

Chapter 6

From Nihilism to Hope: Reframing the Concept of Addiction

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Abstract The treatment of substance use disorders (SUDs) has been characterised by a focus on the goal of abstinence, fostering a sense of therapeutic nihilism when relapse occurs. This chapter will focus on an overview of the factors that determine individuals' vulnerability to SUDs, and how it differs between those at high risk and those at lower risk for an SUD. It will also discuss how this conceptual understanding can be used by clinicians to reframe the focus from an abstinence only model (focused solely on halting drug use) to a more nuanced interventional model that combines harm reduction along with interventions that target the underlying risk factors for addictive disorders, with a longer term focus on recovery.

Keywords Addiction · Externalising spectrum disorders · Dependence · High risk · Intervention · Model · Low risk · Substance use disorder · Vulnerability

Abbreviations

ADHD Attention deficit hyperactivity disorder
ATOD Alcohol, tobacco and/or other drugs
CNS Central nervous system
ERP Event-related potential
fMRI Functional magnetic resonance imaging

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HR	High-risk children, i.e. children of persons with dependence as well as two first-degree family members who also have substance dependence
LR	Low-risk children, i.e. children with no family history of substance dependence in their first- and second-degree relatives
TMS	Transcranial magnetic stimulation
VTA	Ventral tegmental area
SUD	Substance use disorder

6.1 Introduction

The treatment of addiction has been an activity in which its practitioners often feel dwarfed by a visceral perception of inevitable failure. The revolving doorway of sobriety and relapse, through which ‘addicts’ seem to pass so very frequently, has stymied client, therapist and concerned caregiver alike, albeit in different ways. When a client is working toward a goal of abstinence, that terminus can seem like a mirage, more illusion than reality. Even when abstinence is achieved, it can seem like a reward forever perched on the edge of an abyss, waiting for something as yet unseen or unheard to come and push it over into the depths of regret and despair. To understand why this process carries such an aura of nihilism, it is worthwhile to understand how the concept of addiction itself has evolved. The earliest treatment models of addiction were based on the conceptualisations of substance use prevalent in the era they arose in, from the moral model with the concept of demon rum to the temperance movement and prohibition. These models brooked no solution less than a complete cessation of consumption. The Alcoholics Anonymous concept of addiction as a lifelong disease, for which there is no cure with the only treatments available to reduce or suppress the urge to use drugs, has the potential to suggest that the disorder is deterministic with limited ability for the user to change other than to adopt faith in a higher power. The conceptualisation of addiction as a chronic relapsing disorder, which has its roots in individual vulnerability and which behaves similarly to other chronic disease processes (McLellan et al. 2000), has begun to suggest paradigm shifts in the approach to, and the treatment of addiction. This biopsychosocial understanding has helped us understand that while addiction (in the early onset subset of those who develop substance use disorders or SUDs) occupies the extreme end of a spectrum of substance use behaviours, harmful use and hazardous use, especially in young users, also require early intervention. Hence, while achieving abstinence from substance use can be a final goal for treatment providers to aspire to, the harm reduction has been found to be a developmentally congruent approach to the primary and secondary prevention of risky behaviour adolescents (Canadian Paediatric Society 2008). This chapter will focus on the theoretical framework for a change in approach, along with its implications in the management of substance use, and particularly substance use in adolescents.

6.2 Vulnerability to Addictive Disorders

While assessing adolescent substance users, it is important to evaluate certain key factors that underlie their vulnerability to develop a more severe or chronic SUD. These factors can be summarised as follows:

- **Family history of substance use disorders:** A high number of first-degree relatives who have substance use disorders can be understood to convey a higher potential to carry genetic markers of risk. However, it is important to keep in mind that this set of risk factors, that is shared by family members, do not reflect a risk to develop substance use disorders alone, but a wider spectrum of risky behaviours. In addition, when there are family members who use alcohol, tobacco or other drugs (ATOD), factors such as easier access to ATOD as well as modelling can also affect attitudes towards substance use and experimentation (social learning theory). Offspring of parents with alcohol-dependent parents also have an earlier onset of use of substances (Obot et al. 2001). For example, a study done in Thrissur, Kerala, by Chopra et al. showed that the prevalence of alcohol use among offspring of alcohol users (10 %) was significantly higher than in the offspring of non-users (3 %) (Chopra et al. 2008).
- **Externalising spectrum disorders, including attention deficit hyperactivity disorder (ADHD):** ADHD, both during childhood as well as adulthood, serves as a significant risk factor to develop more severe SUDs (Ohlmeier et al. 2008). However, it is important to note that the externalising group of disorders include constructs such as antisocial personality disorder, oppositional defiant disorder and conduct disorders, all of which share in common certain endophenotypes underlying this risk but to differing extents. One such externalising trait is sensation-seeking, which can predispose not only to substance use but to risky sexual behaviour as well (Chandra et al. 2003). In addition, the reward pathway (involving the ventral tegmental area (VTA), the nucleus accumbens and the prefrontal cortex) has been noted to show hypofunctioning in externalising spectrum disorders (Volkow et al. 2011).
- **Chronic stress:** Chronic stress is often conceptualised to mean psychosocial stress that persists for an extended period and is severe (e.g., sexual abuse, physical abuse, homelessness, extreme poverty and negative life events such as parental losses, parental conflict, parental separation, etc.). Sufferers tend to have higher rates of initiation of drug use as well as an increased incidence of other psychiatric disorders too (Sinha 2008; Burt et al. 2003; Kessler 2004; Clark et al. 1997). Other types of biological stressors, including poor nutrition and exposure to environmental pollutants such as lead, can also adversely increase the risk of developing future developmental and psychiatric disorders, thereby enhancing the risk for future substance use as well.
- **Co-occurring mental illnesses:** Anxiety disorders (Hulvershorn et al. 2015), mood disorders as well as schizophrenia all serve as diatheses which increase the likelihood of developing or exacerbating a substance use disorder.
- **Easy availability of substances.**

- Early use: The earlier in life, the substance of abuse are used, the more the chance of altered neuro-development and allostatic load (state of chronic reward dysregulation, wherein a substance user's drug-seeking behaviour becomes driven by a need to chase or recapture the initial rewarding effects of the drug) (Wand 2008). Drugs of abuse serve as pharmacological stressors in terms of brain development, altering developmental trajectories at critical periods of brain development, thereby augmenting the risk of a more severe course of substance use.

6.3 Do High-Risk Children and Adolescents Differ from Their 'Lower Risk' Counterparts?

Studies comparing high-risk children (HR—children of persons with dependence as well as two first-degree family members who also have substance dependence) with low-risk children (LR—children with no family history of substance dependence in their first- and second-degree relatives) have shown a number of differences which could mediate future risk for SUDs, even before either group have had their first substance use experience! Some of these differences are

- Structural differences in brain regions: HR subjects not only have significantly smaller volumes of their superior frontal, cingulate and parahippocampal gyri, amygdala, thalamus and cerebellum, but these reduced grey matter volumes correlated negatively with their externalising symptom scores. Trends toward a reduction of these differences with increasing age suggest a neuro-developmental lag in HR subjects (Benegal et al. 2007). Adolescents with SUDs show significantly greater psychological dysregulation along with prefrontal and parietal white matter disorganisation when compared to controls (Chung et al. 2013; Clark et al. 2012). *In addition, one imaging study done as part of a thesis in NIMHANS has revealed differences in the pattern of white matter tract myelination between HR and LR subjects* (Subbu et al. 2010).
- Electrophysiological differences: In terms of the externalising syndrome, a heritable disinhibitory complex, characterised by generalised increased brain hyperexcitability, is thought to underlie the disorder (Begleiter and Porjesz 1999). In a comparison done by Benegal et al. and Silva et al. between HR and LR children, the P300 component of event-related potential (ERP), a measure of the way the brain pays attention and discriminates between potentially important and non-important stimuli, shows reduced amplitude and this is associated with externalising psychopathology (Benegal et al. 1995; Silva et al. 2007). A difference in terms of the absence of the cortical silent period immediately following transcranial magnetic stimulation (TMS) of the brain, in HR subjects, which indicated inefficient inhibitory capacities of the brain resulting in a state of central nervous system (CNS) hyperexcitability (which strongly predict externalising symptoms) were found by Muralidharan et al. (2013) Hence, a

large number of brain endophenotypes/markers of brain development appear to indicate the presence of a brain development delay—associated with the manifestation of a spectrum of externalising behaviours: Poor attention allocation, impulsivity, difficulty in learning from mistakes, novelty seeking, oppositionality and conduct symptoms.

