

Critically evaluate the cognitive explanations of addiction



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With around a third of the population chronically addicted to nicotine and the ever increasing burden of alcohol related illness on the National Health Service, the study of what causes and maintains addiction is one of the most important psychological issues on the government's health agenda today. Coupled with advances in medical and pharmacological treatments for addiction, which owing to the remit of the present review, are not discussed; psychologists have sought cognitive explanations for the induction, function, mechanisms and persistence of addiction and relapse. Resultantly, the purpose of this essay is two-fold.

Firstly, social-cognitive theories of influence and emotional factors such as stress are presented by way of introduction to mechanisms which may induce and help to maintain addiction. This is followed by more micro-cognitive approaches from the area of cue-reactivity; an area which has played an enormous part in establishing a possible role for preconscious pathological attentional bias toward addictive substances which may be responsible for the automatising of addiction and for producing more conscious cognitive responses such as craving. To conclude, a biological learning and memory theory exploring the underlying neural mechanisms of addictive cognition is tentatively proposed as explanation of the motivational characteristic that drives the addict to consume and thus the prevalence and persistence of relapse after abstinence. Finally, the second aim of this study is to explore, through the material covered, the concept of a 'cognitive explanation' which, it will be seen, refers to both complex meta-cognitions (such as thought and belief) and the intricacies of neural cognition.

Firstly, addiction has been studied as stemming from an interplay of complex social factors such as stress, anxiety and peer pressure. In such a situation, addiction to alcohol and nicotine particularly results from both positive cognitions towards the socially rewarding effects of the drug, and substance dependency over the period of exposure. Described as the outcome expectancy model, addiction is represented in these studies as a gradual shift from positive expectancies and correlated substance use, to increasing tolerance and eventually physical dependence.

In a 'drinking-to-cope' experiment by Carrigan and colleagues (2008), socially anxious individuals were identified from a group of students using self-report (the Social Interaction Anxiety Scale and the Social Phobia Scale) and correlated with their tendency to abuse alcohol (also assessed by self-report) against their non-anxious counterparts. Crucially, the study also measured their attitude towards alcohol's positive effects on social coping in order to explore the extent to which positive outcome expectancy was a factor thus expanding on the findings by Cooper, Russell & George (1988) that drinking to cope behaviour is only correlated with alcohol abuse if positive cognitions towards alcohol's positive effects are present. While their study only inferred drinking motives from measures of social anxiety, they did indeed find that the 'drinking to cope' (anxious) individuals showed higher levels of alcohol consumption and dependence symptoms (also assessed by self-report measures).

Overwhelmingly the literature on adolescent nicotine dependence characterises addiction as initiated by social motives. Research by Valente, Unger and Johnson (2005) into the 'popularity-value' of smoking in <https://assignbuster.com/critically-evaluate-the-cognitive-explanations-of-addiction/>

adolescent social culture has emphasised the link between social desirability and therefore the positive expected outcome of participating in behaviour which may easily become addictive (such as smoking). However, other studies have also highlighted the importance of cognition post-addiction. In a recent study in the Netherlands, Kleinjan et al. (2009) found that, after exposure has led to addiction, adolescents use rationalisations that trivialise the well known health risks such as “ I live healthily otherwise” and “ everyone acts unhealthily sometimes”. This expanded a wealth of research finding the same effect in adult populations. Overwhelmingly they found that readiness to quit and cessation were negatively correlated with such beliefs and reported that “ having stronger disengagement beliefs is associated with higher nicotine dependence” (p. 443).

This summary of their findings seems to suggest a causal relationship between proclivity to disengagement and increasing nicotine dependency or that, while the cognitions are responsible for maintaining the addiction, they are also predictive of the extent of addiction from the outset. It may be however, that unconscious, automatic processes are causative of increasing addiction and that the permissive cognitions described, arise so as to dispel cognitive dissonance between a compunction to smoke and knowledge of the health risks.

Cue-reactivity theorists have proposed that an essential element of addiction is, at base, a preferential cognitive bias for substance related stimuli leading to basic autonomic responses, such as sweating and palpitations. In turn, this elicits more complex subjective cravings which require the cognitive effort necessary to produce conscious thought. Addiction, therefore, is seen

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as largely an unconscious process and is assessed by experiments testing implicit associations occurring before explicit outcome expectancies. For example, the addiction Stroop test (adapted from Stroop, 1935) consists of two categories of words, one substance-related and one neutral, which are matched for confounding variables such as word length and number of syllables. In their experiment, Cox et. al (2006) presented participants with the words in random order and coloured either blue, red, green or yellow. Participants were required to give the name of the font-colour and not the word itself and measures of attentional bias were indexed as the difference between naming latency on substance-word-trials and neutral-trials.

Slower colour-naming on substance word trials were concluded to indicate a stronger bias for the semantic meaning of these words than for the neutral stimuli. However, this has been challenged by other researchers such as Klein (2007) and Field and Cox (2008) who question latency as unequivocally representing attentional bias. For instance, the study by Klein repeated the methodology used by Cox et al. but with one subtle change; asking half of the participants to suppress thoughts of alcohol during the test, and asking half to freely associate. The results by Cox et al. were only reproduced for the group attempting to suppress their automatic attentional bias and no significant difference was found for the group allowed to freely associate. Moreover, Field and Cox in their 2008 review, draw attention to Tiffany's 1990 theory on the cognitive strain of suppression as a confounding variable in the Stroop task, suggesting that it may not be testing attentional bias at all, but merely that a general slow-down in cognitive processing occurs as craving drains cognitive resources.

