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Substance and Non-substance Addiction

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Foreword

Like most other neuropsychiatric diseases, addiction remains stubbornly difficult to treat, with recidivism rates that may often exceed 90%. Indeed, the chronic relapsing nature of the disease is supported by the multiple times an individual presents for treatment before some are able to successfully develop long-term abstinence. The absence of a clinically verified biomarker for disease severity, and thus the ability to assess treatment efficacy, precludes declaring that sustained abstinence, in fact, indicates the absence of disease, rather than the absence of the behavior. This is aptly demonstrated by the high relapse rate seen after an individual returns to society following prolonged inpatient treatment or penal incarceration. The best current measurement of treatment success for drug dependence is urine testing, which is doubly unfortunate since it merely signals the presence or absence of a substance in the body, usually within a limited time window. However, the absence of drug does not signal absence of disease, nor does the presence of drug signal the presence of disease (e.g., the ability of most to partake in social alcohol consumption).

Over the past 40 plus years, tremendous new insights into the molecular, cellular, and neurobiological underpinnings of drug dependence have been gained, and the introduction of preclinical models that attempt to mirror addictive behaviors, e.g., drug self-administration, conditioned place preference and aversion, and cue- and stress-induced drug reinstatement, has led to a better understanding of the behavioral consequences of acute and chronic drug administration. Additionally, the explosion of research that has followed the introduction of truly breakthrough neurobiological and genetic tools, such as optogenetics and DREDD, has led to greater insight into the underlying neuronal cell types, circuits, and networks that demonstrate addiction-dependent plasticity. However, few new therapeutic agents have been discovered, and novel drug targets are still being sought. Indeed, only tobacco, alcohol, and opiate dependence even has approved medications—and these all have limited efficacy. And there are currently no efficacious pharmacotherapies for stimulants, i.e., cocaine and amphetamine or marijuana. Moreover, the so-called behavioral addiction, including pathological gambling, internet addiction, and binge eating, also is without specific pharmacological interventions. That said, various

behavioral interventions, including cognitive behavioral therapy, contingency management, and motivational enhancement therapy, have been shown efficacious in a subset of both substance- and non-substance-dependent individuals.

Why has this veritable explosion of neuroscience knowledge not translated to better outcomes for our patients, whose lives and those of their families have been so devastated by addiction? Many hypotheses have been offered including the inability of animal models to fully capture the totality of addiction and thus do not provide a useful platform upon which to test novel pharmacotherapies. For example, while dopaminergic transmission and specific receptor subtypes have long been known to be dramatically altered following both acute drug administration and chronic drug administration in both preclinical and human studies, neither dopamine receptor agonist nor antagonists have been found to be clinically useful. Nevertheless, medicinal chemists continue the search for better, more specific dopaminergic ligands, with much emphasis today on the D3 subtype.

An alternative hypothesis is that addiction is a uniquely human disease that has very complex and intersecting causes including psychosocial, affective, socioeconomic, genetic, and neurobiological substrates. It is inherently a disease of pathological overlearning of a series of interlocking stimuli (environmental, social, pharmacological) with specific affective and cognitive outcomes. Indeed, the human literature has posited the dysregulation of complex cognitive constructs including reward sensitivity, impulsivity and response inhibition, decision-making and value determination, acute and long-term affective regulation, and ability to foresee and plan for the future. It is not clear how one captures these constructs with one or more preclinical models, some of which may have faced validity although for the most part, none have been shown to have predictive validity. Moreover, multiple constructs are likely to be dysregulated to comprise underlying behavioral regulation. For example, both impulsivity and cognitive control could both be compromised leading to compulsive behaviors, rather than simply too much of the former or not enough of the latter.

To make matters even more complex, it has long been appreciated that those who suffer from addiction also present clinically with other neuropsychiatric disorders, the most prevalent of which include depression, anxiety, and psychosis. Moreover, in most cases, the individual is not simply addicted to a single substance, but rather presents with dependence and/or abuse to multiple drugs. Thus, it would seem difficult with our current knowledge to propose a single pharmacological or even behavioral intervention to capture both the dual dependence and dual diagnoses of most of our patients. Moreover, treatment and research advances in the field of behavioral addictions have significantly lagged that of the pharmacological addictions, even with the growing acceptance that such compulsive behaviors as gambling follow many of the principles learned from substance dependence.

Despite the abovementioned challenges, significant advances have been made in recent years that leave one more optimistic. The field of noninvasive brain imaging, mostly MRI based, has provided for the first time the ability to directly observe changes in brain chemistry, structure, and function in the behaving human. Positron emission tomography has provided exciting insights into brain transmitter and

receptor alterations in disease, and the explosion in human genetics and epigenetics has revealed a number of polymorphisms that may give insights into the risk levels and treatment options (i.e., personalized medicine) for our patients, all of which bring me to the current volume.

