

Expanding the Biopsychosocial Model: The Active Reinforcement Model of Addiction

Annie Hunt
University of Denver

The contemporary understanding of addiction is expanding rapidly as research across special fields informs treatment and intervention techniques. Current prevention efforts work from the well-accepted biopsychosocial model and are aimed at identifying the underlying causes of addiction and attempting to block them from manifesting, primarily through educational methods. However, once an addiction has already emerged, intervention and treatment efforts should operate from a more comprehensive conceptualization of addiction that takes into account more than just underlying factors – these efforts must address how these factors are currently operating and reinforcing one another. The active reinforcement model proposed in this paper addresses the mechanisms of action that govern the relationships among three primary elements of addiction: a) impaired neurological mechanisms; b) unmet psychological needs; and c) dysfunctional behavior. This model serves as a more comprehensive conceptualization of addiction as it accounts for each of the present factors and places them in an interdependent context. Thus, while the biopsychosocial model effectively addresses the underlying causes of addiction, the proposed active reinforcement model addresses the mechanisms of existing addictions in a more comprehensive manner. A better description of the relationship between each element provides a deeper understanding of the full phenomenon of addiction, and may therefore be more effective in generating successful treatment outcomes than previous models.

Introduction to Addiction

Medical, psychological, and social understandings of addiction have evolved rapidly over the past century, and contemporary addiction research and treatment is becoming increasingly interdisciplinary. Addiction studies, initially based in the field of pharmacology, now incorporate psychological, neurobiological, genetic, environmental, social, and spiritual considerations. Furthermore, addiction is being studied in schools of social work, public health, medicine, and psychology. Addiction has thus become a multi-disciplinary construct that necessitates a wide range of understanding from contemporary practitioners, and as this understanding expands, the professional obligation to maintain a consistent and regulated standard of practice becomes significantly more challenging.

Standardized clinical practices in the field of addiction are developed, tested, and disseminated through clinical research, and the myriad factors influencing this field present an interesting challenge for researchers who must take them into consideration when designing and implementing studies. These studies are the mechanism for generating empirically based findings, which are the necessary prerequisite in allowing new considerations to be incorporated into standardized treatment options. In order to in-

crease evidence-based treatment options in response to the expanding conceptualization of addiction, one must first begin with a clear understanding of the current state of the field, and then propose areas for further academic consideration and research. The starting point for this process is the Diagnostic and Statistical Manual of Mental Disorders (DSM), originally published in 1952, which significantly influences treatment options, research, insurance policies, public opinion, and social stigma. While the DSM acknowledges that the diagnostic classification process is a challenging one, and that there are no strict boundaries dividing one disorder from the others or from no mental disorder at all, it does offer a professional consensus about the categorization and identification of mental disorders at the time of its publication (DSM-5; American Psychiatric Association, 2013). Thus, it provides a reliable tool for establishing standards of diagnoses, terminology, and criteria for classification while at the same time emphasizing the importance of flexibility, appropriate training, and cultural sensitivity during clinical application.

Addiction terminology. Chemical and behavioral addictions have long been recognized as serious and prevalent psychological problems throughout history, so it is interesting that the most recent version of the DSM—the Fifth Edition, published in May 2013—does not actually include the term “addiction” (APA, 2013). The category used in the DSM-5 to de-

Annie Hunt is doctoral student at the University of Denver. Correspondence concerning this article should be addressed to: anniehunt1@gmail.com

scribe the phenomenon colloquially referred to as chemical or substance addiction is titled “Substance Use Disorder,” and it combines the former categories of substance abuse and substance dependence (from the DSM-IV) into a single disorder that is to be measured on a scale from mild to severe (APA, 2013). Neither did the previous edition, the Fourth Edition, Text Revision, published in 2000 by the American Psychological Association, include the term “addiction,” as the term ‘dependence’ won out over ‘addiction’ by one vote during the last revision process (DSM-IV-TR, APA, 2000; O’Brien, Volkow, & Li, 2006).

Specific and accurate terminology plays an important role in the conceptualization of this phenomenon. According to the National Institute on Drug Abuse (NIDA), the term “dependence” indicates a physiological need or dependency on a substance. This is evidenced by a biological adaptation to the substance in which the body requires more of it to achieve an effect (the phenomenon of tolerance) and also manifests physical indicators if use is suddenly stopped (the phenomenon of withdrawal; NIDA, 2012). Physical dependence, however, can occur with continued use of many different substances, including those taken as prescribed, and does not necessarily include the psychological or behavioral consequences that characterize an “addiction” or “disorder.”

The term “disorder,” according to the APA (2012), includes the following:

1. A behavioral or psychological syndrome or pattern that occurs in an individual
2. That reflects an underlying psychobiological dysfunction
3. The consequences of which are clinically significant distress or disability
4. Must not be merely an expectable response to common stressors and losses or a culturally sanctioned response to a particular event
5. That is not primarily a result of social deviance or conflicts with society.

Thus, “disorder” indicates the presence of behavioral and psychological symptoms, but fails to clearly articulate the aspect of compulsory repetition that the terms “dependence” or “addiction” include in their

definitions.

Finally, the term “addiction,” according to the National Institute on Drug Abuse, indicates compulsive use of a substance despite harmful consequences, such as failure to fulfill social, work, or family responsibilities, and an inability to stop using the substance of one’s own accord (2013). Additionally, according to the APA website, addiction falls under the category of “Mental Health Disorders/Issues,” and is defined as “a chronic brain disease that causes compulsive substance use despite harmful consequences” (APA, 2012). Arguably, the most comprehensive and specific definition of addiction comes from the American Society of Addiction Medicine (ASAM; 2010), who define an addiction as:

a primary, chronic disease of brain reward, motivation, memory and related circuitry . . . [which] is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors. Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one’s behaviors and interpersonal relationships, and a dysfunctional emotional response (ASAM, 2010).

