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The Psychology of Addiction: A Literature Review

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According to the Canadian Mental Health Association (CMHA); one in ten Canadians over the age of 15 report symptoms of addiction or substance dependence.[1] Addiction is defined as a chronically relapsing disorder characterized by a compulsive dependence on a substance or behaviour, the loss of control of limiting intake, and negative emotional symptoms, such as anxiety and irritability, when the substance is removed.[2]

The most common substance dependency is alcohol, followed sequentially by *Cannabis sativa*, prescription painkillers, nicotine, and illegal drugs.[1] In addition to drug abuse, other common addictive behaviours include, exercise addiction, binge eating and food addiction, compulsive buying, sex addiction, obsessive tanning, computer addiction, self injury and gambling.

The existence of an "addictive personality", first introduced by VanKamm in 1965,[3] has been debated and discussed by numerous researchers.[4] Their consensus is that there is no definitive "addictive personality"; however, there are certain traits or risk factors, which predisposes one to addictive behaviours. Addiction is a multi-factorial syndrome, resultant from a combination of genetic, psychological, and environmental factors ultimately creating neurobiological impairment and dysfunction.

Addiction Risk Factors

Biological or genetic factors

The estimated heritability of substance dependence is approximately 50%, where the impact of genetic factors tends to increase in adolescence.[5] Linkage and association-based genome-wide studies have identified a number of chromosomal regions containing variants that confer susceptibility to substance dependency, most commonly variation in the mid-region of human chromosome 7.[6] Population-based transmission studies suggest common genetic factors contribute to substance dependency equally in both genders. Genetic variations predominately affect dopamine levels in the mesocorticolimbic brain reward pathway.[6]

The mesocorticolimbic pathway is a complex neural pathway essential in higher order cognition. It consists of dopamine-producing cells that originate in the ventral tegmental area of the midbrain, which project to various forebrain regions including the nucleus accumbens (NAcc), medial prefrontal cortex (mPFC) and amygdala. This highly conserved neural circuit is thought to play a critical role in the assignment of motivational value to biologically relevant stimuli, resulting in the production of adaptive behaviors.

Polymorphic variations of dopamine receptors lead to increased dopamine signalling in the ventral striatum, where aggregates of multiple dopamine receptor single nucleotide polymorphisms (SNPs) are associated with addictive behaviour. In addition to genetic variability in dopamine systems, gene studies have also linked genetic modification of opioid and GABAergic systems in addiction.[4] These findings suggest that the genetic factors involved in addiction are cumulative, where an individual with greater genetic variation will have a higher chance of developing addictive behaviours.[4,7,8]

Genetic predisposition to addictive behaviour has been demonstrated in family-based studies - including family/siblings, adoption and twin studies. Bierut *et al.* reported elevated rates of alcohol dependence, compared to control, between siblings, 50% for men and 25% for women.[9] Similar trends were noted for cannabis, cocaine, and nicotine dependence.[6] Merikangas *et al.* reported an eightfold increased risk of drug dependent disorders among biological relatives.[10] Similarly, studies have reported that alcoholism is familial and having an alcoholic parent is associated with a fivefold increase in the risk of alcoholism.[11,12] Rates of substance dependence was found to be significantly higher in adoptive children with biological parents with addictive behaviours, providing support for a genetic component.[13] Twin studies also show genetic susceptibility to addiction, estimating the heritability of alcohol and nicotine dependency around 50-70%, and, cannabis dependency around 34-78%, respectively.[14]

Environmental Factors

Both genetic and environmental factors are co-involved when evaluating the probability whether an individual develops an addiction. Most studies find a greater environmental role on the initiation of substance use and greater genetic component in heavier substance use.[13-15] Adolescents are primarily influenced by their peer

group; hence, environment and social pressures are a dominant factor in determining substance dependency. *Cannabis sativa* is the most abused substance in adolescents, arguably because of peer pressure to maintain a certain image.[16] Availability and cost of substances also influence dependency. Alcohol is the most readably available and affordable substance, hence it is the most abused substance in all ages. Other important environmental influences in the development of substance dependency, revealed through adoption studies, which are able to isolate the influence of environmental exposures from potential genetic confounds, are parental divorce and parental psychiatric disorder.[17]