Zhang and his colleagues provide an extensive review of the current state of the art in human addictions. They powerfully demonstrate the behavioral and cognitive parallels between substance and non-substance dependence and argue compellingly in several of the chapters how further knowledge of the latter may profitably inform the former. That is, from a neurobiological perspective, the behavioral addictions may have some research advantages in that detected alterations in brain structure and function are not likely the result of an exogenously administered drug, which has the ability to engage multiple brain and peripheral (e.g., hormonal) systems both directly and indirectly. Indeed it is often difficult to disambiguate the pharmacological effects of a drug (e.g., attentional and working memory improvements following nicotine administration) from the dependence-producing properties of the agent. This is not the case for behavioral addictions where brain alterations are most likely the result of (or directly cause) the compulsive and destructive behaviors underlying the disease. Thus, what is importantly argued by many of the chapters is that much of what can be learned by studying these behavioral addictions may be profitably applied to all addictive disorders.

An important strength of the chapters in this book is the continued attempt to link factors that relate the behavioral to drug addictions; similarities and differences in the cognitive aspects of sensation seeking, intertemporal choice behavior, attentional bias, or inhibitory control are compared and contrasted. An important discussion of the difficulties of developing predictive preclinical models of compulsive behavioral addictions is also presented. Another strength of this book is the inclusion of somewhat less studied and less appreciated factors in dependence including neurotrophic factors, inflammatory factors (which notably has become much more appreciated of late in the field), neurovascular injury, as well as potential genetic and epigenetic biomarkers such as peripheral microRNAs. Finally, various potential therapeutic interventions are reviewed from the more traditional pharmacological and behavioral treatments to those less well studied including, nutritional, physical therapy and traditional Chinese medicine approaches.

What may be missing from the data presented and arguments made in these chapters is the appreciation that it may take more than understanding *differences* between the addicted and non-addicted brain and differences in substance and non-substance abuse to ultimately provide better treatments. While of course such basic knowledge is extremely important, what is needed is a better understanding of which one or more of these differences are, in fact, *predictive* of disease severity and treatment outcome success. This will require significant additional and logistically difficult and financially costly longitudinal studies. Much of the literature is comprised of cross-sectional research where a particular dependent variable is often shown to be different between populations. But two things that are different from each other are not necessarily predictive of the future. They could merely be different, and while such differences could be the result of the addiction, they could also

represent premorbid, inherited differences and not be related to disease trajectory. This can only be determined from longitudinal studies.

Another missing area of research is that of risk factors for developing addiction. Once again, this is both vitally important and very difficult to obtain data. Moreover, such risk factors are often confounded by various normal neurodevelopmental changes to the individual. It is well known that most addictions begin during the early to late adolescent years—a time when the brain is undergoing rapid changes and thus very susceptible to environmental and pharmacological challenges.

That said, it is almost impossible for any single volume to cover the totality of addiction, and some topics are inevitably given less attention than others. This volume has taken a different approach and, rather than tackling the totality of the field, has chosen and successfully accomplishes the more novel and socially and medically important aspects of comparing the behavioral and drug addictions, with the premise that they share common neurobiological mechanisms and thus knowledge of one can inform the other. This book is recommended for students just starting out in the field, experienced treatment providers, and others who are interested in better understanding the complexities of the addictive disorders that so devastate communities around the globe. It provides a timely and up-to-date review of many of the current clinical and basic research issues and points out important gaps in our knowledge that need to be filled to improve the outlook of our patients.

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Part I
Overview of Substance and Non-substance Addictions

Chapter 1
Received View of Addiction, Relapse and Treatment

Yamikani Ndasauka, Zhengde Wei, and Xiaochu Zhang

Abstract It is important to highlight that attempts at understanding and explaining addiction have been made for centuries. It is, however, just five decades ago, with the growth of science and technology that more interest has been observed in this field. This chapter examines different views and theories that have been posited to understand and explain addiction. More attention will be given to prominent views that seem to draw consensus among researchers and medical practitioners. The first section of the chapter introduces the addiction debate, the different theories that have been provided to explain it from different perspectives and disciplines such as neurosciences, philosophy and psychology. Then, the chapter discusses different views on the role of relapse and what it entails in understanding addiction. The second section discusses different proposed and used forms of treating addiction. Thus, the chapter discusses the received view of addiction, the understanding of relapse as a critical element in addiction and treatments.

Keywords Addiction • Relapse • Treatment

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1.1 Understanding Addiction

What is addiction? In this section, we discuss this question as tackled from three different perspectives (psychological, biological and social-cultural perspectives) and demonstrate how each perspective, if independently conceived and propounded falls short in adequately addressing the questions. We will consequently present a nuanced view of addiction, largely accepted among scholars termed biopsychosocial model of addiction, taking into account the three perspectives whilst avoiding the weaknesses of independently holding either. Although this model has been discussed in key literature on addiction, especially substance related addiction, there is minimal discussion of the sociological/cultural perspective in behavioural addictions. This chapter seeks to add to this knowledge gap and will pay much attention to the social-cultural connotations of addiction.

