even though behavior principles apply to all species. Alternatively, studying cats' preference for catnip might be more informative. Comparisons of diverse superficial behavioral similarity across species have limited potential to educe underlying dynamics and principles in proportion to the extent that the comparators are crucially disparate. It would be possible to program a self-driving car to periodically swerve the vehicle but this would not provide much help in understanding erratic driving by intoxicated human drivers.

The limitations of translating complex behavior principles from rodents to humans are not problems that can be addressed by simply doing more research using the current rodent model approach. As a thought experiment, suppose that a machine learning analysis of the extensive existing research literature on rodent behavior produced a simulated e-rat model that was highly accurate in predicting rodent alcohol and drug use behavior in a wide range of already studied laboratory conditions on which it was based as well as many similar as yet untested situations. Decades of research and thousands of studies could readily be conducted in a short period of time. The a-theoretical e-rat simulation might be useful as an economical and efficient way to conduct incremental extensions of current rodent research and some pilot projects, initial passes for drug screens, and probably other productive tasks. But no matter how many research studies were conducted with the rodent simulation, it would not shed new light on human addiction risk factors, recovery and relapse, diagnostic algorithms, or new interventions. The e-rat simulation would neither be based on nor elucidate uniquely human determinative characteristics of addiction any more than it could explicate depression, personality disorders, or suicide. Granted these are psychiatric conditions, but so is addiction.

The task of the rodent addiction model is not only the modeling of people's normal, controlled, socially acceptable alcohol use but also the differentiable modeling of compulsive dysregulated use. It is the distinguishing human characteristics of cognitive linguistic modeling of self and the environment, future oriented appraisal, and a cognitively informed self-regulation interface with affect and behavior that are at least in part what differentiates human compulsion despite awareness of proximal and distal negative consequences from an animal's persistence of use despite concurrent or proximal negative consequences, and differentiates human loss of control despite intention from an animal's using more of a substance over time or with additional effort. Crabbe states that addiction is "fundamentally defined by its behavioral expression." It is more accurate to say that addiction is fundamentally identified by its behavioral expression.

Human addiction related substance use behaviors demonstrating compulsion and loss of control are major cognitively and emotionally involved dysfunctions, i.e. psychopathology, while the animal's substance use behavior is normal rodent behavior in specifically designed non-native environments. It is not difficult to manipulate the rodent's environment to stop the rodent's substance use behavior even after it is well established. Obviously, when animals simulate addiction they are not addicted. It is very difficult to curtail an addicted person's established substance use because they are addicted. As such, rodent models have told us little about recovery or relapse. Furthermore, rodent models do not model either supportive or opposing social influences. Other than pharmacological interventions, it is those distinguishing cognitive human characteristics and social influences that are typically the targets of prevention and treatment. Therefore, it is not surprising that rodent models have contributed little to the development of new intervention approaches beyond those that, although not specifically derived from rodent models, are already implied by behavior theories, e.g., contingency management programs.

Criticisms of animal models of addiction and SUDs began at the same time as the early research (Cicero,1979; Lattal, 2001, Ramsden, 2015). The concern discussed here is that the approach as currently formulated has reached the point of diminishing returns for usefully modeling current critical human relevant addiction questions, especially regarding risk factor and correlate modeling, characterization of disorder trajectories, and improved interventions. Breaking down complex human behaviors into simplified simulations of key observable facets faces the risk of underestimating the emergent qualities and crucial distinctiveness of the original behavior being studied. Even laboratory studies with human subjects can be criticized for relying on necessarily limited simulations (e.g. Weatherly and Phelps, 2006). Of greater concern, while stress, dysregulation, and dysfunction are cardinal characteristics of addiction, they are not incorporated into most rodent studies. This is related to the point made by Sher in the article introduction when he references Martin (Martin et al., 2001) to note that "Not all SUD criteria [i.e. behaviors] are fully consistent with clinical notions of addiction" using the example of hazardous alcohol use which may be associated with disordered and compulsive use or may merely be a product of non-clinical general heedlessness. The point is that modeling observable behaviors may or may not model their distinguishing underlying clinical character. As Sher writes, animal model researchers need to focus more on the "human condition" and clinical relevance.

