A neuroscientific approach to nicotine addiction

Despite the health risks, nicotine addiction is still prominent today. While smokers are aware of these risks, they continue to engage in the practice of smoking. The purpose of this essay is to identify the potential causes of nicotine addiction, as well as an exploration of the current literature related to nicotine cessation methods. With research, it was found that the determinant of addiction to nicotine involves an interplay of genetics, social and environmental factors, pharmacology as well as learned or conditioned behaviours (Benowitz 2010).

In terms of genetics, personality can play a role, as ‘ addictive personality’ has been implicated in nicotine addiction. Addictive personality can be described as when an individual is significantly more likely to develop addictive behaviours compared to regular people. However, there is a great debate as to whether such a thing even exists, many believe that other factors play a role in so called ‘ addictive personalities’ such as undiagnosed or untreated disorders, for example, Attention Deficit Hyperactivity Disorder, Obsessive Compulsive Disorders, Bipolar, Depression, or Anxiety. This is because people with these conditions are known to suffer things like impulsivity and distractibility or exhibit addictive behaviours as a means of self-medication which can therefore lead to addictive behavioural choices (Gwinnel & Adamec, 2016). The environmental factors of addiction cannot be ignored. Children who are exposed to parents who are heavy smokers are more likely to develop nicotine addictions compared to those who have not (Benowitz, 2010). Majority of smokers (80%) begin smoking by 18 years of age (Bonnie & Lynch 1994). Benowitz (2010), suggests that an adolescent may start to experiment with smoking because of parental or peer pressure

or behavioural problems, which can lead to additional stress. Benowitz (2010), elaborated that genetic influences are crucial in developing addiction. Studies exploring the developing brain in animals suggest that early exposure to nicotine induces permanent changes that leads to being more susceptible to addiction in adulthood (Benowitz, 2010).

Nicotine is a highly toxic liquid contained in the leaves of numerous plants and is typically found and used in tobacco products such as cigarettes, cigars, and chew (Craighead, & Nemeroff, 2004). When inhaled, nicotine as well as 4000 other chemicals are delivered to the nose, mouth, and lungs, which results in an assimilation through cell membranes into the pulmonary capillary blood flow (Grunberg, 1999, as mentioned in Benowitz (2010). After inhalation, the nicotine is transferred to the brain within 10 seconds from the first puff (National Institute on Drug Abuse [NIDA] 1998 as mentioned in Benowitz (2010). Cigarette smoking and addiction can be caused by many things, but another contributor is the distinctive gustatory and olfactory stimulation that is involved. The sense of taste depends on the sense of smell, these chemical senses are grouped together because they both need a chemical stimulus, they are known to interpret the environment by discriminating between different chemicals (Gazzaniga, Ivry, & Mangun, 2014) Sensory cues associated with nicotine delivery have a critical role in nicotine addiction (Palmatier et al, 2007). With this, the manipulation of smoking materials that effects the taste, smell or sensation in the throat further reinforces a pleasurable effect.

Nicotine affects the mesolimbic system, or the pleasure centre of the brain, creating increased levels of dopamine, a neurotransmitter essential to the

functioning of the central nervous system and emotion regulation (Benowitz, 2010, P. 2295). An increase of dopamine elicits induces pleasure and reduces stress and anxiety, smokers use this chemical stimulus to modulate levels of arousal and to ultimately, control mood. Benowitz (2010) states that once nicotine enters the arterial circulation and moves throughout to the brain, it binds to nicotinic cholinergic receptors which are the ligand-gated ion channels which normally bind acetylcholine. Stimulation of these receptors release a variety of neurotransmitters, which signals pleasurable experiences and is critical for the reinforcing effects of nicotine. Nicotine induces glutamate release, which facilitates the release of dopamine, as well as GABA, which is known to inhibit dopamine release. With long-term exposure to nicotine, nicotinic cholinergic receptors become desensitized. The GABA-mediated inhibitory tone reduces while glutamate-mediated excitation occurs thereby, excitation of dopamine neurons consequently heightens responsiveness to nicotine in the brain (Benowitz, 2010). In nicotine dependant rats, experimentally induced lesions in dopamine releasing neurons prevented the self-administration of nicotine (Benowitz, 2010). The causes of cravings (induced by smoking cues, stressors, or a desire to relieve withdrawal symptoms) triggers the physical act of smoking (Benowitz, 2010). When nicotine is delivered to the brain, the nicotinic cholinergic receptors are activated, inducing the development of new neural circuits (Benowitz, 2010). With the association of environmental cues, and behavioural conditioning the receptors ultimately become desensitized, forming a tolerance which leads to short-term reduced satisfaction from smoking (Benowitz, 2010). During the time since last smoking, nicotine levels decline leading to craving and the associated withdrawal symptoms.