1.1.1 Biological/Medicinal Perspective of Addiction

American Society of Addictive Medicine defines addiction as a primary, chronic disease of brain reward, motivation, memory and related circuitry [4]. Addiction is thus characterized by inability to constantly abstain, diminished behavioural control, craving, diminished recognition of significant problems with one's behaviour and dysfunctional emotional response. Addiction affects neurotransmission and exchanges within reward structures of the brain, including the nucleus accumbens, anterior cingulate cortex, basal forebrain and amygdala, such that motivational structures are altered. Addictive behaviours supersede healthy and self-care related behaviours. Addiction also affects neurotransmission and interactions between cortical and hippocampal circuits and brain reward structures, such that the memory of previous exposures to rewards (such as food, sex, alcohol, drugs and the internet) leads to a biological and behavioural response to external cues, in turn triggering craving and/or engagement in addictive behaviours [4].

It is widely accepted that the initial reinforcing effects of most addictive substances and behavior rely heavily upon the induction of large and rapid increases in the level of DA in the nucleus accumbens. DA, a multifaceted neurotransmitter, is involved in the fine-tuning of motor and cognitive function, modulation of salience attribution and attention, and regulation of reward and motivation. For instance, imaging studies have shown that in drug-addicted individuals, supra physiological levels of DA in the nucleus accumbens are followed by marked decreases in dopamine function [5, 20, 39–41, 77].

Further, in trying to understand and treat addiction, the biological/medicinal perspective relies on the view of addiction as being driven by reward. Reward is generally identified with pleasure, which reinforces the addictive behaviour. "The mesolimbic dopamine circuit is a hard-wired system in the brain … that provides pleasure in the process of rewarding certain behaviour" ([8]:132). In addicted individuals, natural rewards as well as the entire reward system are compromised. Addictive substance

and behaviors are simply effective at temporarily blocking the negative reinforcement that addicted individuals experience during abstinence, resulting in conditioned learning towards the substance or behavior and previously neutral stimuli associated with it expounds [2, 31]. This increases the risk for relapse when an addicted person is exposed to the addictive substance or behavior or their cues [78].

In this view, both seeking and use or engagement in behaviour are motivated by innate pleasures caused by the addictive substance/behaviour. When the substance/behaviour no longer produces the same positive effect, people tend to increase use or engagement thereby leading to abuse. Addicts enter into withdrawal, taking or engaging more and more of the substance or the behaviour to produce pleasurable effects to counter the negative experiences of withdrawal [34].

Some neurological studies have proposed a different approach on the role of brain systems in addiction that emphasizes how addictive substances and behaviours affect motivations and incentives that individuals experience [12, 54, 62]. The emphasis of these theories is on seeking over using or engagement, proposing that "wanting" and seeking are central components of addictive behaviour.

Based totally on analysis with animal models, Robinson and Berridge [60, 61] developed a psychological model of brain performance and abuse. Rather than reinforcement, Robinson and Berridge projected that the central brain system concerned in substance abuse- the mesolimbic Dopastat system mediates incentive saliency. They "suggested that it's the method of incentive saliency attribution that transforms … the neural and psychological representations of stimuli, in order that they become particularly salient stimuli, stimuli that attract attention and become particularly engaging and wished, thereby eliciting approach and guiding behaviour to a particular goal" ([61]:104). Robinson and Berridge highlight 'wanting' as the individual feeling of incentive saliency, providing the need to pursue and use a substance or have interaction during an explicit behaviour.

In addition, Robinson and Berridge's theory emphasised the importance of associative learning and context in shaping the attribution of saliency, instead of substances/behaviour making an interior feeling of enjoyment. This view, wherever saliency is an element of larger activity interactions with the surroundings, helps open the door for biopsychosocial analysis through the thought of psychological and cognitive content processes. Robinson and Berridge did not deny the importance of positive reactions to addictive behaviour and substances, positing that "liking" plays a central role within the initiation of substance use and engagement in additive behaviour. However, they projected that association in nursing individual's sensitization to wanting drives problematic use. Through sensitization or associated accumulated reaction to substance/behaviour, "substance cues trigger excessive incentive motivation for substances, resulting in compulsive seeking" ([63]:36). Thus, Robinson and Berridge's theory projected that with increasing levels of use, the affected brain cells manufacture a greater-than-normal saliency signal [38]. Supported, this increase within the quantity of saliency signalled the drug-addicted individual experiences a strong need for substance/behaviour.

In clinical terms, this heightened prominence of substance and activity cues and connected behaviours corresponds to the compulsive seeking seen in drug abuse. In different words, the excessive prominence drives the compulsion to use, from seek-

ing out medicine that now do not offer an equivalent enjoyable result to issues with relapse once addicts are trying to keep up abstinence. Thus, as compared to the classic reward approach, this theory helps make a case for sure problematic aspect of substance abuse. However, the analysis that led to the creation of this theory was primarily based totally on work with rats and needed some remodelling to be utilized in a social science project [38].

Robinson and Berridge [60–62] have conjointly emphasised wanting because of the subjective expertise associated with incentive prominence attribution. This wanting- an acutely awareness need for substance/behaviour drives each seeking and relapse. Thus, the excessive wanting of incentive prominence is seen because of the proximate mechanism driving cravings, the compulsive urge and/or need to use substance or interact in habit-forming behaviour [25]. Anthropology analysis has confirmed that "wanting" is a typical means that drug abusers represented the expertise of desire [45]. Thus, wanting received support as a relevant domain for exploration.

