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CHALLENGES FOR MEDICAL MODELS OF ADDICTON

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It is standard in current neurobiological research on addiction to describe addiction as a chronic disease. Working from this perspective, much effort has gone into characterizing the symptomology of addiction and the brain changes that underlie them. Evidence for involvement of dopamine transmission changes in the ventral tagament area and nucleus accumbens have received the greatest attention. Kaur and Malenka (2007) put it well: “drugs of abuse can co-opt synaptic plasticity mechanisms in brain circuits involved in reinforcement and reward processing”.

Our goal in this paper is to provide an explicit description of the assumptions of medical models, the different forms they may take, and the challenges they face in providing explanations of addiction with solid evidence. We first spell out the requirements of disease models and use them to point out ambiguities in the claims of those who defend medical models. After that, we ask to what extent DSM categorization is suitable to medical models and what aspects of the life course of addiction medical models are supposed to explain. In the third section, we note that a primary aim of the neurobiology of addiction is to localize the causes of addiction to cellular and molecular pathways in the brain´s reward system. We investigate how well current neurobiological approaches approximate towards such a unified causal account of addiction, and what challenges current approaches still face. We finish with a survey of some of the important social processes in addiction and the challenges they raise for medical models.

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