It is interesting to compare the terms to identify differences as well as overlaps. According to the above definitions, dependence on a substance does not necessarily constitute an addiction, and an addiction does not necessarily involve physiological dependence (NIDA, 2012). Furthermore, a disorder does not necessarily constitute an addiction. These terms therefore cannot be used interchangeably, as they each indicate the presence of different symptoms.

It should be noted, however, that the DSM-5 does use the category of “Addictive Disorders” to describe pathological gambling as a behavioral addiction (APA, 2013). This is the only condition listed in this category, despite general clinical recognition of other non-chemical, behavioral addictions such as sex addiction, internet addiction, compulsive tanning, and compulsive shoplifting (e.g., Grant, Potenza, Weinstein, & Gorelick, 2010). Neither the NIDA definition of disorder nor the APA defini-

tion of addiction include non-chemical addictions, despite the widespread social, medical, and cultural call for recognition and treatment of these particular problems (Griffiths, 2000; Wang et al., 2013).

This paper argues that one of the most critical aspects of active addiction treatment is addressing the phenomenon of being unable to stop certain substance use or behaviors solely of one's own volition – namely, the compulsive aspect of the condition regardless of known negative consequences. As of the most current edition, the DSM still does not comprehensively articulate the presence and significance of this symptom. The continued exclusion of this component indicates that at this point in time, contemporary research has still not sufficiently pinpointed what exactly this phenomenon is, what it involves, how it operates, and how to address it. It is this phenomenon that the proposed active reinforcement model attempts to describe, validate, and address by drawing together parts of prior conceptualizations and constructing a more comprehensive model. This paper argues that once an addiction is manifest, it is actively reinforced by the relationships between three essential factors: a) impaired neurological mechanisms; b) unmet psychological needs; and c) dysfunctional behavior, the combination of which results in the compulsive aspect of the phenomenon. Understanding the relationship between these factors and how they actively reinforce addiction would offer a starting point for developing interventions aimed at disrupting these relationships.

Previous conceptualizations of addiction.

Addiction has previously been conceptualized using different models, with the most historically prominent being the “adaptive” and the “disease” models. The adaptive model preceded the disease model, and suggested that addictions develop when specific psychological needs – such as acceptance, autonomy, competence, or confidence – are not met; this was also termed “integration failure” (Alexander, 1990). The adaptive model argues that addictions develop to meet these specific psychological needs, and that the addictive behavior provides a sense of support, reassurance, or meaning that individuals feel is missing in their lives. The disease model, also sometimes

called the medical model, posited that addiction is neither an issue of failed willpower nor the result of conscious repeated habitual behavior, but is rather a chronic, progressive medical illness characterized by abnormalities or defects in brain functioning (Sheehan & Owen, 1999). Silkworth (1939) was one of the pioneers of this model, originally applying it to alcohol dependence. In the primary text of the Alcoholics Anonymous program, he described alcoholism as an unusual or distorted behavioral response to alcohol consumption, and described problematic chronic drinking as a manifestation of a “physical allergy” to alcohol (Alcoholics Anonymous, 2001). Sheehan and Owen (1999) argue that the disease model represents a more comprehensive explanation of addiction through its depiction of neurological deficits and abnormal behavioral responses. These models offered initial foundations for the development of addiction studies and treatment, and they remained prominent until the emergence of George Engel's “biopsychosocial model” (Engel, 1978).

Current conceptualization of addiction. The biopsychosocial model, which is used to describe many different mental disorders, is arguably the most prominent construct used to conceptualize addiction today (Alonso, 2004). This model built upon the disease model by accepting that addiction involves abnormalities in brain functioning, but then expanded that model by integrating the subjective psychological experiences of individuals into the conceptualization of illness. It suggests that an understanding of the patient's subjective experience is critical in developing accurate diagnoses and successful treatment options (Borrel-Carrio, Suchman, & Epstein, 2004). The biopsychosocial model thus seeks to explain suffering, disease, and illness as generated by multiple causes, including social, biological, and psychological factors. This inclusion of subjective psychological components into the disease model expands the concept of addiction to include individual experiences, perceptions, stressors, and perspectives as mediating factors in the expression of clinical illnesses and medical problems. Thus, this model helps to bridge the gap between the medical and psychological fields, and is extensively accepted in the field of addic-

tion studies and psychology today (Alonso, 2004).

Drawing from the above-mentioned previous and contemporary definitions of addiction, three core elements of addiction can be identified – unmet psychological needs, impaired neurological mechanisms, and problematic behaviors. However, the adaptive and disease models do not sufficiently describe the relationships involved in the phenomenon of addiction because they suggest simple linear causality between either coping and problematic behaviors or disease and problematic behaviors. This paper argues that the previous conceptualizations of addiction, including the biopsychosocial model, do identify the core elements of addiction, but fail to sufficiently demonstrate the reciprocal relationships among them. Emerging research reveals evidence that unmet psychological needs, impaired neurological mechanisms, and problematic behaviors can act as both causes and effects in the construct of addiction (Castellani, Wedgeworth, Wootton, & Rugle, 1997; Grant, Brewer, & Potenza, 2006; Hyman & Malenka, 2001).

While the biopsychosocial model offers a more substantive argument for acknowledging the presence of combined psychological and biological/pharmacological factors in the development of addiction, and partially identifies some of the relationships involved among these factors, it does not offer a comprehensive conceptualization of all of the relationships between these factors that contribute to active addiction and compulsive behaviors. It states that these different factors play a role in impacting outcomes, and that there is a relationship between biological/psychological elements and external functioning, but it makes use of an eclectic approach that does not specifically or explicitly describe how that relationship functions and impacts addiction (Ghaemi, 2009). Furthermore, it fails to establish the impact that psychological factors have on neurobiological factors, the impact that neurobiological factors have on psychological factors, and the reverse impact that dysfunctional behaviors may have upon psychological and biological functioning—an important new relationship that has been demonstrated in emerging research (Hyman & Malenka, 2001). This paper will propose a new model that includes the critical elements of addiction and places them in an interdependent context

that offers a more comprehensive understanding of how addictions function. It will also defend each of these relationships with recent research findings.