1.1.2 Psychological Perspective of Addiction

Addiction and Recovery, a popular site for information on addiction and people seeking help for addiction in the USA, defines addiction as a relationship with drugs or alcohol [behaviour] in which you use more than you would like to use, and you continue to use despite negative consequences [1]. People use drugs or alcohol and engage in some behaviour to escape, relax, or to reward themselves. But over time, drugs/alcohol and even some behaviour make people believe that they cannot live without them, or that they cannot enjoy life without using or engaging in them.

Psychology Today [55] defines addiction as a condition that results when a person ingests a substance (e.g., alcohol, cocaine, nicotine) or engages in an activity (e.g., gambling, sex, shopping) that can be pleasurable but the continued use/act of which becomes compulsive and interferes with ordinary life responsibilities, such as work, relationships, or health. Users may sometimes not be aware that their behaviour is out of control and causing problems for themselves and others.

According to the criteria of the American Psychiatric Association (DSM-IV) and World Health Organization (ICD-10), addiction should meet three of the following; (1) **Tolerance;** using more and more drugs/alcohol or engaging more and more in a particular behaviour over time. (2) **Withdrawal**; experiencing physical or emotional withdrawal when you have stopped using or engaging in a particular behaviour. Some signs of withdrawal include anxiety, irritability, shakes, sweats, nausea, or vomiting when abstaining from the particular addictive drug or behaviour. (3) **Limited control;** using a substance or engaging in behaviour more than you would like. This often times leads to regret after the activity but you still feel the need to continue using the substance or engaging in the behaviour. (4) **Negative consequences; c**ontinued use of substance or engagement in a behaviour even after experiencing negative consequences to mood, self-esteem, health, job, education or family. (5) **Neglected or postponed activities;** putting off or reducing social, recreational, work, educational or household activities because of substance use or

engagement in some behaviour. (6) **Significant time or energy spent;** spending a significant amount of time obtaining, using, concealing, planning, or recovering from use of substance or engagement in behaviour. This also involves thinking about the substance or behaviour, concealing and minimizing usage and engagement but failing to sustain it. (7) **The desire to cut down;** thinking about cutting down or controlling usage of substance or engagement in behaviour and unsuccessfully attempting to cut down or control usage or behaviour [55].

Although these criteria are largely applied to substance addiction, they have recently been adopted in some behavioural addictions like gambling addiction, Internet addiction and video and computer game addiction. One key question that arises when discussing addiction, especially behavioural addiction, is the fact that most behaviours that society may consider improper may well meet the above criteria. So, what really distinguishes addiction from other 'improper' behaviours or substances? This question is what distinguishes the biological, psychological cum philosophical and social cum cultural perspectives.

The above descriptions of addiction make mention or two critical notions that merit further discussion, thus, addiction as loss of control and thus involuntary and addiction as an impulsive disorder.

1.1.2.1 Addiction and Voluntary Action

Does an addicted person act freely and is the engagement in addictive behaviour a voluntary act? As noted earlier, addicted persons feel a strong urge to engage a particular behaviour. These persons find it difficult to resist the urge and consequently feel obliged to fulfil it in order to curb the pain that follows from not fulfilling it. For the medicinal perspective of addiction, this struggle and failure to resist indicates a form of compulsive disorder that ultimately is some sort of dysfunction in the brain pattern. As a result, an individual is not herself but is compelled to act in a particular manner by the 'disease'. On the contrary, the psychological perspective holds that addicted persons act voluntarily. In this section, we argue that addicted persons act voluntarily in the minimalist sense. That is to say, the disruption of the reward system in the brain acts as a major hindrance for the psyche to put into action decisions produced under the normal deliberative process. Aristotle's conception of *akrasia*, or weakness of the will, gives us an intermediary interpretation on the cause and process of addiction. This compromising position between the medicinal perspective and psychological perspective assumes both physical and mental impairment of an individual as the cause of addiction.

One characteristic of *akrasia,* which seems to be the dividing point of the two perspectives, concerns freewill and intentionality. This characteristic, as noted by Mele [43], is that incontinent action is "free, intentional action contrary to the agent's better judgment." However, not all intentional actions against one's better judgement may be considered as *akratic*. For Mele [44], some actions are compelled. This is the main point employed by the medicinal perspective in explaining drug addiction. The medicinal perspective claims that addicts are compelled. Though they act intentionally against their better judgement, they are compelled to

act such by the disease in them. For the psychological model, this compulsion in addiction does not determine action; thus, it is not a necessary cause of action. Now, how is it possible to act voluntarily against one's own better judgment?

For Aristotle, the weak-willed know in so far as the relevant facts are available to them. As Sarah Broadie and Christopher Rowe [9] note, these individuals "are not unconscious or hypnotized," and they see no need to check if they have made the right decision. On the other hand, their knowledge seems not to be making any difference to their choices. Thus, "it is not on active duty when it ought to be, or not fully so (for it might be making them ashamed even as they act)" [9]. The knowledge that these individuals have is not practically realised because it is not impacting them or making any difference in them. Aristotle is a man of action. For Aristotle, the actual point of knowledge or being aware of what one should be doing is to do it, and not to contrast it with what one thought would have been done and feeling ashamed. The weak-willed demonstrate a failure to translate universals into particulars and use them in their present situation. As pointed out earlier, this failure is due to lack of discipline. For Aristotle, proper training and character building is fundamental in making the mind the right motivator of human action.