Expanding rodent model simulations to include closer analogs of negative influences on human addiction related behavior might foster new advances in addiction research. One way this could be done would be by incorporating aspects of stress, dysregulation, and dysfunction which would enhance the human relevance of questions rodent models could address. For example, rodents raised or living under high versus low stress conditions, in solitary versus social environments, or in barren versus stimulating circumstances (e.g. Perry et al, 2019) could be observed to see if they demonstrate a greater preference for or consumption of alcohol or drugs. Selectively bred alcohol-preferring and low-alcohol-drinking rats (e.g. McBride et al., 2014) could be observed to see if they behave differently in stress and challenge environments and if other typical reinforcers lose effectiveness as reinforcers more readily. Different rat strains (e.g. Hamilton, Potenza, and Grunberg, 2014) and individual rats could be rated on behavior analogous to anxiety, stress, impulsivity,

activity level, stimulus seeking or other human traits to see if their substance use behavior is correlated with these ratings. Or, as Crabbe mentions, study of long-term rodent behavior following extended substance exposure could be conducted to see the effects on abstinence trajectories and subsequent likelihood of increased preference for alcohol in stressful environments. Rodent model researchers should explore addressing these types of questions as such research would help characterize the related human circumstances and test the prospects of rodent models.

Crabbe recognizes that there are limits to partial models and to animal models especially for studying psychiatric disorder and negative affect. He notes that most addiction theories assume a disease model including concepts related to dysregulated motivation which animal models do not incorporate. Crabbe speculates that rodent research could be useful in further understanding of recovery and relapse, long term use of the substance, and substance use patterns that exceed the threshold for intoxication. Accomplishing such human relevant research goals would be a challenging but crucial advance for animal models.

A Behavioral Economic Model of Addiction

Bickel describes a behavioral economic model of addiction focusing on the question of why some individuals place consequentially greater value on alcohol and drug use than they do on pro-social reinforcers. The core concept of this approach is that addiction is the excessive preference for immediate reinforcers. The primary description of the phenomenon is delay discounting which is the reduction in value of a reinforcer as a function of the delay to its receipt. This phenomenon has a long history of empirical support. Research in the 1940s found that persistence of behavior that is “consistently more punishing than rewarding” can be explained at least in part by the temporal order or time delay of the punishing or rewarding consequences (Mowrer and Ullman, 1945). This concept became a well-researched element in a number of approaches to related phenomena such as delay of gratification (Mischel, 1974; Tobin and Grazino, 2010), impulsiveness (Ainslie, 1975; De Wit, 2009; Perry and Carroll, 2008), self-regulation (Karoly, 1977; Baumeister and Heatherton, 1996), and sensation seeking (Hittner and Swickert, 2006; Crane et al., 2018). Behavioral economics has not incorporated research on these related phenomena or their associated interventions although doing so would strengthen the model’s explanatory and intervention potential (e.g. . Zheng et al., 2019).

Keeping with the article’s premise, Bickel discusses the research and theoretical applications of behavioral economics to diagnostic themes of the DSM-5 criteria. A well-established research literature applies the concept of delay discounting to describe the behaviors and DSM-5 criteria entailing continuation of substance use despite subsequent negative consequences. DSM-5 criteria involving inability to regulate consumption are depicted in terms of preference reversals as a function of deeply bowed hyperbolic discounting curves.

The last grouping of DSM-5 symptoms, criteria involving the apparent increase in the value of substance use to the detriment of the valuation of typically socially valued reinforcers such as interpersonal relationships and employment, are described in terms of reinforcer pathology, a recent expansion of the theory. This version describes individuals with very

high levels of delay discounting and reward valuation and incorporates the influence of these individual's relative distorted expectations of the reinforcers during the time of their anticipated experience inverting the value of the proximal brief substance use over the distal but extended prosocial reinforcers. Bickel describes Episodic Future Thinking (EFT), a developing intervention intended to foster a greater valuation of future events.

Bickel shows how, to a large extent, the behavioral economics model augmented reinforcer pathology is able to describe the behaviors of the DSM-5 SUD diagnostic symptoms by referencing delay discounting, valuation, preference reversals, and reinforcer pathology, concepts generally applicable to a broad range of both common and problem behavior. A fuller account of the behavioral economics model of addiction with emphasis on reinforcer pathology is available (Bickel et al., 2014).

