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Reward deficiency syndrome: a biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors.

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Abstract

The dopaminergic system, and in particular the dopamine D2 receptor, has been implicated in reward mechanisms. The net effect of neurotransmitter interaction at the mesolimbic brain region induces "reward" when dopamine (DA) is released from the neuron at the nucleus accumbens and interacts with a dopamine D2 receptor. "The reward cascade" involves the release of serotonin, which in turn at the hypothalmus stimulates enkephalin, which in turn inhibits GABA at the substania nigra, which in turn fine tunes the amount of DA released at the nucleus accumbens or "reward site." It is well known that under normal conditions in the reward site DA works to maintain our normal drives. In fact, DA has become to be known as the "pleasure molecule" and/or the "antistress molecule." When DA is released into the synapse, it stimulates a number a DA receptors (D1-D5) which results in increased feelings of well-being and stress reduction. A consensus of the literature suggests that when there is a dysfunction in the brain reward cascade, which could be caused by certain genetic variants (polygenic), especially in the DA system causing a hypodopaminergic trait, the brain of that person requires a DA fix to feel good. This trait leads to multiple drug-seeking behavior. This is so because alcohol, cocaine, heroin, marijuana, nicotine, and glucose all cause activation and neuronal release of brain DA, which could heal the abnormal cravings. Certainly after ten years of study we could say with confidence that carriers of the DAD2 receptor A1 allele have compromised D2 receptors. Therefore lack of D2 receptors causes individuals to have a high risk for multiple addictive, impulsive and compulsive behavioral propensities, such as severe alcoholism, cocaine, heroin, marijuana and nicotine use, glucose bingeing, pathological gambling, sex addiction, ADHD, Tourette's Syndrome, autism, chronic violence, posttraumatic stress disorder,

schizoid/avoidant cluster, conduct disorder and antisocial behavior. In order to explain the breakdown of the reward cascade due to both multiple genes and environmental stimuli (pleiotropism) and resultant aberrant behaviors, Blum united this hypodopaminergic trait under the rubric of a reward deficiency syndrome.

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