

Short Note on Dark Side of Addiction

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ABSTRACT

Addiction is a biopsychosocial disorder characterized by repeated use of drugs, or repetitive engagement in a behavior such as gambling, despite harm to self and others. According to the "brain disease model of addiction," while a number of psychosocial factors contribute to the development and maintenance of addiction, a biological process that is induced by repeated exposure to an addictive stimulus is the core pathology that drives the development and maintenance of an addiction. Many scholars who study addiction argue that the brain disease model is incomplete and misleading. Emotions are "feeling" states and classic physiological emotive responses that are interpreted based on the history of the organism and the context. Motivation is a persistent state that leads to organized activity. Both are intervening variables and intimately related and have neural representations in the brain. The present thesis is that drugs of abuse elicit powerful emotions that can be interwoven conceptually into this framework.

Keywords: Allostasis; Corticotropin-releasing factor; Dynorphin; Extended amygdala; Incentive salience; Opponent process

INTRODUCTION

The brain disease model posits that addiction is a disorder of the brain's reward system which arises through transcriptional and epigenetic mechanisms and develops over time from chronically high levels of exposure to an addictive stimulus (e.g., eating food, the use of cocaine, engagement in sexual activity, participation in high-thrill cultural activities such as gambling, etc.). DeltaFosB (Δ FosB), a gene transcription factor, is a critical component and common factor in the development of virtually all forms of behavioral and drug addictions. Two decades of research into Δ FosB's role in addiction have demonstrated that addiction arises, and the associated compulsive behavior intensifies or attenuates, along with the overexpression of Δ FosB in the D1-type medium spiny neurons of the nucleus accumbens. Due to the causal relationship between Δ FosB expression and addictions, it is used preclinically as an addiction biomarker. Δ FosB expression in these neurons directly and positively regulates drug self-administration and reward sensitization through positive reinforcement, while decreasing sensitivity to aversion

The term behavioral addiction refers to a compulsion to engage in a natural reward – which is a behavior that is inherently rewarding (i.e., desirable or appealing) – despite adverse consequences. Preclinical evidence has demonstrated that marked increases in the expression of Δ FosB through repetitive and excessive exposure to a natural reward induces the same behavioral effects and neuroplasticity as occurs in a drug addiction. Reviews of both

clinical research in humans and preclinical studies involving Δ FosB have identified compulsive sexual activity – specifically, any form of sexual intercourse – as an addiction (i.e., sexual addiction). Moreover, reward cross-sensitization between amphetamine and sexual activity, meaning that exposure to one increases the desire for both, has been shown to occur preclinically and clinically as a dopamine dysregulation syndrome; Δ FosB expression is required for this cross-sensitization effect, which intensifies with the level of Δ FosB expression.

Reviews of preclinical studies indicate that long-term frequent and excessive consumption of high fat or sugar foods can produce an addiction (food addiction). This can include chocolate. Chocolates' sweet flavour and pharmacological ingredients is known to create a strong craving or feel 'addictive' by the consumer. A person who has a strong liking for chocolate may refer to themselves as a chocoholic. Chocolate is not yet formally recognised by the DSM-5 as a diagnosable addiction. Gambling provides a natural reward which is associated with compulsive behavior and for which clinical diagnostic manuals, namely the DSM-5, have identified diagnostic criteria for an "addiction". In order for a person's gambling behavior to meet criteria of an addiction, it shows certain characteristics, such as mood modification, compulsivity, and withdrawal. There is evidence from functional neuroimaging that gambling activates the reward system and the mesolimbic pathway in particular. Similarly, shopping and playing video games are associated with compulsive behaviors in humans and have also been shown to activate the mesolimbic pathway and other parts of the reward system. Based

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Received: February 08, 2021; Accepted: February 15, 2021; Published: February 22, 2021

Citation: Samson R (2021) Short Note on Dark Side of Addiction. J Alcohol Drug Depend 9: e103.

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upon this evidence, gambling addiction, video game addiction, and shopping addiction are classified accordingly.

REFERENCES

1. Werawatganon D, Rakananurak N, Sallapant S, Prueksapanich P, Somanawat K, Klaikeaw N, et al. Aloe vera attenuated gastric injury on indomethacin-induced gastropathy in rats. *World J Gastroenterol.* 2014; 20:18330-18337.
2. Grigoryants V, Hannawa KK, Pearce CG, Sinha I, Roelofs KJ, Ailawadi G, et al. Tamoxifen up-regulates catalase production, inhibits vessel wall neutrophil infiltration, and attenuates development of experimental abdominal aortic aneurysms. *J Vasc Surg.* 2005;41:108-114.
3. Bancroft JD, Gamble M, editors. *Theory and practice of histological techniques.* Elsevier health sciences; 2008.
4. Yue M, Ni Q, Yu CH, Ren KM, Chen WX, Li YM, et al. Transient elevation of hepatic enzymes in volunteers after intake of alcohol. *Hepatobiliary Pancreat Dis Int.* 2006; 5:52-55.
5. Singh A, Bhat TK, Sharma OP. Clinical biochemistry of hepatotoxicity. *J Clinic Toxicol* 2011.
6. Silva MA, Rao VS, Souza CM, Neves JC, Menezes DB, Santos FA, et al. Evaluation of thalidomide against indomethacin-induced small intestinal damage and systemic toxicity in rats. *Biomedical Research.* 2012; 23:125-133.
7. Olusegun Taiwo V, Lawal Conteh O. The rodenticidal effect of indomethacin: pathogenesis and pathology. *Veterinarski arhiv. Veterinarski Arhiv.* 2008;78:167-178.