Now, are addicts free or do they engage in addictive behaviour voluntarily? Addiction may be understood as a case of weak-will. As noted in Aristotle, it may well be categorised as a battle of the mind and body/desires. What is central at this point is the power of deliberation and its ability to motivate action. The soul, despite being influenced by physical processes of the body, retains its agency and ability to influence the body. An influential approach on motivation of action is what Wallace calls the hydraulic conception of desire [79]. This position is inclined towards Hume's conception of passion as the motivating factor of action. Desires are thought as vectors of causal force to which we as agents are subject and which determine the actions we end up performing. This approach seems inadequate because it deprives an individual of her self-determination and the agent is depicted as subject to forces which are irresistible in that situation. This conception goes against phenomenological evidence of human agency and self-determination.

1.1.2.2 Addiction, Compulsivity and Impulsivity

To clearly discuss the concept of compulsivity, it is essential to discuss its relation to impulsivity, a multifaceted construct bearing numerous possible definitions [59]. Key elements of impulsivity include a predisposition toward rapid reactions, automatic and quick response to desires, insensitivity to delayed rewards and lack of reflection when making decisions [14]. Although healthy individuals may possess impulsive personality traits, excessive impulsivity is a key defining feature of many neuropsychiatric conditions, including attention deficit/hyperactivity disorder, substance use disorder, antisocial behaviour, as well as many behavioural addictions [11, 58].

Compulsivity refers to persistent or perseverative behaviour that's inappropriate to associated things, which have no obvious relationship to an overall goal. Compulsive behaviours are typically unpleasantly monotonous and done as habits [13]. Compulsive behaviours show hanging persistence, generally enduring for long

periods despite being harmful to the individual. Compulsiveness may be a hallmark of many psychiatric conditions and is determined most notably in neurotic disorder (OCD). Alternative conditions that are thought to exhibit compulsive options embody uptake disorders, substance dependence and behavioural addictions like compulsive Internet use. The same as impulsivity, compulsiveness might arise from failures in response inhibition or "top-down" reasoning, associated over-stimulated drive state, or a mixture of those factors [23, 65].

Clinical observation has shown that patients with primary impulsive psychopathology (addictive gambling or addictive gaming) might demonstrate neurotic options, whereas patients with primary compulsive psychopathology (e.g., OCD) might score high on impulsivity ratings, and/or have impulsive-aggressive symptoms or comorbid impulse management disorders [66]. Additionally worth noting is that overlapping impulsive and compulsive options might develop in otherwise equivalent disorder. Totally different trajectories of impulsivity and compulsiveness among single disorders complicate the manner in which these disorders are understood by clinicians, also on how they're treated [23]. Rather than considering the two constructs as polar opposites, an alternate and additional realistic position is to contemplate impulsivity and compulsiveness as orthogonal factors across a variety of disorders, wherever either construct could be identified, there is a likelihood, higher or lower for the availability of the opposite construct.

Now, addiction provides a very important opening into debates regarding compulsiveness and impulsivity. Current psychobiological models perceive addiction as transition between impulsive and compulsive behaviour [11, 35]. Impulsivity may be at the foundation of an inclination to pursue short-run rewards led to by addictive behaviour and substances and is a powerful mechanism within the early stages of addiction (thus, problematic use or abuse) [18, 76]. With regular engagement in addictive behaviour or use of substance over extended amount of your time, impulsivity as a learning mechanism becomes over-trained and then develops into compulsive habits.

Two vital options of habitual behaviours and substance taking are: (1) the behaviour is driven by associations triggered by stimuli and not by an evident goal/reward; and (2) the individual is unable to reverse the repetitive activity pattern, therefore resulting in compulsive behaviour. In biological science terms, the transition from voluntary action in behaviour or substance use to a lot of habitual or compulsive modes looks to represent a shift in brain systems. This shift on top of things correlates clinically with a move aloof from absolutely strengthened behaviour actuated by reward seeking in problematic behaviour or drug abuse toward negatively strengthened behaviour actuated by the rejection of withdrawal symptoms in full-blown dependence and addiction [15].

Impulsivity plays a significant role in the early stage of addiction while maladaptive learning processes and habit formation lead to the development of compulsive behaviour in the later stage of addiction. The two should not hence be taken as parallels, but rather as one that builds on another. Dealing with impulsivity may help prevent addiction, but dealing with compulsivity may help in treating addiction.

In summary, a psychological cum philosophical perspective of addiction views addiction as a product of psychological maladjustment and functional impairment.