Social experiences and the quantity and quality of social attachments and interactions during early development and throughout life greatly influence the susceptibility to addiction. Another important environmental factor, maternal neglect or childhood trauma, is correlated to addictive behaviours later in life.[18] *In vivo* animal studies have shown neurotransmitter system modification within one week of maternal separation. When animals were exposed to prenatal stress they exhibited increased risk and characteristics of addiction.[18] Studies of drug addicts have repeatedly shown high percentages of childhood trauma (sexual, physical, emotional or abandonment) where for each traumatic event the initiation of substance abuse increased two to four fold, and seven to ten fold for more than five traumatic events.[19] Traumatic events alter neurobiological stress mechanisms in the child's brain making them hypersensitive to stress and more susceptible to substance abuse.

Psychological factors

There are a multitude of psychometric scales measuring significant psychological traits to predict addiction predisposition. The most accurate and widely used scale is the Addiction Scale of the Personality Inventory by Eysenck.[20] Personality is comprised of the following dimensions: extroversion/introversion, neuroticism/stability, and psychoticism/socialisation.

Psychoticism is one of three traits used by the psychologist Hans Eysenck in his psychoticism, extraversion and neuroticism (P-E-N) model of personality. Psychoticism refers to a personality pattern typified by aggressiveness and interpersonal hostility.

Addiction is correlated with psychoticism, and the specific Addiction Scale measures levels of emotional reactivity, proneness to stress, impulsivity, and negative effect. Elevated levels on the Addiction Scale are positively correlated with predisposition to addictive behaviours.[21] Psychotic characteristic traits seen in addiction are aggression, coldness, egocentricity, impersonalizing, impulsivity, antisocialism, no empathy, creativity, and tough-minded.[22]

In addition to common psychological traits, there is a distorted cognitive belief system shared by addicts.[19] This belief system involves polarized "all or none" irrational thoughts and negative self talk (such as "I am not good enough"). This distorted belief system also dominates in individuals with depression and poor self esteem, and may be the causal link between the high occurrence of depression together with substance abuse.

Neurobiology of Addiction

Genetic susceptibility and environmental cues augment an individual's neural circuitry by down regulating specific receptors and/or neurotransmitters, leading to increased occurrence of addictive behaviours and substance abuse. Specific neurotransmitter and/or receptor deficiencies lead to dependency on specific substances. The neurobiological systems commonly altercated in addiction include the dopamine and opioid circuits, the limbic or emotional brain, and the stress apparatus and impulse control cortical area. As the addiction continues, the brain undergoes further modification and neuroadaptive changes, further increasing substance dependency through positive reinforcement.[23]

The dopamine and opioid circuits

Dopamine is a neurotransmitter inside the brain which regulates motor control, motivation, arousal, cognition and reward. The brain reward cascade starts in the hypothalamus (the mesolimbic system) where serotonin activates endorphins.

Endorphins are released in the hypothalamus stimulating mu receptors in the substania nigra that contains gamma-aminobutyric acid (GABA). $GABA_B$ receptors project onto the ventral tegmental area (VTA) brain region to activate dopaminergic neurons to release dopamine at the nucleus accumbens (reward site of brain).[24,25] Substances that increase dopamine levels will increase the feeling of reward, hence leading to addiction.

Most studies have show that reduced dopamine levels are a predisposing factor for addiction.[25] Genetic studies have shown that polymorphisms in the alleles of dopaminergic receptors, subsequently leading to their down regulation and decreased dopamine levels, are a genetic risk factor in addiction.[7] Also, animal studies have shown that when separated/abandoned by their mothers, rats have reduced dopamine receptors/levels and increased substance dependency, and monkeys have reduced levels of serotonin and increased aggression and tendency to consume alcohol.[26,27]

However, some studies argue that heightened dopamine function is responsible for addictive behaviours. Their reasoning being that low dopamine levels in Parkinson's Disease are associated with low incidence of addictive behaviours; whereas, when medicated with dopamine they develop compulsive and addictive behaviours.[28] Also, there is a positive correlation with childhood trauma, a risk factor in addiction, and seizure activity caused by increased dopamine levels.[29]