Proposed “Active Reinforcement Model of Addiction.” Current research consistently indicates the presence of neurological, psychological, and external/behavioral components in the overall conceptualization of addiction, though each element may have varying degrees of intensity and causality (i.e., one element may be more powerful or have more influence than others, depending on the individual; Grant, Brewer, & Potenza, 2006). This paper argues that there is a cause-effect relationship between all three elements, meaning that each element both influences and is reinforced by the other two elements. In light of this knowledge, a new model, entitled the “Active Reinforcement Model of Addiction” (Figure 1), is proposed. From this conceptualization, the critical principle that emerges is not the importance of determining which element came first, or finding an underlying reason for why the addiction emerged. Rather, the focus is shifted to the importance of acknowledging how all three coexist and reinforce one another in an interdependent context once an addiction has become active. From there, an altered focus for treatment and interventions can be proposed, again shifting the focus of treatment techniques from why to how addictions function.

Prevention efforts generally focus on the question of why addictions develop and use education, risk protection techniques, and resilience training based on the proposed answers to this question, though there still is no consensus on exactly what causes addiction (SAMHSA, 2013). The field is full of varying hypotheses and theories that attempt to explain addiction’s underlying causes in order to create successful prevention efforts and education programs aimed at stopping addiction before it starts. This paper, however, is not focused on prevention efforts, but rather proposes use of a different model to guide treatment of active addictions. Prevention efforts are distinctive from treatment efforts, according to the American Society of Addiction Medicine, though they may both be used concurrently in certain circumstances (ASAM, 2005). Treatment, according to ASAM, is aimed at helping individ-

ACTIVE REINFORCEMENT MODEL OF ADDICTION

uals currently suffering from an addiction. Once addiction symptoms (according to either the DSM or APA criteria) are manifest, prevention models should be substituted for a working understanding of how the addiction is actively functioning. While research on preventive measures is valuable and will continue to hold its place in the field, this paper argues that addiction treatment and intervention should be based on a more comprehensive conceptual model of how addictions are actively sustained.

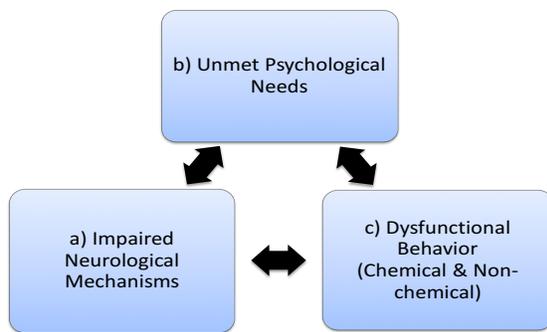


Figure 1: Active Reinforcement Model of Addiction. This figure illustrates the critical relationships that reinforce active addiction.

As illustrated by the figure above, the phenomenon of addiction is sustained by the presence of and relationship among three elements: a) impaired neurological mechanisms; b) unmet psychological needs; and c) dysfunctional behavior. This model serves as a comprehensive conceptualization of addiction that incorporates the main elements of addiction and places them in an interdependent context. It helps to organize the concept of active addiction into a structured mechanism defined by reinforcing relationships, which may allow studies and interventions to specifically focus on particular sections or relationships in the model. At the same time, it also offers a full depiction of the phenomenon, which may help to remind practitioners that each element and relationship must be attended to in treatment. This paper will describe how many existing theories and interventions fit directly into the active reinforcement model, and will explain how each encompasses one or some of the six relationships described: $a \rightarrow$ (causing) b , $a \rightarrow c$, $b \rightarrow a$, $b \rightarrow c$, $c \rightarrow b$, and $c \rightarrow a$. Any element,

standing alone or impacting only one of the other elements, does not necessarily lead to the emergence of an addiction. This model theorizes that all relationships must be present to some degree in order to constitute an active addiction. This paper will begin to justify this theory by examining the current working definition of what the term “addiction” means.

Definition of Addiction (a, b & c)

All people engage in self-regulatory behaviors in response to their biological, psychological, social, and spiritual wants and needs (Bandura, Adams, & Beyer, 1977). Many people use substances or engage in risky behaviors to meet these needs, and these do not always develop into disruptive or chronic patterns of use. The crossover from substance use or occasional dysfunctional behavior to the full manifestation of addiction is ambiguous and difficult to define. One must consider the motivations for, frequency and intensity of, and consequences of the substance use or dysfunctional behavior in order to determine whether it can be considered problematic (c).

As noted previously, the DSM acknowledges that there are no absolute boundaries in diagnoses, and the same flexibility must be utilized when attempting to define addiction (APA, 2013). Despite the challenge the phenomenon of addiction poses, it is still critical to develop a generally accepted working definition so that researchers and clinicians can work toward standardizing the field, developing best practices, and regulating the associated treatment options for addiction.

The American Society of Addiction Medicine’s definition of addiction, previously quoted in this paper, clearly supports the concept of a biological element in the reinforcement model of addiction, suggesting that neurological dysfunction (a) directly causes problematic psychological (b) and behavioral (c) manifestations ($a \rightarrow b$, $a \rightarrow c$). It also indicates that the behavior (c) in turn can affect psychological and emotional components (b) of the individual’s life ($c \rightarrow b$). In the longer definition, ASAM goes on to explain that brain “morphology, connectivity, and functioning are still in the process of maturation during development and young adulthood, and early exposure to substance use is another significant factor in the development of addiction” (ASAM, 2011). This suggests that dysfunctional behaviors such as chem-

ical abuse (c) also impact brain development (a) in a phenomenon known as “neuroadaptation” ($c \rightarrow a$; ASAM, 2011). It is worthwhile to note that this definition includes both “substance use and other behaviors” in its definition, and that the inclusion of both chemical and non-chemical addictions is a critical element to the reinforcement model, as will be discussed later. Thus, ASAM’s definition of addiction fits into the proposed model, and supports some of the relationships it describes ($a \rightarrow b$, $a \rightarrow c$, $c \rightarrow a$, $c \rightarrow b$).