- **Cerebral Activation patterns:** Functional magnetic resonance imaging (fMRI) has shown altered patterns of brain activation to go/no-go tasks, though there are contrasting results in terms of whether there is increased or decreased activation in the frontal cortex and other cortical areas (Clark et al. 2013). On the anti-saccade task, a behavioural inhibition task that may be presented during fMRI, HR children made more anti-saccade task errors than did controls; and in the group of HR children, those with ADHD made more errors than those without ADHD (Habeych et al. 2006). On affect-laden tasks, HR children had greater amygdalar activation in response to seeing affect-laden faces than controls, indicating greater frontolimbic network hyper responsiveness to such stimuli (Thatcher et al. 2014).
- **Altered CNS responsiveness to substance use:** While HR subjects, after ingestion of alcohol, show greater CNS stimulation (a faster onset and longer duration of a switch from beta to alpha activity) and a greater reinforcing effect of alcohol (Jagadeesh et al. 1999), they can have low subjective awareness of intoxicating effects. Moreover, this is a genetically influenced characteristic that impacts on the alcoholism risk of HR subjects (Schuckit 2009).

This difference in the maturity, and hence the functioning, of brain circuits which mediate top-down patterns of information processing versus those that mediate the salience incentive and emotions (Casey et al. 2008). A confluence of mutually interacting environmental influences and genetic factors modulates this risk over time, resulting in a delay in brain maturation. Once again, it is important to understand that HR children carry a greater risk or propensity for certain behavioural characteristics when compared to LR children, and that these characteristics can predispose them to many types of risky behaviours, one of which is substance use and/or abuse.

6.4 What Does This Mean for Clinical Practice?

For illustration, let us take the case of Ms. X, 16-year girl, who was brought straight from a rehab centre (from where she has been going her class X exams) directly to the outpatient department (OPD), with her parents being very concerned about safety and escape from custody. X was being treated for multiple substance use disorders fulfilling criteria for dependence (Inhalants, cannabis, tobacco and alcohol) and severe emotional reactivity, with violence to self and others. She had been diagnosed as Borderline PD and had already had multiple admissions in rehabilitation and psychiatric nursing homes. X has also had early

sexual experimentation with multiple medical terminations of pregnancies. She is currently on a combination of two antipsychotics with Carbamazepine and has been on long-term psychotherapy. The dominant theme X brings up during interviews is her reaction to her father's affair and his temporary abandonment of the family, and she does not see herself stopping alcohol and cannabis use at present. Her parents are seeking long-term confinement and care, as they believe that this is the only possible solution, while X is adamant that she 'will not go to rehab'. All3 of them are tired of the way things have been going...

It is obvious that HR children would seem to require a set of interventions, of which those directed specifically towards substance abuse would form a subset. The other components include interventions targeted at

- the early identification of both substance use disorders as well as comorbidities
- the amelioration of externalising disorders (with an armoury of both pharmacological and psychosocial methods)
- treating psychiatric comorbidities (such as anxiety, schizophrenia and mood disorders)
- life skills training
- stress management
- family interventions
- facilitating access to resources that further education and facilitate vocational skill building
- promoting healthy behaviours and/or positive addictions such as exercise and hobbies
- Parent Management Training
- making contingency plans and learning how to deal with lapse and/or relapse of substance use
- maintaining follow-up and aftercare.

In essence, adolescents with substance use who have been brought to treatment have a variety of needs that the clinician is able to help them with, many of which they may be more motivated to address than substance use. For example, a client is more likely to agree to help with an inability to finish projects or tasks because of ADHD, than address their use of cannabis which they perceive as normative. As we have seen that it has been suggested that ADHD shares underlying risk factors for SUD.

This refocusing from the initial problem on every caregiver's and therapist's mind, i.e. substance use, which the client may not be willing to address immediately or even acknowledge, to a vulnerability factor which the client acknowledges as problematic *in other dimensions of well-being* allows both the reluctant client and the eager therapist to set a common goal for treatment. Addressing the vulnerability factor helps to improve quality of life and drive change. It also facilitates a longer term engagement of the client in treatment, allowing time to build rapport and perhaps reconsider, eventually, their desire to continue substance use. While abstinence is a worthy final goal, this approach affords the therapist the ability to reduce ongoing harms from a number of risky behaviours and enhance quality of

life. It allows clients to build the skills and the resilience factors which could help them reduce drug use as well as maintain this reduction or abstinence over time.

Hence, this approach of *reframing* the problem of substance use as a single aspect of a larger, overarching problem (i.e. vulnerability) can be a powerful way of engaging even reluctant clients and facilitating incremental change. In addition, it allows us to educate client and families about their vulnerability better. It allows parents and adolescents to set common goals for change, even if substance use is something they continue to disagree on. As a significant number of adolescents may not have developed a severe SUD but still have harmful or hazardous use, it allows a refocusing of priorities on underlying vulnerability factors and enhancing of the adolescent's quality of life. Let us see how this helped in Ms. X.