All abusive substances in some way exert their rewarding effects by augmenting dopamine levels in the nucleus accumbens. The specific pathway modulated depends on the substance abused. Opioid

neurotransmission, affected by heroin, is crucial for signalling in the neural reward circuitry, modulating acute and chronic responses to other abusive substances.[30] Increased GABA neurotransmission, affected by alcohol, is the main inhibitory neurotransmitter and synergistically affects the rewarding effects of other substances.[31] Increased nicotine neurotransmission synergistically increases the levels of dopamine, norepinephrine and serotonin by decreasing monoamine oxidase activity, leading to the euphoria and relaxation.[32]

Limbic Brain

In addition to the mesocorticolimbic dopamine system and its projections, specific components of the basal forebrain and amygdala have recently been identified with neuroadaptations to acute drug reward. Ventral corticolimbic control pathways incorporate fast associative learning that is adaptive in low-predictable environments, suggesting an innate tendency to urgently react or give attention to novel addictive stimuli. This physiologically explains why impulsivity is an addictive personality trait. The reward response is the default mechanism in the limbic brain and needs to be actively retrained in order to control addictive tendencies.

Another major contributing factor to the development and maintenance of addiction is the ability of substances to disrupt the neurobiological learning and cellular plasticity, exerting long lasting influences on behaviour. Brain regions are often involved either directly or indirectly. Directly through the prefrontal cortex and hippocampus which form long-term and episodic memories. Indirectly through the amygdala, involved with emotional modulation of memories, and striatum, involved in reward-based memories and decision making in declarative and episodic memory processes.[33,34] The ability of drugs to alter this system may be one factor why they can exert such a strong control on behaviour, becoming the central focus of an addict.[35] Numerous studies have shown smoking addiction to remain prevalent in addicts because of the effect and control of addictive behaviours on memory. Additionally, smoking is a habitual social activity and hence involves negative modification of oxytocin-dopamine interaction.[32]

Oxytocin increases resilience against addiction by facilitating the processing of social and attachment-related information and its consolidation in internal working models, thus promoting a shift from novelty seeking towards preference of social familiarity through the cortical route.[36] Oxytocin is a neurohypophysial hormone released during and after childbirth which facilitates contractions, stimulates lactation, and promotes maternal bonding. Recent studies have shown the role of oxytocin in social recognition and bonding.[36] Oxytocin and dopamine work synergistically, where increases in level of oxytocin from social attachment and love increases expression of dopaminergic neurons and consequently positively affecting neural plasticity and memory.[29] Animal studies have shown that maternal abandonment permanently decreases the production of oxytocin, leading to impaired social skills, anxiety, aggression, trust issues, and increased substance abuse.[29] Recent studies indicate that social attachments protect against addiction and health consequences of stress; whereas, drug abuse and chronic stress can undermine social attachments.[36] Therefore, a healthy social support network is crucial for modifying addictive behaviours.

Stress response mechanisms

Stress is a real or perceived threat, ultimately activating a "fight or flight" response. This response results from the activation of the autonomic sympathetic nervous systems and the hypothalamic-pituitary-adrenal (HPA) axis. Activation of the HPA axis is characterized by pupil dilation, increased heart rate, bronchodilation, and increased sweat. Infants have no ability to regulate their own stress apparatus, hence a child is completely dependant on the relationship with their parents to regulate and dissipate their stress. Maternal deprivation in times of stress will result in permanent high levels of cortisol, which damages the midbrain dopamine system and shrinks the hippocampus, inhibiting memory and emotional processing.[37]

Cortisol is a steroid hormone produced in the adrenal cortex secreted in response to stress, primarily to increase blood sugar and aid in metabolism.

Early stress (such as childhood trauma and/or abandonment) establishes a low set point for a child's internal stress system, leading them to become hypersensitive and overactive to stressful situations, exponentially so when trauma is experienced in adulthood. A brain pre-set to be hypersensitive to stress may actively seek out short-term relief from stress, turning to addictive substances as a form of stress relief. Addiction is an ingrained and learned response to stress, where abusive substances are used to numb or "cope with" stress. However, it should be noted that childhood trauma is not the only determinate of addiction and not all people with childhood trauma will turn to addiction.