Another widely-recognized definition of addiction came from the former president of the American Society of Addiction Medicine, Dr. Michael Miller, who stated:

At its core, addiction isn’t just a social problem or a moral problem or a criminal problem. It’s a brain problem whose behaviors manifest in all these other areas . . . [the] disease is about brains, not drugs. It is about underlying neurology, not outward actions. (Smith, 2011, p. 901)

This statement also directly supports the reinforcement model, and demonstrates the significance of neurophysiology in affecting the full conceptualization of addiction ($a \rightarrow b$, $a \rightarrow c$). Goodman (1990) proposed a similarly well-accepted definition of addiction:

Addiction may be defined as a process whereby a behavior that can function to produce pleasure and provide relief from internal discomfort, [and] is employed as a pattern characterized by (1) recurrent failure to control the behavior (powerlessness) and (2) continuation of the behavior despite severe negative consequences (unmanageability).

This definition highlights the behavioral element included in the reinforcement model, and defines what is meant by “dysfunctional” behavior (c) and psychological causes and consequences (b). While it does not specifically mention non-chemical behaviors, it does not explicitly exclude them, and as such they too can be incorporated into this conceptualization. Furthermore, this definition addresses the impact that these behaviors have on psychological functioning, and supports and fits into the reinforcement model ($b \rightarrow c$, $c \rightarrow b$). Smith and Seymour (2004) included the additional element of “compulsive use or engagement in the behav-

ior” to this definition, which suggests an underlying biological urge ($a \rightarrow c$). Moreover, they suggested that addictive behaviors (c) are used to gain either psychic (mood-related), recreational (social or activity-related), or instrumental achievement (performance-related) rewards (b). This also directly ties into the reinforcement model in that it explains the relationship that dysfunctional behaviors can have on psychological functioning and the attainment of needs ($c \rightarrow b$).

Application of the term “addiction” to both chemical and behavioral disorders. One critical theoretical consideration that supports the reinforcement model is the inclusion of non-chemical or behavioral dependencies (c) in the broader conceptualization of addiction. Chemical dependencies alone are not necessarily considered dysfunctional, as many medical patients develop dependencies even when they take their medications as prescribed. These dependencies do not necessarily result in a compulsive or disruptive pursuit of chronic use. Dependencies become dysfunctional only once they develop causal relationships with psychological and biological processes ($c \rightarrow b$, $c \rightarrow a$). Additionally, certain behaviors, such as gambling, hand-washing, or exercising, are not considered dysfunctional on their own—these behaviors only become dysfunctional when they disrupt psychological and biological processes ($c \rightarrow b$, $c \rightarrow a$), resulting in a non-chemical addiction. For example, hand-washing is a normal human behavior, but if it becomes a psychological obsession and an individual feels compelled to do it repetitively, then it has developed into an addiction ($c \rightarrow a$, $c \rightarrow b$).

Traditionally, the clinical terms “addiction” and “addictive behavior” have been applied exclusively to substance abuse and dependency, but there is growing empirical evidence of a related category of “non-chemical” addictive behaviors, “including gambling, eating disorders, and sexual behavior,” that have recently been included in the conceptualization of addiction (Donovan & Marlatt, 2005, p. 4). There are easily recognizable external patterns that are similar between chemical and behavioral addictions, including tolerance, withdrawal, repeated unsuccessful attempts to stop, and significant impairment in areas of life functioning. However, the emerging recognition of biology’s influence on addiction has en-

couraged researchers to explore whether behavioral addictions and substance addictions recruit similar biological processes ($a \rightarrow c, c \rightarrow a$; Grant, Brewer, & Potenza, 2006). Brain science and neural imaging have begun to validate food and sex addictions, compulsive shopping and gambling, and eating disorders, among others, as “process,” “non-chemical,” or “behavioral” addictions that can be included in the new, broader category of addiction disorders.

Emerging research indicates that dysfunctional behaviors can be powerful determinants of psychological functioning ($c \rightarrow b$) and can also cause neuroadaptation ($c \rightarrow a$; Lubman, Yucel, & Pantelis, 2004; Hyman & Malenka, 2001). It has also been demonstrated that dysfunctional behavior can be the direct result of brain deficits or maladaptive psychological states ($a \rightarrow c, b \rightarrow c$) (Grant et al., 2006). These findings support the incorporation of non-chemical addictions into the reinforcement model, as they follow the same relationship patterns of chemical addictions. This recognition greatly enhances the argument for the reinforcement model of addiction, as it helps to illuminate the interconnectedness of both internal and external factors involved in this phenomenon, regardless of the involvement of substances and chemicals.

The 2013 DSM revisions reflect this increasing awareness of the role of non-chemical dysfunctional behaviors in the construct of addictions through their inclusion of “Gambling Disorder” (APA, 2012). This movement to include a non-chemical addictive disorder demonstrates that researchers and practitioners are migrating toward the general understanding that both chemicals as well as behaviors can impact neuroadaptation within the brain’s reward system ($c \rightarrow a$). Smith (2012) reinforced this by stating that addiction disrupts the areas of the brain responsible for regulating cognitive, emotional, and social behaviors, and Marks suggested that “syndromes of behavioral addiction share [similar neurological] features with those of substance abuse . . . [including] obsessive-compulsive disorder, compulsive spending (including gambling), overeating, hypersexuality, kleptomania, and perhaps trichotillomania, tics, and the Tourette syndrome” ($c \rightarrow a, c \rightarrow b$; Marks, 1990, p. 1389).