Benowitz (2010) suggests, that smoking improves concentration, reaction time, and performance in certain tasks, which may be correlated to relief from the symptoms of withdrawal. These symptoms include irritability, depressed mood, restlessness, anxiety, and drowsiness. In the absence of nicotine, the receptors regain their sensitivity and will only then become reactivated in response to a new dose, which is why attempts to quit are often unsuccessful (Benowitz, 2010).

Another aspect worth discussion is that of the combination of positive reinforcements of nicotine addiction, or enhancement of mood and avoidance of withdrawal symptoms (Benowitz, 2010). It can be said that conditioning plays a major role in the development of nicotine addiction. Smokers develop social related cues for many specific situations, moods or environments. If a person is trying to quit, these cues can trigger relapse (Benowitz, 2010). The urge to resume is persistent and known to occur long after the initial withdrawal symptoms settle. The association between cues for example, seeing someone spark a lighter and the anticipated effects of nicotine, results in the urge to smoke, which constitutes a form of conditioning (Benowitz, 2010). Nicotine enhances the behavioural response to conditioned stimuli, which may contribute to compulsive and excessive smoking. Due to conditioning, the desire to smoke is maintained because smokers usually follow a routine, which can represent powerful cues. To further reinforce the aspect of conditioning, many smokers will find that when they are feeling unpleasantness such as anger or are irritable, a cigarette will provide the desired relief. Yalachkov, & Naumer (2011, p. 3) relay the importance of understanding how “ motivation, memory and

executive control processes interact with drug-associated cues that trigger drug seeking behaviour.”

Benowitz (2010, P. 2299) suggests that nicotine increases activity in the prefrontal cortex, thalamus, and visual system, which reflects activation of corticobasal ganglia–thalamic brain circuits (which is part of the reward network). Furthermore, Wagner et al. (2011) offered an interesting perspective on the issue of action-related brain regions for drug cue activity. Because smoking is comprised of a manual action component and many smokers engage in cigarette smoking in an automatized manner, it was said that the brain regions involved are the anterior intraparietal sulcus (aIPS) and the inferior frontal gyrus (IFG), which are responsible for the planning, initiating, and imitating of manual actions. Wagner et al (2011) looked at the effects of environmental cues. Participants in the study were required to watch a movie, in which the characters were seen smoking. Through the use of fMRI, the blood-oxygen-level-dependent signal was measured while the participants watched movies containing smoking and neutral scenes. The participants were unaware of what data was to be collected therefore they had no expectations that they were to be witnessing smoking-related stimuli. The results showed that the anterior intraparietal sulcus and inferior frontal gyrus are part of the mirror neuron systems of smokers. However, the middle frontal gyrus, premotor cortex, and superior parietal lobule represent the smoking-related action knowledge motivated by smoking stimuli (Yalachkov & Naumer, 2011). Furthermore, the dorsal anterior cingulate cortex (dACC), orbitofrontal cortex (OFC), and dorsolateral prefrontal cortex (DLPFC) are brain regions known for their role in the reward, craving, and

executive functions of addiction. These findings indicate that smoking related stimuli elicits the higher activation in the dACC, OFC, and DLPFC, when compared to those of non-smokers (Wagner et al., 2011). Wagner et al., (2011) made an important contribution to the understanding of how the sensorimotor brain regions are localised and involved in addiction. With this,

Studies of the nicotine dependant brain and the effects of nicotine patches reducing cravings have been the predominately used treatment for addiction Yoder, et al., (2012). Yoder, et al., (2012) used position emission tomography (PET) to assess changes in dopamine, they claimed that transdermal nicotine patches (TNP) can control nicotine craving, however the effects of nicotine patches on the chemical stimulus binding is unknown and difficult to corroborate for conclusive data. They suggest there are many types of challenge paradigms involved, which are cognitive, motor, and pharmacologic (Yoder, et al., 2012, p. 220). Current smoking pharmacotherapy or nicotine patches, relies on replacing nicotinic receptor stimulation with compounds that have relatively close characteristics that consequently will decrease nicotine craving without producing the significant reward functions, therefore no longer reinforcing the anticipated effects (Brody et al., 2004).

Current literature has highlighted that the methods for smoking cessation are not efficient (Gipson et al., 2013). Developments in this area are crucial as the rate of relapse is significant (Gipson et al., 2013). Future research would benefit from examining the neurological changes produced by cigarette use which underpin persistent relapse vulnerability (Gipson et al., 2013). Nicotine addiction occurs when people rely on smoking to modulate

mood and arousal or to relieve withdrawal symptoms. The current research supports findings from Wagner et al. (2011), demonstrating that smoking-related cues elicit higher activation in localised brain regions. Due to excessive exposure to nicotine neuroadaptation occurs creating a tolerance which is believed to play a role in dependence (Benowitz, 2010). However on the contrary, Brody et al., (2004) claimed that smoking-induced reductions in nondisplacable binding potentials are associated with decreased cravings for cigarettes and proposed that increases in dopamine actually alleviate cigarette cravings. Many factors interplay in determining nicotine addiction, these factors are either learned or conditioned, genetic, and social and environmental factors (Benowitz, 2010).

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