Additionally, stress is an influential factor in continued drug use and relapsing. Exposure to stress is the most powerful and reliable experimental manipulation used to induce reinstatement of alcohol or drug use.[38] Stress renders one vulnerable and susceptible to self-medicate or relapse. Animal studies have shown that physical and psychosocial stressors evoked a relapse to habitual addictive behaviours, causing neural atrophy and increased dopamine transmission.[19] Cohort studies have shown that chronic administration of abusive substances deregulates both the hypothalamic-pituitary-adrenal (HPA) axis and brain stress response mechanisms.[39]

Considerations for practitioners treating addiction

Self Disclosure and Childhood Experiences

Many addicts are reluctant to share their life histories with practitioners, requiring trust to be first established.[40] Individuals may be unaware of the link between their childhood experiences and their addictive behaviours, and others will have no memory of trauma. The memory may be purposely undisclosed, or the memory

may be repressed as a defence mechanism to deal with pain and emotion. Acknowledging and working through childhood traumas are necessary for healing and should be addressed as they are an integral part of the addiction psychology.

Withdrawl, Relapse, and Stress Coping Mechanisms

When treating addiction, withdrawl from substance dependency and occasional relapsing are common occurrences. Human studies have shown that the presentation of drugs themselves, or the stimuli previously associated with their drug delivery, or the state of stress, increased the likelihood of relapse as well as self-reports of craving and motivation to engage in drug-taking.[2] Therefore, it is important for the addict to remove all temptation of substances, including replacing their social network with healthy and supportive social interaction. Taking away an addict's abusive substance is like taking away their "stress coping mechanism". This is why perceived stress is a large determinant of an addict relapsing. Before withdrawing the dependant substance, a healthier mechanism of coping with stress needs to be adopted and practiced. Stress coping therapeutic tools included guided meditation, cognitive behavioural therapy, art/music therapy, social interaction, reading, and exercise.

Technology and Adolescence

Substance dependence peaks in young adults aged 18-25 years-old, and has doubled in youths aged 15-24 years-old during the past five years.[1] Young adults are the most susceptible age group to environmental cues such as peer pressure and parental conflict, hence, prevention and education are key. Part of a health practitioner's role is to educate parents on implementing healthy stress coping skills, and fostering healthy self-esteem to decrease the likelihood of their children developing long-term addictions.

Technological addictions are becoming more prevalent in adolescents. Common technological addictions include use of the Internet, video games, cell phones, TV and social media. Adolescents with Internet addiction, seen in approximately 10% of elementary students, have higher rates of depressive mood, subjective stress, suicidal ideation, anxiety, and other mental health issues.[41] Social network site addiction is associated with health, academic, and interpersonal issues such as depression, anxiety and poor self esteem.[42] Cell-phone addiction is most prominent in females aged 7-13 years-old with low self esteem, and is often associated with depression.[43] Smart phones combine phone technology with Internet, video games, and texting, where the user can access these services at anytime, from anywhere, making smart phones highly addictive. Since the average age a child acquires a cell phone is 7 years-old, preventative measures need to be instated early.[44] Educating parents on establishing healthy boundaries for their children surrounding technology usage and encouraging social interaction at a young age will be protective against addiction.

Summary

Addiction does not have a concrete "personality"; it is a multifactorial syndrome influenced by quantitative genetics, environmental influences, psychotic personality characteristics and childhood trauma. These factors combine to influence and modify the neurochemistry and circuitry of an individual, leaving them susceptible to addictive behaviours and substance abuse. Neurobiological modification includes changes in dopamine levels and the reward circuitry, changes in the limbic brain, and dysfunction of stress response mechanisms. Although these factors predispose an individual to addiction, they do not guarantee that addiction will absolutely occur. In light of predisposition, addiction is a learned and reinforced behaviour which can be prevented and avoided if proper stress coping skills and healthy self esteem are implemented early enough in development.

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