As supported by these findings, both chemical and behavioral dependencies can be included in the

reinforcement conceptualization of addiction. The emerging understanding of the ability of external behaviors to influence brain chemistry, as well as the recognition that brain chemistry affects external behaviors, demonstrates that these two factors are mutually influential ($a \rightarrow c, c \rightarrow a$). Thus, including problematic and dysfunctional behaviors that do not include chemicals greatly supports the active reinforcement model, and further illuminates the extent to which external factors influence internal processes and vice versa.

Three Core Components and Six Core Relationships

The psychological and behavioral components of addiction have been well established in contemporary research, so an extensive discussion of these elements is not necessary in this paper and they will be only briefly mentioned below. Research on the neurobiological components, however, is the more recent and emerging area that will be discussed in more detail.

Psychological Components of Addiction ($b \rightarrow a, b \rightarrow c$)

The active reinforcement model indicates a distinct relationship between psychological components (b), such as stressors, the effects of environmental influences, thoughts, and emotions, upon both the brain as well as behavior ($b \rightarrow a, b \rightarrow c$). There are numerous studies that support the causal relationship between unmet psychological needs and dysfunctional behaviors, as well as the impact of psychological stress on brain chemistry and neurological functioning ($b \rightarrow c, b \rightarrow a$) (Castellani et al., 1997; Whang, Lee, & Chang, 2003; Young, Boyd, & Hubbel, 2000; Sinha, 2001). These two relationships in the reinforcement model of addiction are well established, and a detailed discussion of these two mechanisms can be easily found in contemporary literature and is thus beyond the scope of this paper. One particularly good explanation of these relationships can be found in Franken (2003), who explains the relationship between psychological urges and their impact upon neurological functioning and addictive behaviors.

Behavioral Components of Addiction ($c \rightarrow a$, $c \rightarrow b$)

As previously mentioned, dysfunctional or risky behaviors alone do not comprise addiction. Dysfunctional behavioral or chemical dependencies only become addictions when they develop relationships with the psychological and neurobiological elements of the active reinforcement model. This means that when a behavior or chemical use becomes a method of psychological coping to deal with stress, or when the individual feels compelled to engage in this behavior due to pressing internal impulses, the full relationship of these factors manifests as an active addiction ($c \rightarrow a$, $c \rightarrow b$). There are numerous studies that demonstrate the wide range of addictive behaviors, including both non-chemical and chemical dependencies, and a discussion of all of the behavioral elements associated with addiction is beyond the scope of this paper. For a more detailed discussion of these relationships, see Smith (2012), Smith and Seymour (2004), Marks (1990), or Hyman and Malenka (2001).

Neurobiological Components of Addiction ($a \rightarrow b$, $a \rightarrow c$)

While the current definitions of addiction explain some of the neurological and psychological influences on behavior ($a \rightarrow c$, $b \rightarrow c$), it is critical to also incorporate recent research that indicates that behaviors influence neurological and psychological functioning ($c \rightarrow a$, $c \rightarrow b$). This research indicates that behaviors can contribute to neuroadaptation and psychological problems (Lubman, Yucel & Pantelis, 2004). An individual may not necessarily have an underlying neurological deficit to begin with, but could develop one as a result of engaging in dysfunctional behavior or encountering psychological stressors (Lubman et al. 2004). Thus, neurobiology is not necessarily the primary causative factor of this phenomenon. The active reinforcement model effectively demonstrates both the significance as well as the relationship of neurobiology to the overall conceptualization of addiction, instead of placing it as the primary causal factor.

The active reinforcement model indicates that neurobiology both influences and mediates the relationship between dysfunctional behaviors and psy-

chological issues, and in combination with these factors it can generate chemical or behavioral addictions. Neuroimaging and neuropsychological studies have revealed clear differences in brain function between chronically addicted and non-addicted individuals, suggesting that addiction is indeed associated with alterations in brain functioning and neuropsychological changes (Lubman, Yucel, & Patelis, 2004; $a \rightarrow c$, $c \rightarrow a$). The research they describe has been primarily focused on the brain's reward pathways, which involve dopamine and serotonin receptors. Dopamine and serotonin are neurotransmitters released by the brain as a result of certain actions and behaviors, and they are associated with the experience of pleasure and reinforcement and can function as 'rewards' in the brain. Hyman and Malenka (2001) report that the chemicals released as a direct result of engagement in addictive behaviors are both rewarding, or interpreted as intrinsically positive by the brain, as well as reinforcing, meaning that the behaviors involved with these rewards tend to be repeated ($c \rightarrow a$, $a \rightarrow c$). Thus, substances and behaviors that produce these neurotransmitters can become very powerful reinforcers that influence future behavior and can result in inhibitory dysregulation (Hyman & Malenka, 2001). This means that individuals either develop lowered inhibitions against risky behavior or experience urges so strong that they overwhelm typical inhibitions ($c \rightarrow a$; Lubman et al., 2004).

