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Dopamine and Addiction: using PET to Explore the Neurobiology of Substance Abuse

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Monday, January 28, 2013
2:30pm (*Please note special time*)

Brady Auditorium, 310 Cedar St.

Abstract The involvement of dopamine in addiction has its origins in studies investigating reward and reinforced behavior in preclinical studies. Much of this research has been explored in the human brain using Positron Emission Tomography (PET) imaging of striatal dopamine transmission. These studies show that addiction is associated with a decrease in dopamine D2/3 receptors and a decrease in pre-synaptic dopamine release, and that this decrease occurs across different types of addiction, including cocaine, alcohol, and heroin dependence. However, these imaging studies also show that, in cocaine abuse, blunted dopamine transmission is predictive of cocaine seeking behavior. Low D2/3 receptor binding and low dopamine release are associated with the choice to self-administer cocaine over alternative reinforcers (such as money), which can be viewed as a failure to shift between competing rewards.

It is striking that addictions to different substances of abuse are accompanied by the same alteration in neurobiology, independent of their primary impact on the dopaminergic system. Moreover, similar alterations of the dopaminergic transmission and D2-like receptor system have been described in psychiatric diseases other than addiction. Although these psychiatric disorders differ in their phenomenology, they share a common deficit in reward-related behavior, particularly with respect to impulsivity and motivation. This presentation will describe the animal and human studies that link alterations in dopamine transmission and the D2-receptors with impulsive and motivated behavior. The hypothesis that these alterations in dopamine transmission represent the neurobiological underpinnings that facilitate impulsivity and undermine motivation, rather than the consequences of addiction itself, will be discussed.