However, Field and Cox (2008) do not deny that this craving response is elicited by cue-reactivity and suggest other, more valid paradigms for testing such a hypothesis. One such approach, the attentional cueing task, has been used by researchers such as Mogg et al. (2003) to test the difficulty with which smokers were able to distract their attention from a drug-related cue on a computer screen. Controlling for the problems associated with reaction time, as aforementioned, Mogg and colleagues also recorded participants' eye movements as the stimuli were presented. In the attentional cueing task, participants were presented with twenty drug-related and twenty neutral cues all presented twice with additional filler pictures for filler trials. Drug related pictures were almost identical to neutral pictures however, for example, where a woman was seen to be smoking in a drug-related picture, she may be applying lipstick in a neutral picture. On each trial, two pictures appeared, one in the left and one the right visual field for 2000 milliseconds, after which, a target cue either (:) or (..) appeared over one of the pictures. Participants were required to indicate the identity of the cue and horizontal eye movement was recorded for each trial. Finding that smokers, but not non-smokers, maintained their gaze for longer on smoking-related cues and that they were faster to detect probes that replaced smoking-related than control pictures, the authors concluded that their research supports an attentional bias hypothesis of addiction.

However, a critical question remains answered by the evidence presented thus far. That is, how does attentional bias lead to the behavioural patterns observed in addiction? While cue-reactivity addresses the cognitive elements of perception and attention in drug addiction, it is not made explicitly clear

how the induction of subjective craving brings about the behaviour necessary for drug seeking and consumption. Moreover, while it appears from the social evidence presented that there is a social reward for drug-taking behaviour, be that the admiration of adolescent peers in smoking, or a perceived increase in social ability in those suffering from social anxiety; such social and personality-based theories cannot account for the fairly consistent behavioural patterns observed in serious drug addiction. The pathological learning and memory theory, developed by Steven Hyman, assesses cognitive processes at their most primitive functional level to explain the behaviour of the addict.

Hyman (2005) suggests that, given current understandings of the neural processes of cognition, addiction is a process of learning and memory hijacked by the positive and somewhat overwhelming effects of drugs - particularly illicit drugs of abuse. In his model, addiction becomes a basic homeostatic need, often above hunger and thirst as it mimics and supplants such natural consummatory behaviours. He describes the process by which feeding and hydrating become automatic as being overlearnt and suggests that drug consumption becomes overlearnt by the same mechanisms. But how do drug processes become overlearnt and what gives certain drugs (such as opioids and cocaine) their special quality to supersede other homeostatic behaviours?

For Hyman the key concept is reward. Kelley and Berridge (2002) outline a number of studies in their review suggesting that drugs of abuse act on centres within the brain normally associated with pleasures such as taste and liking for food and so mimic the normal processes of pleasure-learning.

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For instance, rats injected with an opioid compound have consistently been found to consume more sweet tasting sucrose water, in line with the prediction that the drug enhanced food palatability. But, as Hyman and Kelley and Berridge have stated, unlike food consumption, which directly rewards pleasurable taste centres, it is not necessary for a drug to be enjoyable in itself to produce rewarding effects in multiple sensory areas. In fact, in Hyman's view, drugs of abuse gain their competitive advantage over normal rewards (such as sweet tastes) by producing higher levels of dopamine than possible for the normal reward systems.

As such, it seems that an account of addiction could (and has been) construed in a purely biological vein; that dopamine acts hedonically to produce pleasure, and by increasing intake of drugs eliciting the dopamine 'pleasure response' the body becomes physically addicted. However, when this has been tested in animal models, by blocking, lesioning or removing the ability to respond to dopamine post-addiction learning, animals continue to prefer the pleasurable consummatory behaviours (e. g. an increased consumption of sucrose water) as before. (see Berridge and Robinson, 1998; Cannon and Palmiter, 2003).

Thus dopamine, Hyman argues is involved in a complex learning and memory process between dopamine as a pleasure signal and normal modes of associative learning and memory such as Long-term Potentiation. He refers to a series of experiments by Schultz and colleagues (1993) in which alert monkeys were trained to expect sweet juice (reward) after a certain period of time and a cue. Recording brain activity, they found that different patterns of dopamine firing occurred when sweet juice was delivered on-time, <https://assignbuster.com/critically-evaluate-the-cognitive-explanations-of-addiction/>

when it was delivered late and when delivered before expected. As such, Hyman proposes that dopamine signalling provides the motivational element to pleasure-learning by signalling behaviours that produce pleasure. These waves of dopamine activity are then associated by long-term memory processes with the exact cue, context and action that produced the pleasure and the addiction is committed to memory. Finally, the overwhelming and unnatural amounts of dopamine produced saturate these cognitive processes and ensure that the addiction is over-represented in memory as highly pleasurable, contrary to reduced perceived feelings of being 'high' due to physical tolerance.

However these micro-cognitions do not exist in isolation and it is through identifying the basis of contextual learning that the wider components of addiction in perceptual and social context can be fully explained. Overall, all three approaches contribute to a full and detailed understanding of the mechanisms of addiction and, while there exist biological treatments to suppress the compulsion of alcohol and other addictions, it will not be without continued progress in all areas of the cognitive domain that better addiction therapies will be created.