What's deficient in reward deficiency?

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It has been proposed that people with substance use disorders suffer from a reward deficiency syndrome. Sometimes this is framed as a pre-existing trait; sometimes it is framed as an acquired condition. Although the reward deficiency idea is often used as an explanation for excessive substance use, there is little consensus about the deficiency's basis or how it might increase drug and alcohol use.

Is there deficient reward in the absence of drug use?

At least 3 populations at elevated risk of addictions show evidence of having fewer rewarding experiences when abstaining from drugs and alcohol. First, individuals in depressive states typically have a reduced ability to experience pleasure (anhedonia) or a loss of interest in potential pleasures.[1–3] Second, people with high sensation-seeking traits are often characterized as being easily bored and actively engaged by intense stimulation only.[4*] Third, those who use drugs can have transient reductions in rewarding experiences in the immediate aftermath of a drug binge[6,7] and, possibly, more enduring reductions following quite extensive substance use.[6,8] In all 3 groups, it seems difficult to explain the initiation of reward seeking. Deficit states, in and of themselves, do not induce reward-seeking behaviours.[9] Deficits can, however, produce a contrast effect, increasing the drawing power of rewards once the reward has been experienced.[10] One can therefore imagine individuals who, after these initial experiences, are attracted to intensely rewarding substances and little else.

Is there deficient drug-induced reward?

In a second version of the reward deficiency hypothesis it is the drug-induced responses that are reduced, and the only way for an affected individual to experience a reasonable amount of pleasure is to consume a greater amount of drug in a given length of time. One potential example is that elevated susceptibility to alcohol use disorders is associated with a lowered alcohol intoxication response.[11] Of note, though, this low level of response predictor of alcohol use problems is based on reduced body sway. Thus, the diminished response might primarily reflect decreased sedative effects of alcohol rather than decreased positive affective experiences.[†] Contradicting the idea of a deficiency in the pleasure response is an equally compelling literature indicating that most individuals considered at risk for addictions report larger positive affective responses to drug challenges,[14–16] raising the possibility that these affective features are either less relevant than has been suggested or instead reflect secondary features of other underlying processes.

Is there deficient dopamine?

One prominent idea commonly embedded in the reward deficiency hypothesis is that drug use is the consequence of a low functioning midbrain dopamine (DA) system.[17] For example, Blum and colleagues[18] note that, despite the many incarnations of the hypothesis, it "continues to posit that hypodopaminergic function predisposes an individual to seek psychoactive substances and behaviours to release DA in reward circuits of the brain to overcome DA deficits." This proposition is interesting and seems consistent with the proposal that some individuals at risk for addictions experience depressive mood states. However, the specific formulation also seems to reflect the now largely abandoned

[*] Compared with most other people, sensation seekers appear to have reduced appetitive responses to low salience stimuli. Their response to motivationally salient stimuli, though, might be greater than average in absolute magnitude, greater in proportion to their response to low salience stimuli, or neither. Behavioural and neuroimaging studies suggest that the latter possibility is the least likely.[5]

[†] Disentangling sedative from stimulant effects of ethanol has been difficult. In a self-report measure of the low alcohol response, the items considered most sensitive are Alcohol Effect, Clumsy, Confused, Dizzy, Drunk, High and Trouble Concentrating.[12] Of note, this low intoxication response is reported to be associated with large, rather than blunted, striatal dopamine responses when individuals drink alcohol.[13]

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idea that increases in DA equal pleasure. Contemporary views are that elevated DA transmission enhances the ability of motivationally salient stimuli to draw and sustain approach, but these behavioural effects do not derive from increased pleasure.[19–23‡]

What, then, is the empirical evidence for altered DA transmission in addictions in humans? In those with severe and sustained substance use disorders, reduced amphetamine-evoked DA responses have been seen with considerable consistency.[24,25] Moreover, there is recent evidence that this muted response may be a pre-existing (or rapidly developing) vulnerability trait.[26] The basis and meaning of these low DA responses, however, remain unclear. They could reflect neurotoxic effects in late-stage addictions,[27] heritable endophenotypes, conditioned inhibitory effects induced by cues that signal the absence of reward,[5,28] or some combination of these effects (e.g., initial predispositions aggravated by rapidly developing conditioned effects and later-stage neurotoxicity).

In support of the idea of conditioned inhibitory effects, low DA and other striatal responses have been observed in high-risk individuals when they are tested with cues associated with the absence of drugs.[5,28] In comparison, augmented striatal responses have been seen when high-risk individuals are tested in the presence of drug-paired cues.[5,13,28] Indeed, across stages of addiction, striatal DA release is induced by drug-paired cues alone.[5,28,29] Thus, although the full clinical picture of addiction may well include periods of both increased and decreased DA transmission, the appearance of drug-related cues can provoke DA release, transforming a low DA state into a high one, fostering impulsive actions[30] and drug-seeking behaviours.[19,21,31–33]

Conclusion

The above observations suggest that 2 variants of a deficiency hypothesis might have some utility, though not as originally envisaged. First, there may be individuals for whom many reward effects are muted, with only quite potent events able to induce DA release and motivate approach and desire. This might be seen most typically during and immediately after an extended drug binge; in late-stage substance use disorders where the DA system may have suffered damage; or, possibly, in individuals with pre-existing traits, such as sensation seeking or aspects of depression. Second, there may be individuals for whom DA-related approach responses are augmented in the presence of cues previously paired with the addictive substance and inhibited by cues associated with their absence. Central to the current discussion, in each of these scenarios, individual bouts of drug seeking are precipitated by increases in DA transmission rather than decreases.

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‡As summarized by John Salamone, "We don't seek DA; DA makes us seek" (personal communication, 2014).

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