Tolerance. Neurochemical changes in response to addiction often manifest in the development of tolerance, defined as a decrease in the effect of an addictive substance that often results in more frequent or intense engagement in addictive behavior ($a \rightarrow c$). Individuals who develop these altered brain states may demonstrate tolerance as "reward deficiency syndrome, a hypothesized hypo-dopaminergic state involving multiple genes and environmental stimuli that puts an individual at high risk for multiple addictive, impulsive, and compulsive behaviors" ($a \rightarrow c$; Grant et al., 2006, p. 925). This hypo-dopaminergic state is one of the proposed mechanisms of brain chemistry in addiction. Lower levels of dopamine (or decreased activation of dopamine circuits and receptors) may result in an individual's increased at-

tempts to compensate for these deficits through particular chemicals or behaviors ($a \rightarrow c$). Higher levels of tolerance can promote increased and frequent use, which may then result in dependence ($c \rightarrow a$). Sensitization. Another result of chronic addiction is sensitization. This occurs through enhanced reward responses in the brain resulting from repeated administration of a substance or engagement in an addictive behavior ($c \rightarrow a$; Hyman & Malenka, 2001). Individuals who develop more sensitive brain states may experience a higher level of euphoria after engaging in behaviors that release dopamine or serotonin. Due to the experience of more substantial “rewards,” they may have greater difficulty in controlling impulses to engage in and continue addictive behavior ($c \rightarrow a$, $a \rightarrow c$). With chronic use of or engagement in these behaviors, adaptations at genetic, molecular, and cellular levels occur within distinct brain regions that counter acute drug effects in an attempt to maintain internal homeostasis ($c \rightarrow a$). When intake of the substance ceases, these neuroadaptations initially persist and act unopposed, resulting in a characteristic rebound syndrome, or “withdrawal” (Lubman et al., 2004). Hyman and Malenka (2001) note that this response can develop beyond just a physical or psychological liking of one’s addictive behavior into the experience of intense urges or “wanting.” It is at that point that physical dependence can cross over to compulsive desire and pursuit as neurological systems become hypersensitive, which significantly increases the incentive to seek out these stimuli ($a \rightarrow c$, $c \rightarrow a$; Hyman & Malenka, 2001).

Disrupting the Relationships between the Active Elements of Addiction

As discussed, the active reinforcement model of addiction demonstrates six relationships between three primary elements of addiction: biological deficits (a), unmet psychological needs (b), and dysfunctional behaviors (c); these relationships are described as $a \rightarrow b$, $a \rightarrow c$, $b \rightarrow a$, $b \rightarrow c$, $c \rightarrow a$, and $c \rightarrow b$. Any of these elements in isolation do not necessarily indicate an addiction, and as such cannot be considered primary causal factors of this phenomenon. For example, an individual can engage in dysfunctional or risky behavior without it affecting their

psychological or biological functioning. Similarly, one may experience stress from unmet psychological needs but never turn to dysfunctional behavior as a coping mechanism, or may suffer from neurological deficits without the additional experience of unmet psychological needs or attempting to compensate for these deficits behaviorally. Rather, it is the relationships among these factors – not the factors themselves – that indicate an active addiction. Therefore, this model suggests that addiction treatment research should be devoted to disrupting these mechanisms and developing interventions to block the relationships between the factors that combine to sustain addiction (indicated by the sign X). A comprehensive treatment plan must therefore involve interventions to disrupt these relationships ($a \times b$, $a \times c$, $b \times a$, $b \times c$, $c \times a$, $c \times b$). Addressing and resolving one element can reduce the severity of the addictive behavior, but this paper argues that attention to all three factors and their respective relationships with one another is vital to successful, comprehensive addiction treatment.

Treatment of Biological Factors ($a \times b$, $a \times c$)

Emergency care. The very first step in addiction treatment is to focus on the most urgent needs of the client (Wallace, 2005). This typically involves meeting essential physiological requirements and ensuring that basic physical functioning is supported and maintained, as many clients cannot take action in addressing problem behaviors if they are not first stabilized (Wallace, 2005). Regardless of whether clients enter addiction treatment voluntarily or not, they can initially present in a state of shock, trauma, severe emotional disturbance, despair, depression, and other varying states of instability, and at that stage they may not be capable of identifying or attending to their basic needs. Thus, prior to any psychological interventions, the client must be placed in a safe, calm environment without easy access to their addictive drug or behavior, and they must be thoroughly screened for any pressing physiological problems that can be immediately addressed ($a \times b$, $a \times c$). This includes any kind of treatment of overdose symptoms, medicine for management of withdrawal symptoms during detoxification, administration of essential nutrients or electrolytes for severe cases of

eating disorders, and other related medical treatment.

Pharmacological treatment. The use of prescription drugs to treat neurobiological deficits is a critical component of addiction treatment. This has become the subject of extensive research as the conceptualization of addiction as a brain disease becomes more prevalent. While a full discussion of the pharmacological component of addiction treatment is beyond the scope of this paper, it is worth noting that the emerging trends of successful prescription drug use in the treatment of addiction often involve drugs associated with supporting or enhancing the serotonin or dopamine pathways of the brain (Grant et al., 2006). Alleviating these neurological deficits can directly promote psychological health and reduce engagement in dysfunctional behaviors as coping mechanisms (a X b, a X c; Grant et al., 2006).

Treatment of Psychological Factors (b X a, b X c)

Interdisciplinary approaches to addiction studies have allowed for more comprehensive incorporation of the psychological aspects of addiction. There have been several widely recognized movements that have defined and influenced the field as it relates to addiction treatment, and while there are many different orientations to psychological counseling, one of the primary approaches – cognitive behavioral therapy – will be discussed here as an example of a psychological treatment that can be directly incorporated into the active reinforcement model.

The cognitive-behavioral orientation is one of the main approaches used in addiction treatment today, and it purports that that human thoughts and behavior are driven by the conditioning and reinforcement that people experience throughout their lives (b → c). Dysfunctional thoughts and behavior, such as behavioral and chemical addictions, are considered to be the result of the development of inaccurate and unhealthy life schemas, which are the mental framework used to organize information about the self and the external world (b → c) (Alford & Beck, 1997). The goals of cognitive-behavioral therapy are therefore to focus on individual problematic behaviors and thoughts, identify their origins and influences, and challenge and change them

as needed to promote healthier psychological functioning and recovery (b X c). Cognitive-behavioral therapy, or CBT, developed out of this psychological orientation and is prominent in addiction treatment programs today. It is a highly standardized therapeutic process that utilizes such techniques as identifying individual goals, focusing on present problems, exposure therapy, cognitive restructuring, behavior change, and psychoeducation (b X c). Thus, while there are many approaches to the treatment of the psychological factors of addiction, CBT is one example of a well-established practice that is compatible with and supports the active reinforcement model.