1.1.3 Social/Cultural Perspective of Addiction

We have been discussing the Biopsychosocial model of addiction. We have used this model to answer the questions: "what is addiction?" "What leads to addition?" and "what sustains addiction?" So far, we have reviewed the biological reasons people can get addicted. The biological portion of the BPS model considers addiction a brain disease with biological, chemical, and genetic roots. We have also reviewed the psychological reasons people can get addicted. The psychological portion of the model views addiction as a learned behaviour, a problem of faulty thinking, or of developmental delay. Other psychological disorders also contribute directly or indirectly to the development of an addiction. However, as we have attempted to demonstrate, holding polar views of either psychological or biological/medicinal perspectives does not fully account for the problem of addiction. Rather, a more nuance position, emanating from the discussion of voluntary action in Aristotle avoids weaknesses of the independent perspectives whilst retaining their strengths. This position acknowledges the role of impulsivity and compulsivity as a learning process and takes into account vulnerabilities that affect the biological brain processes or reward.

As psychology is concerned with understanding individual human behaviour, sociology is concerned with understanding the behaviour of larger groups (families, organizations, societies, cultures). Sociologists and psychologists both study the influence of these groups on individual behaviour. From a sociological perspective, addiction is a harmful behaviour that affects both individuals and groups. As such, we can only understand and correct addiction within the context of the society in which it occurs.

In this section, apart from referring to published texts and discourse on sociology of addiction, we take a steep turn to refer to text from literally writer, who attempts to elucidate the problem of addiction in a literal manner. This text is written by an individual who has had experience living with addicted persons and done informal research and talked to people from different backgrounds and cultures on this topic. Before getting into the text, we briefly discuss the social-cultural perspective of addiction.

Milkman and Sunderwirth [46] view addiction as a learned behaviour. The authors also note the consistently strong correlation between one's addiction and concurrent engagement or substance use by friends—a finding that suggests greater support for a sociological understanding of addiction. Social construction explanation posits that addiction is meaningful only within the conceptual categories available within culture and framed by social context [6]; therefore, the "particular features of and the meanings attributed to addiction experiences, as well as the behaviour thought to follow from them, are culturally specific" ([57], p. 316).

Culture may simply be defined as a group's learned and shared pattern of values and beliefs. These values and beliefs guide group members' behaviour and their social interactions. Cultural norms, practices and conceptions are transmitted from one generation to another through families. For instance, if one culture experienced oppression in the past; through learning of family history and imitation, feelings developed as a result of the oppression such as feelings of hopelessness, fear and loss are passed on to next generation who pass on to the next generation and so forth.

Such understanding of social and cultural forces helps in answering the question of how people get addicted. Three primary socio-cultural influences are important in responding to this question, namely, culture, families, and social support. Below, I will briefly discuss an excerpt from a TED TALK in order to highlight the role of family and social support in the development process of addiction. Johann Hari speaking in 2013 at a TED Talk Show- titled *Everything You Think You Know About Addiction Is Wrong*, said:

> Professor Alexander built a cage that he called "Rat Park," which is basically heaven for rats. They've got loads of cheese, they've got loads of coloured balls, and they've got loads of tunnels. Crucially, they've got loads of friends. They can have loads of sex. And they've got water bottles, the normal water and the drugged water. But here's the fascinating thing: In Rat Park, they don't like the drug water. They almost never use it. None of them ever use it compulsively. None of them ever overdose. You go from almost 100 percent overdose when they're isolated to zero percent overdose when they have happy and connected lives… Now, when he first saw this, Professor Alexander thought, maybe this is just a thing about rats, they're quite different to us. Maybe not as different as we'd like, but, you know—but fortunately, there was a human experiment into the exact same principle happening at the exact same time. It was called the Vietnam War. In Vietnam, 20 percent of all American troops were using loads of heroin, and if you look at the news reports from the time, they were really worried, because they thought, my God, we're going to have hundreds of thousands of junkies on the streets of the United States when the war ends; it made total sense. Now, those soldiers who were using loads of heroin were followed home. The Archives of General Psychiatry did a really detailed study, and what happened to them? It turns out they didn't go to rehab. They didn't go into withdrawal. Ninety-five percent of them just stopped… Professor Alexander began to think there might be a different story about addiction. He said, what if addiction isn't about your chemical hooks? What if addiction is about your cage? What if addiction is an adaptation to your environment?...Looking at this, there was another professor called Peter Cohen in the Netherlands who said…, maybe we shouldn't even call it addiction. Maybe we should call it bonding. Human beings have a natural and innate need to bond, and when we're happy and healthy, we'll bond and connect with each other, but if you can't do that, because you're traumatized or isolated or beaten down by life, you will bond with something that will give you some sense of relief. Now, that might be gambling, that might be pornography, that might be cocaine, that might be cannabis, but you will bond and connect with something because that's our nature. That's what we want as human beings. [26]