The behavioral economics model references some concepts and mechanisms from several non-competing approaches. Bickel notes for example that substantial discounting of delayed rewards combined with high valuation, the defining characteristics of reinforcer pathology, are primary contributors to addiction and could be considered an extension of the dopamine hypothesis and other related theories. This compatibility with other theories and research findings allows the behavioral economics model to be an agile framework for describing addiction that is congruent with a broad range of research findings. To a large extent, the behavioral economics model of addiction is a descriptive model with predictive utility drawing heavily on references to mechanisms from other approaches to provide explanations. For example, while individual differences in delay discounting are assumed by behavioral economics, the model does not by itself provide an explanation although proponents could refer to any other model's mechanism for explanation. For another example, the model describes the phenomena of addiction related high discounting and high drug valuation and references neurobiological substrates and genetics as at least compatible empirically supported mechanisms. A review of related neuroimaging research reports relatively consistent findings of predicted neural correlates with delayed discounting in individuals with addictive disorders (Owens et al., 2019). If behavioral economics were to incorporate or at least propose some commitment to these neurobiological mechanisms as explanatory concepts rather than just referencing their compatibility, the model would be strengthened and further developed by findings from an additional field of research. It would also be more subject to empirical validation or disconfirmation.

Without the model having specified mechanisms, it is difficult to think of a substance use behavior or pattern that could not be described in terms of the behavioral economics model regardless of how complex or variant. Explanations relying on references to the variable state and unspecifiable interaction of possible reinforcer valuations including pragmatically unpredictable preference reversals supplemented with references to any mechanism proposed by other modalities allows behavioral economics to account for almost any addiction related behavior, albeit often as a post hoc inference. For example, failed attempts at abstinence are conceptualized as preference reversals although this does not account for successful attempts or the distinguishing circumstances when abstinence is the outcome especially after multiple failed attempts. However, speculation about presumed occurring or not occurring shifts in reinforcer valuation or reinforcer pathology could provide an account

even though this would not really provide an explanation or a system for practical outcome prediction. Other examples include the common situations in which addicted individuals extensively engage in prosocial activities such as work to later acquire alcohol or drugs, or for such individuals to remain abstinent as a means of achieving self-professed desirable prosocial goals, such as visitation with their children, but then relapse. There are different patterns of problem drinking such as intermittent binge drinking and daily moderate continuous drinking. Individuals with comorbid psychiatric disorders or under highly stressful circumstances are more likely to relapse. Many drug dependent individuals say that they use drugs to feel normal rather than to get high but many choose to intermittently get high rather than to titrate their use to lower levels over longer periods of time thus minimizing feeling bad. In those cases where medication assisted treatment is available (methadone and buprenorphine for opioid use disorder, disulfiram for alcohol use disorder, and nicotine patches and gum for nicotine dependence), these medications are often not used by addicted individuals including those professing interest in abstinence; even when they are, the individuals often relapse. While all of these behaviors and others could be interpreted or at least described in terms of the behavioral economics model and reinforcer pathology, any truly expository characterization, especially ones with strong implications for intervention, would require references to explanatory mechanisms drawn from other models for which behavioral economics at most notes potential compatibility. While this may be an issue for many partial models, in the case of behavioral economics, the lack of specifity of the approach which allows it to account for almost any addiction related behavior or research finding also makes it a largely untestable model.

There are a few interventions derived specifically from behavioral economics such as EFT and enhancements of Brief Motivational Interventions. Generally, these interventions encourage a greater focus on and valuation of longer term prosocial reinforcers. Cognitive priming tasks have been shown to decrease delay discounting and are discussed as an intervention, (Sheffer et al., 2016). Overall however, behavioral economics has had little impact on implemented interventions outside of research settings. This may in part be because the explanatory mechanisms integrated in an addiction model are typically the targets and foundation for intervention and such mechanisms are largely absent in behavioral economics.

It seems likely that in order to be successful, behavioral economics based interventions must go beyond efforts to reduce delay discounting of prosocial goals and add efforts to reduce preference for and positive anticipation of alcohol and drug reinforcement. An example of such an approach would be to expand EFT to include revaluation of individuals' probably distorted memories of past substance using events. Another challenge for behavioral economics derived treatments is the likelihood that neuroadaptations associated with addiction so significantly alter experience and therefore expectations of both prosocial and substance related reinforcers (e.g. Tatia et al., 2002) that modest changes to individuals' appraisal of reinforcers may be helpful but insufficient. Pharmacological interventions may help to diminish the positively reinforcing experience of some addicted individuals' substance use. However, the associated neuroadaptations as well as the impoverishment, social losses, and limited practical opportunities common in the lives of addicted individuals make overcoming addiction related reduced sensitivity to non-drug reinforcers and the

restoration of the reinforcement value of prosocial experiences a formidable challenge for an unaugmented behavioral economics based intervention. This is a significant limitation inherent in the current behavioral economics based interventions which would benefit from inclusion of social support and Cognitive Behavior Therapy (CBT) treatment components whose goal would be to increase the pragmatic attainment and maintenance of prosocial reinforcers.