Treatment of Behavioral Factors (c X a, c X b)

A final critical component of the active reinforcement model is the existence and relationship of dysfunctional behaviors and their impact on both psychological functioning and neurology. The most prominent treatment approach is the promotion and facilitation of abstinence or sobriety from dysfunctional behaviors or chemical dependencies as a way to disrupt the final relationships in this model (c X a, c X b). Other approaches, such as the harm-reduction model, emphasize the importance of moderation, self-regulation, honest and open self-reporting of engagement in problematic behaviors, and other measures to significantly reduce engagement in disordered behavior (Marlatt & Tapert, 1993). One critical aspect of intervention efforts aimed at treatment of behavioral factors is the consideration of replacement behaviors that may be utilized in the absence of the typical addictive behavior, and the importance of providing the client with adaptive rather than harmful substitutes for these behaviors (Shaffer et al., 2004). Treatment of behavioral factors also involves a combination of the techniques used to treat the neurological and psychological aspects of addiction, and each of the interventions described above also work to disrupt the relationships between dysfunctional behaviors and psychological or neurological mechanisms (c X a, c X b).

Conclusion

The working conceptualization of addiction

ACTIVE REINFORCEMENT MODEL OF ADDICTION

continues to develop as emerging research across multidisciplinary fields informs treatment and intervention techniques. While prevention efforts are often aimed at identifying the underlying causes of addiction, intervention and treatment should operate from a more comprehensive conceptualization of addiction that is focused on the mechanisms of action among three primary elements: a) impaired neurological mechanisms; b) unmet psychological needs; and c) dysfunctional behavior. This paper argues that all three must be present and involved in an active relationship with one another for an active addiction to be manifest. Thus, the proposed active reinforcement model serves as a more comprehensive conceptualization of addiction that accounts for and incorporates all of the elements of addiction and places them in an interdependent context that may be more effective in generating successful addiction treatment outcomes than previous models.

The next steps in validating the proposed model are to evaluate it using research studies and psychometric evaluations. One of the most critical aspects of this model is its comprehensive incorporation of multiple concepts of addiction, and demonstrating its effectiveness empirically could start with an evaluation of each of these elements and the six relationships described by the active reinforcement model. Demonstrating the validity of these individual relationships empirically and introducing intervention efforts intended to disrupt them would support the relevance of this model and demonstrate the need for continued exploration of this conceptualization of addiction. Once the individual relationships have been empirically validated, a treatment approach that addresses all components should be implemented, evaluated, and compared to models that operate from a less comprehensive conceptualization of addiction.

In conclusion, the active reinforcement model serves as a more comprehensive conceptualization of addiction as it accounts for multiple interrelated factors. While the currently accepted biopsychosocial model effectively addresses the underlying causes of addiction, the proposed active reinforcement model addresses the mechanisms of existing addictions in a more comprehensive manner. A better description of the relationship between each element

provides a deeper understanding of the full phenomenon of addiction, and may therefore be more effective in generating successful treatment outcomes.

References

- Alcoholics Anonymous. (2001). *Alcoholics anonymous* (4th ed.). New York: A.A. World Services.
- Alexander, B. K. (1990). The empirical and theoretical bases for an adaptive model of addiction. *Journal of Drug Issues*, 20, 37-65.
- Alford, B. A., & Beck, A. T. (1997). The relation of psychotherapy integration to the established systems of psychotherapy. *Journal of Psychotherapy Integration*, 7, 275-289. doi:10.1023/B:JO-PI.0000010884.36432.0b.
- Alonso, Yolanda. (2004). The biopsychosocial model in medical research: the evolution of the health concept over the last two decades. *Patient Education and Counseling*, 53(2), 239-244. doi:10.1016/S0738-3991(03)00146-0.
- American Psychiatric Association. (2000). Introduction. In *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). doi:10.1176/appi.books.9780890423349.7443.
- American Psychiatric Association. (2012). Substance use and addictive disorders. In *DSM-5 Development*. Retrieved from <http://www.dsm5.org/proposedrevision/Pages/SubstanceUseandAddictiveDisorders.aspx>
- American Psychiatric Association. (2013). Substance-related and addictive disorders. In *DSM-5 Development*. Retrieved from <http://www.dsm5.org/Documents/Substance%20Use%20Disorder%20Fact%20Sheet.pdf>
- American Society of Addiction Medicine. (2011). Public policy statement: definition of addiction. Retrieved from <http://www.asam.org/research-treatment/definition-of-addiction>.
- Bandura, A., Adams, N. E., & Beyer, J. (1977). Cognitive processes mediating behavioral change. *Journal of Personality and Social Psychology*, 35, 125-139.
- Borrell-Carrio, F., Suchman, A. L., & Epstein, R. M. (2004). The biopsychosocial model 25 years lat-