The series of experiments quoted in this excerpt highlight the role of the environment; of society and family; and their role in addiction. The history of a culture and some cultural values may advertently lead to excessive engagement in some behaviour. For instance, in "cultures where drinking is integrated into religious rites and social customs, where the place and manner of consumption are regulated by tradition and where, moreover, self-control, sociability, and 'knowing how to hold one's liquor' are matters of manly pride, alcoholism problems are at a minimum, provided no other variables are overriding. On the other hand, in those cultures where alcohol has been but recently introduced and has not become a part of pre-existing institutions, where no prescribed patterns of behaviour exist when 'under the influence,' where alcohol has been used by a dominant group the better to exploit a subject group, and where controls are new, legal, and prohibitionist, superseding traditional social regulation of an activity which previously has been accepted practice, one finds deviant, unacceptable and asocial behaviour, as well as chronic disabling alcoholism. In cultures where ambivalent attitudes toward drinking prevail, the inci-

dence of alcoholism is also high," [7]). With such cultural differences in perspectives towards alcohol, prevalence of addiction to alcohol will also differ between cultures. This same understanding of cultural differences can also be applied to problematic engagement in other behaviours as predicted by culture. However, there are minimal studies to this effect, an area requiring further research, hence part of this thesis. In addition, as we discuss in the next chapter, studies have also shown that negative real life events, lack of social support and loneliness are some of key predictors of behavioural addictions. So, society and environment play a critical role not only in drug addiction but also in behavioural addictions.

From the reviews and deductions made, addiction is a crosscutting phenomenon, thus, it should be explained from different perspectives to present a holistic picture of what it is and its developmental process. Each perspective, separately, has limitations. The biological/medical perspective seems not to consider some phenomenological element of tendencies expressed by addicted persons. Whilst some people take a long road to recovery, often characterised by relapse and withdrawal symptoms, some people seem to find it easy to deal with addiction. Such evidence shows the limit of the biological perspective of addiction.

From this limitation, I note the importance of another perspective of addiction, thus, the socio-cultural perspective. Studies have also shown that addicted persons that receive proper support; are not estranged by family members but are accepted and supported to deal with addiction, find it less hard to recover from addiction than those who lack social and family support. On its own, the social cultural perspective tends to undermine the compulsive power of addiction, its compulsive nature that cannot be resolved by social support alone, but by directed and deliberate therapeutic intervention. For instance in behavioural addictions, people begin to engage in a particular activity for right reasons. However, due to excessive use and psychological factors not related to social factors, some people end up compulsively engaging in the activity. Though in its infancy, recent neuroscience evidence has shown that some people are genetically more vulnerable to addiction than others whilst some are vulnerable as a result of non-social psychological traits.

These studies reveal the complex nature of addiction. The non-social psychological traits that people attain in childhood development also play a critical role in explaining addiction. The interplay of these factors may lead to different results in different people. From the foregoing, it is hence imperative and essential to promote the biopsychosocial perspective of addiction, which will help develop and encourage diverse interventions to deal with the problem by addressing important factors.

1.2 Treating Addiction and Relapse: Biopsychosocial Perspective

As a clinical disorder, addiction requires long-term treatment that should and can only be measured in months and years. Is it recommended to individualize the treatment process of addiction [50]. Further, a complete evaluation is required in order

to trace any co-existing medical, psychiatric and social problem that require redress together with the addiction treatment. Key to successful treatment of addiction is long-term prevention of relapse by pharmacological and behavioural means.

A biopsychosocial model of addiction entails treating addiction from all three fronts, thus, medicinal, psychological and social. It should be noted that addiction treatments vary depending on the form and level of addiction. Traditionally, strategies for preventing relapse have involved counselling and/or psychotherapy. However, more recently, pharmacotherapies and technologies combined with cognitive psychotherapies have been adopted and employed in treating addiction and preventing relapse.

1.2.1 Pharmacological Perspective

Some of the effective medications for treating opiate dependence include buprenorphine (commonly known as Suboxone®), Vivitrol® (extended-release naltrexone), and methadone [71–75]. Each of these three medications has been proven significantly more effective at preventing drug use relapse than a placebo in rigorous, double-blind experimental studies [3, 32, 33, 67].

The types of medication that have been found to be effective when combined with behavioural treatment in preventing relapse (like those stated above) can be classified as agonists, antagonists and anti-craving medications. These medications work through a variety of mechanisms. For instance, methadone is a full agonist and works by activating the opiate receptor, diminishing cravings for opiates and preventing euphoria if the patient abuses opiates [68]. On the other hand Vivitrol® contains extended-release naltrexone, which is a complete mu-receptor antagonist, meaning it completely blocks the mu-receptor. As a result, Vivitrol® prevents an individual from experiencing euphoria if he or she abuses any opiate, helping to prevent relapse [36], whilst Buprenorphine is a partial mu-agonist [72]. It prevents the patient from going into withdrawals or experiencing cravings, while preventing euphoria from any opiate used (including too much buprenorphine).

Aside pharmacological interventions, recently, researchers have explored the use of non-invasive brain stimulation techniques to treat addiction. From a symptomatic approach, it is tempting to think that non-invasive brain stimulation (NIBS) techniques, such as rTMS and transcranial Direct Current Stimulation (tDCS), may be of interest for individuals suffering from IA, as is the case in SUD. Indeed, the PubMed/Medline database contains more than thirty studies on the use of NIBS techniques to treat substance use disorder, including alcohol, tobacco, cocaine, cannabis, and methamphetamine. In most of these studies, brain stimulation seemed to lead to a significant decrease in craving, both in baseline and cue-induced craving, and may have led to an improvement in decision-making by reducing both impulsivity and risk-taking behaviour [16, 17, 21, 22].