Behavioral economics models are based on cognitive models of decision-making which implicitly assume that while individuals may make misinformed, biased, or otherwise suboptimal decisions, they are basically rational decision makers. However, the model seems to maintain the assumption that even when individuals exhibit severe addiction behavior they are making cognitively intact albeit self-injurious and antisocial decisions based on suboptimal valuations of reinforcers. Even when being applied to addiction and SUD, behavioral economics does not include any concept of psychopathology or cognitive dysfunction. It is very likely that addicted individuals are, at least at times, not fully rational decision makers and that their decision making processes are themselves impaired if not disordered and are therefore a necessary target for intervention. As such, incorporating CBT into behavioral economics based interventions might enhance their effectiveness.

Conclusion

The discussions by Crabbe and Bickel are excellent descriptions of their approaches' conceptualization and operationalization of addiction. Sher raises many useful issues related to addiction, SUDs, and the criteria by which they are identified. He emphasizes that addiction and SUD are complex phenomena probably involving multiple related processes which are depicted by different often competing theoretical models. He points out that SUD is not a single homogenous set of symptoms even though the diverse symptom expressions may reflect some common underlying mechanisms. Sher notes that behavior indicators, risk factors, and consequences are not necessarily equivalent to addiction or to each other. Perhaps most importantly, he distinguishes recovery from the return to a pre-addiction state.

In addition to the points raised here about the individual models, it is clear that both models would be further strengthened by addressing certain common gaps in their conceptualizations. For example, addiction and SUDs are frequently comorbid with other psychiatric disorders (Grant et al., 2004; Kessler et al., 2005; Kessler et al., 2011) and addiction models that can account for comorbidity have more applicability to understanding human disorder, prevention, and treatment (Glantz, 2010; Swendsen et al., 2010). Similarly, craving and the experience and avoidance of negative affect are so much a part of the experience of addicted individuals that they must also be accounted for by robust addiction models (Barker et al., 2004). There are multiple likely relationships between comorbid disorders (Kessler, 1995) including that some seeming comorbidities may be the product of categorically defining nosologies such as DSM-5. Alternative dimensional models (e.g. Caspi et al., 2014; Kotov et al., 2017) propose not only different approaches to diagnosis but also conceptualizations of mental health disorders that emphasize more general underlying characteristics. Protective factors play a role in preventing and mitigating addiction (Glantz and Sloboda, 2002) and developmental factors are major influences on the vulnerability to

and trajectory of addiction (Glantz and Leshner, 2000). Prevalence and trajectories of SUDs vary among different sex and ethnic groups. The two models would be strengthened by at least exploring these perspectives and issues.

The ability of an addiction model to account for DSM criteria is not a measure of its potential utility or impact. If addiction were just problem behavior, a bad habit, or a physical dependence it would be readily addressable in animals and people. Addiction is a psychiatric disorder, a pathology of mental and emotional function exacerbated by the reinforcing and neuroadaptive effects of alcohol and/or drugs. While neither the rodent model nor the behavioral economics models are psychiatric models built on concepts of dysfunction or disorder, they have contributed to our understanding of substance use problems. Both are elegant models with strong metrics and appealing concepts, each supported by more than 40 years of research and theory development. However, neither has had much impact on the real world circumstances of addiction and substance use disorders, on prodromal or diagnostic identification, or on prevention, treatment, or relapse prevention. The impact and value of future contributions by the rodent and behavioral economic models is likely to depend on the extent to which they further explicate the psychiatric and disordered aspects of human addiction and whether they lead to the development of more effective and widely implemented prevention and treatment interventions. At a time when deaths and damaged lives due to alcohol and drug use are at epidemic levels, model-derived research and interventions that have significant real world impact on these human crises are the crucial measure of an addiction model’s value.

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