- er: Principles, practice, and scientific inquiry. *Annals of Family Medicine*, 2(6), 576-582.
- Brauser, D. (2001, Aug 31). Addiction a brain disease, ASAM says. *Medscape*. Retrieved from <http://www.medscape.com/viewarticle/748867>.
- Buhler, C. (1971). Basic theoretical concepts of humanistic psychology. *American Psychologist*, 26(4), 378-386. doi:10.1037/h0032049.
- Castellani, B., Wedgeworth, R., Wootton, E., & Rugle, L. (1997). A bi-directional theory of addiction: Examining coping and the factors related to substance relapse. *Addictive Behaviors*, 22, 139-144.
- Donovan, D. M., & Marlett, G. A. (Eds.) (2005). *Assessment of Addictive Behaviors* (2nd ed.). New York: Guilford Press.
- Engel, G. (1978). The biopsychosocial model and the education of health professionals. *Annals of New York Academy of Sciences*, 310, 169-181. doi:10.1111/j.1749-6632.1978.tb22070.
- Frager, R. (1979, Fall). What is transpersonal psychology? *Association for Transpersonal Psychology Newsletter*, 6.
- Franken, I. H. A. (2003). Drug craving and addiction: integrating psychological and neuropsychopharmacological approaches. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 27(4), 563-579.
- Ghaemi, S. (2009). The rise and fall of the biopsychosocial model. *The British Journal of Psychiatry*, 195, 3-4. doi:10.1192/bjp.bp.109.063859.
- Goodman, A. (1990). Addiction: definition and implications. *British Journal of Addiction*, 85, 1403-1408.
- Grant, J. E., Brewer, J. A., & Potenza, M. N. (2006). The neurobiology of substance and behavioral addictions. *CNS Spectrums*, 11, 924-930.
- Grant, J. E., Potenza, M.N., Weinstein, A., & Gorelick, D. A. (2010). Introduction to behavioral addictions. *American Journal of Drug and Alcohol Abuse*, 36, 233-241.
- Griffiths, M. (2000). Internet addiction – time to be taken seriously? *Addiction Research and Theory*, 8, 413-418.
- Hall, A. (2001). Internet addiction: college student case study using best practices in cognitive behavior therapy. *Journal of Mental Health Counseling*, 23, 312.
- Hastings, A. (1999). Transpersonal psychology: The fourth force. In D. Moss (Ed.), *Humanistic and transpersonal psychology: A historical and biographical sourcebook* (pp. 192-208). Westport, CT, US: Greenwood Press/Greenwood Publishing Group.
- Hyman, S. E., & Malenka, R. C. (2001). Addiction and the brain: The neurobiology of compulsion and its persistence. *Nature Reviews Neuroscience*, 2, 695-703.
- Keutzer, C. S. (1984). Transpersonal psychotherapy: Reflections on the genre. *Professional Psychology: Research and Practice*, 15, 868-883.
- Lubman, D. I., Yucel, M., & Pantelis, C. (2004). Addiction, a condition of compulsive behavior? Neuroimaging and neuropsychological evidence of inhibitory dysregulation. *Addiction*, 99, 1491-1502.
- Marlatt, G. A., & Tapert, S. F. (1993) *Harm reduction: Reducing the risks of addictive behaviors. Addictive Behaviors across the Lifespan: Prevention, Treatment, and Policy Issues*. Newbury Park, CA: Sage.
- Marks, I. (1990). Behavioural (non-chemical) addictions. *British Journal of Addictions*, 85, 1389-1394.
- Maslow, A. H. (1968). *Toward a Psychology of Being*. New York: Van Nostrand.
- May, R. (1983). *The discovery of being*. New York: W.W. Norton.
- National Institute on Drug Abuse. (2012). *Principles of Drug Addiction Treatment: A Research-Based Guide* (Third Edition). NIH Publication Number: 12-4180. Retrieved from: http://www.drugabuse.gov/sites/default/files/podat_1.pdf
- O'Brien, C. P., Volkow, N., & Li, T. K. (2006). What's in a word? Addiction versus dependence in DSM-V. *American Journal of Psychiatry*, 163, 764-765.
- Shaffer, H. J., LaPlante, D. A., LaBrie, R. A., Kidman, R. C., Donato A. N., & Stanton, M. V. (2004). Toward a syndrome model of addiction: multiple expressions, common etiology. *Harvard Review of Psychiatry*, 12, 367-374.
- Sheehan, T., & Owen, P. (1999). The disease model.

- In B. S. McCrady & E. E. Epstein (Eds.), *Addictions: A comprehensive guidebook* (pp. 268–286). New York: Oxford University Press.
- Sinha, R. (2001). How does stress increase risk of drug abuse and relapse? *Psychopharmacology*, 158, 343–359.
- Slife, B. D., & Barnard, S. (1988). Existential and cognitive psychology: Contrasting views of consciousness. *Journal of Humanistic Psychology*, 28, 119-136. doi:10.1177/0022167888283008.
- Smith, D. E., & Seymour, R. B. (2004). The nature of addiction. In R. H. Coombs (Ed.), *Handbook of addictive disorders: A practical guide to diagnosis and treatment* (pp. 3–30). New Jersey: John Wiley & Sons, Inc.
- Smith, D. E. (2011). The evolution of addiction medicine as a medical specialty. *American Medical Association Journal of Ethics*, 13, 900-905.
- Smith, D. E. (2012). Editor's note: The process addictions and the new ASAM definition of addiction. *Journal of Psychoactive Drugs*, 44, 1-4.
- Substance Abuse and Mental Health Services Association. (2011). *Leading change: A plan for SAMHSA's roles and actions, strategic initiative #1: Prevention of substance abuse and mental illness*. SAMHSA Pub ID: SMA11-4666. Accessed from: <http://store.samhsa.gov/shin/content/SMA11-4629/03-Prevention.pdf>
- Wallace, B. C. (2005). *Making mandated addiction treatment work*. Lanham, MD: Rowman & Littlefield Publishers, Inc.
- Wang, L., Luo, J., Bai, Y., Kong, J., Luo, J., Gao, W., & Sun, X. (2013). Internet addiction of adolescents in China: Prevalence, predictors, and association with well-being. *Addiction Research and Theory*, 21, 62-69.
- Whang, L., Lee, S., & Chang, G. (2003). Internet over-users' psychological profiles: A behavior sampling analysis on internet addiction. *Cyberpsychology and Behavior*, 6, 143-150.
- Yalom, I. D. (1980). *Existential psychotherapy*. New York: Basic Books.
- Young, A. M., Boyd, C., & Hubbell, A. (2000). Prostitution, drug use, and coping with psychological distress. *Journal of Drug Issues*, 3, 789–800.