X is an overweight teenager with over-sexualised clothing and appearance. She is angry and blames her father and mother for escalating their interpersonal problems into a need for admission. She is tired of therapists and believes she knows exactly what every clinician is going to say. However, motivational interviewing, you are able to understand and reflect back to her that she truly feels sad about dreams and goals that now seem unrealizable to her. On the Adult ADHD self-rating scale (ASRS), she has high scores. When these concerns are amplified by questions about impulsivity and poor attention allocation, both she and her parents are concerned about her inability to achieve hopes and goals despite her high intelligence. She does not seem to have had a mood disorder.

Her difficulties in achieving her dreams and fulfilling her potential are what you reframe as the central tragedy of her story. This was something that she and her parents readily concur with and want to work on. You illustrate her ADHD's contribution to this by comparing her brain to a car, with her frontal cortex acting as the brakes (allowing her to regulate her behaviour) and her anterior cingulate acting as her gears (allowing her to change her behaviours in response to new situations). You describe her limbic system and the reward pathway in her brain as providing the drive to perform tasks that are important to her, or her engine. She is able to see that impulsivity and an impaired ability to finish tasks are barriers to her goals. Drug use has served as self-medication. X agrees to work on "tuning brakes and gears" while placing reduction of drug use as a "why use kerosene as fuel instead of petrol and ruin your car" argument.

Once agreement is achieved, treatment plans need to be made. Psychosocial interventions that could be used range from individual therapy to family therapy to community-based programmes. In terms of psychological programmes, in addition to CBT, dialectic behaviour therapy and interpersonal therapy have also been reported to show promising outcomes. Psychological interventions could have a range of components that target anxiety, mood disorders such as bipolar disorder and depression, eating disorders, disruptive and oppositional behaviours, self-harming or self-injurious behaviours, poor assertiveness, anger and aggressive behaviours, major psychiatric disorders such as schizophrenia along with substance use disorders (Ollendick and King 2004). Crucially, the intervention should focus on building confidence between adolescent and care-givers, planning for lapses/relapses and developing contingency plans, agreeing on follow-up in both

routine and emergency situations and building positive hobbies and activities that are rewarding to the adolescent. These 'positive addictions' can help reduce the need to self-medicate with drugs and boost self-efficacy and esteem.

Comorbidities once identified need to be treated, both with psychosocial as well as pharmacological interventions based on need. One group that we have discussed is those who have externalising disorders. The pharmacological treatment for ADHD has been shown to not increase the risk for subsequent SUDs, as well as show protective effects from developing SUDs (Nigg 2013). While the literature has shown varying effects in terms of the effect of treating ADHD on comorbid SUD outcomes, a study by Benegal et al. in 262 males showed significant externalising symptom reduction, longer abstinence, shorter turnaround time and better quality of life while on atomoxetine (Benegal et al. 2013).

In terms of pharmacotherapy for severe substance use disorders in young adults, some promising new agents which can serve as agents that modify the allostasis, or altered hedonic tone caused by prolonged substance use are available. In terms of alcohol use, baclofen, topiramate and naltrexone have been used in early onset disorders. Case reports of baclofen in the treatment of solvent use have also been reported (Muralidharan et al. 2008). The glutamatergic agent, N-acetylcysteine, has been shown to increase the odds of reduction of cannabis use, as well as having some promise in other SUDs such as cocaine use and nicotine use (McClure et al. 2014). Opioid treatment guidelines now allow the use of opioid substitution therapy in adolescents when indicated. However, the use of these anti-craving agents as well as substitution therapy in adolescents needs to be evaluated much further. A proposed treatment paradigm is shown below in Fig. 6.1.

Ms. X is at first struck by the fact that she is not being labelled as a drug addict. She recognises that the formulation of persons with difficulties in sustaining interest and motivation—describes her well. She says that she also despairs of reaching her goals and fulfilling her desires, as she is never able to finish a task. Despite being clever and more creative, she lags behind her other friends and her repeated failures have made her despair of ever being good enough. And Yes! She would very much like to change that aspect of her if there is a solution. Perhaps even reduce her use of drugs! She is taken up by the concept of tuning her car/brain, now that she no longer has to defend herself of charges of being a drug addict and a bad person...