1.2.2 Psychological Perspective

There are a number of behavioural interventions that have been found to effectively treat addictions. These interventions mainly involve behavioural therapy including motivation interviewing, contingency management therapy and the most adopted interventions- cognitive behavioral therapy. Motivational Interviewing is a counseling approach used to explore and resolve ambivalence about behavior change. There is a strong evidence base that it reduces substance use problems and a growing evidence base for other problems [30]. It has been defined as "a client-centered, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence" ([47], p. 25). Contingency management provides tangible reinforcers for achieving target behaviors to increase the likelihood of those behaviors reoccurring. Typically, contingency management interventions identify an appropriate target behavior (e.g., abstinence as verified by a negative urine toxicology test) and provide tangible reinforcers each time the target behavior occurs [52]. The reinforcers are most often monetary- based vouchers exchangeable for retail goods and services or the chance to win prizes of varying magnitudes. If the target behavior does not occur, the reinforcers are removed [28, 53].

Cognitive behavioral therapy is an individualized, collaborative approach to psychotherapy that emphasizes the importance of thoughts, feelings, and expectancies and also incorporates more traditional behavioral approaches that utilize counterconditioning and contingency management in addressing the problem of addiction [51]. It combines two very effective kinds of psychotherapy—cognitive therapy and behavioral therapy. Cognitive behavioral therapy is based on a number of theories including, social learning theory, stress theory and coping theory. It underlines that the learning processes play an important role in the development and continuation of addiction as well as reducing and treating addiction. Further, this intervention is cognizant of the view that stressors are likely to trigger addictive behavior as a coping strategy to avoid experiencing distress. As such, cognitive behavioral therapy focuses on challenging individuals' positive expectancies about substance use, enhancing their self-confidence and self-efficacy to resist addictive behavior and tendencies.

Mainly, cognitive behavioral therapy helps clients in two major behavioral ways. The first is to help reduce the intensity and frequency of their urges to use or engage in addictive substance or behavior, by undermining their underlying beliefs or cognitions about the substance or behavior. The second is to teach the clients specific techniques for controlling or managing their urges to use or engage in addictive substance or behavior. Cognitive behavioral therapy has been demonstrated to facilitate effectively improvement for a number of mainstream addictions. Reductions in drinking and drug use were seen mostly when clients were motivated to change and possessed at least a low average intelligence level needed to process and relate thought patterns with behavioral reactions [42]. Treatment gains with respect to stimulant use have been well established, with evidence that gains persist and grow over periods of 6–12 months [10, 56].

1.2.3 Social Perspective

In treating addiction, a biopsychosocial perspective highlights the role of the society/community in prevention of relapse for addicted persons. As highlighted earlier, parents, siblings, friends and the community at large play a critical role in safeguarding treatments of addiction and encouraging addicted persons to stay on course of their recovery. One of the systematic community approaches that has been proposed and found to be effective is the community reinforcement approach therapy. Hunt and Azrin [29] developed this intervention and tested it on persons with alcohol dependence. The community reinforcement approach is based on the theoretical view that individuals use substances or engage in some behavior for their positive, reinforcing effects and that the relative lack of alternative, non-drug and non-addictive reinforcers maintains dependence. The development of alternative reinforcing activities that are incompatible with drug use is therefore central to the community reinforcement approach [52].

In the community reinforcement approach, the therapist places a great deal of emphasis on changing environmental contingencies in the client's life. Employment, recreation, and family systems are all addressed to promote a lifestyle that is more reinforcing than substance use and/or additive behaviour. Rather than being entirely office-based, the community reinforcement approach is typically performed, at least in part, in the community. If clients do not attend treatment or do not follow through with an employment or recreational goal, the therapist may go to their homes, take them to job interviews, or help them try a new recreational activity. The purpose of expanding the treatment beyond the office setting is to increase the positive reinforcing effects of non-substance-using activities by direct exposure.

Studies have found the community reinforcement approach to be of therapeutic benefit to alcohol-dependent individuals [49, 64]. Further, several reviews and meta- analyses have concluded that the community reinforcement approach is an important, established, and effective treatment for alcohol use disorders [19, 48].

1.3 Conclusion

This chapter has presented a biopsychosocial perspective of understanding and treating addiction. It has stressed the role of society and community in treating addiction, underscoring the social nature of addiction. The assumptions, studies and recommendations presented herein are general, thus, covering both substance and behavioural addictions. This is based on the understanding that there are striking similarities between substance and behavioural addictions at the clinical, neurobiological and neurofunctional level [24, 37]. This understanding suggests therefore that interventions in treating addiction such as neurostimulation techniques, which are effective in treating substance addiction, could also be effective in treating similar symptoms in behavioural addictions.

As we conclude, it is important to stress that treatment of addiction is complex and should require a combination of techniques in order to adequately treat it and prevent relapse, hence a biopsychosocial approach to treating addiction. Experimental studies have found that the combination of medication and counselling is more effective than counselling alone at preventing relapse [27, 73]. Also, combining pharmacological and behavioural treatment leads to greater rate of retention than for either counselling or 12-step groups [69, 80]. According to the World Health Organization, the most effective treatment for opiate dependence is medication combined with counselling [70].

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