Ms. X was started on methylphenidate for the ADHD and initially baclofen for anti-craving. She received elements of Dialectical Behaviour Therapy and specific tips on handling problems with studying, emotional reactivity, craving and relapse. We intended to get the family into structured Family therapy but were never able to as they lived in another part of the country and could only come back for brief follow-up visits.

The girl went back to school and completed her class 10 and class 12 exams successfully. Briefly she was troubled by teachers who she felt, continued to scapegoat her, but subsequently she got into an undergraduate course in London, and is studying successfully, living alone. She continues to drink along with her friends at the pub on weekends, but has given up smoking cannabis. Importantly, she is happy on the whole and her parents are very proud of her achievements.

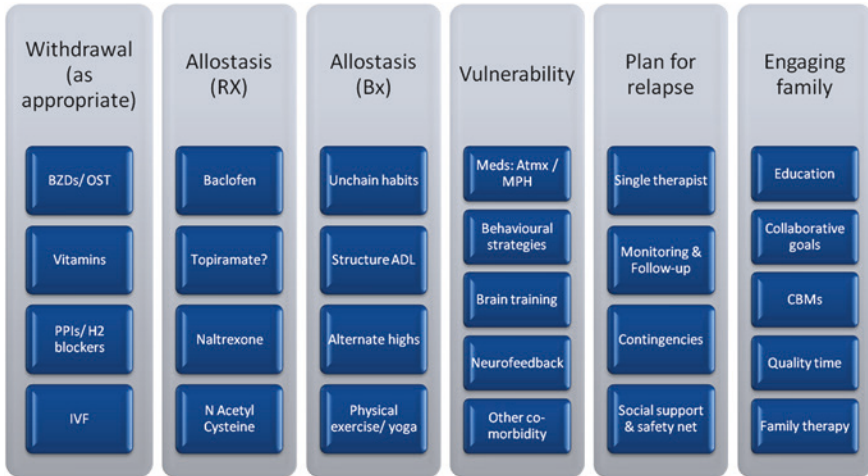


Fig. 6.1 Proposed treatment paradigm for early onset substance use disorders. *BZDs* Benzodiazepines; *OST* Opioid substitution therapy; *PPI* Proton pump inhibitors; *IVF* IV fluids; *ADL* Activities of daily living; *Atmx* Atomoxetine; *MPH* Methylphenidate; *CBM* Confidence building measures

In conclusion, we can see that a modified approach to substance use disorders in adolescents that focuses on addressing the vulnerability factors and comorbidities that have a direct bearing on SUDs has the potential to help clients with early substance use but not a disorder as well as clients with more severe SUDs. Therapists can use reframing to educate families as well as overcome resistance to therapeutic engagement. This approach allows the adolescent as well as her/his parents to set common goals and emphasise long-term and more holistic outcomes (e.g. quality of life) as their goals. It allows the therapist and the client to incorporate harm reduction measures and plan for contingencies, while not allowing relapses to derail treatment plans. Admissions should be considered when necessary, but long-term admissions which do not address the factors underlying substance use are unlikely to be effective.

It is important to understand that the risks that underlie addictions are also risk factors for other psychiatric disorders as well. Early identification, and interventions, for these very risks would allow us to help the young people who most need help, at the developmental period during which they need it most. Hence, a significant amount of research remains to be done on the factors, both environmental and individual, that crystallise the translation of these vulnerabilities into disorders. Some of these answers will come from cohort studies following children at risk. This long-term approach to addictive disorders as epiphenomena of underlying difficulties promises to be a more hopeful one to all stake-holders involved.

Learning outcomes

After reading this chapter, readers would understand that:

- Addictive disorders arise from a host of vulnerability factors, both individual and environmental.
- These vulnerability factors not only determine the risk for addiction, but to a significant number of psychiatric comorbidities, and can have a deleterious impact on an individual's quality of life.
- High-risk individuals show differences from those with low risk in various ways (including structural, electrophysiological and neurochemical alterations; cerebral activation patterns; altered CNS responsiveness to substance use, etc.), even before they develop an SUD.
- Treatment plans do need to incorporate harm reduction strategies for substance use, but they must target underlying risk factors with a combination of pharmacological and psychosocial interventions.
- Reframing the SUD as a problem arising from underlying factors which affect other aspects of a person's life helps generate shared treatment goals, and enhances the client's investment, as well as engagement, in treatment.
- Early identification of those young people at high risk, and the optimal timing of interventions that ameliorate the risk of developing SUDs, are important thrust areas for research.

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