BACKGROUND

The economic cost of substance use disorders (SUDs) is higher than the cost of other prevalent diseases such as heart disease. Further, only 10% of individuals with SUDs receives treatment—a phenomenon known in the field as “treatment gap.” Of those that receive treatment, less than 50% remain abstinent after treatment discharge. Hence, a better understanding of the underlying causes that promote or reduce the risk for addiction is crucial to provide new insights for prevention and treatment. Multiple theories of addiction posit that repeated use of drugs can alter the neurocircuitry of reward processing in ways that bias motivational systems toward drug pursuit, at the expense of naturally rewarding activities.

• The incentive-sensitization theory of addiction posits that cues signaling drug availability take on the incentive value of the drugs themselves.
• Reward-deficit models posit that risk for drug use is conferred by a blunting of the incentive-motivational value of natural (i.e., nondrug) reinforcers.
• Behavioral economic and value-based decision-making models assert that the ratio of substance-free and substance-related reward is critical to addiction etiology.

The current work reviews the most influential theoretical perspectives of vulnerability to addiction and recent empirical support for the idea of reward dysregulation as a neurobehavioral marker of addiction risk.

THEORETICAL MODEL

Integrative Theoretical Model of Vulnerability for Addiction

Drugspecific Reactivity

REWARD DYSREGULATION

Drug-dependence

Drug use, drug craving, and withdrawal

Mark of Risk

Intention

Reactivity to Natural Rewards

Addiction Recovery

Personal drug use history

EMPIRICAL EVIDENCE

REWARD DYSREGULATION AND TOBACCO DEPENDENCE

Reward dysregulation predicts long-term smoking abstinence:

Smokers with low neural reactivity to non-drug-related pleasant images but high reactivity to smoking-related cues were more likely to relapse after a quit attempt.

REWARD DYSREGULATION AND COCAINE USE DISORDER

Reward dysregulation can be reversed with abstinence:

Parvaz et al. demonstrated that this reward dysregulation phenotype can be reversed in a sample of individuals with cocaine use disorder (CUD).

EMPIRICAL EVIDENCE (CONT.)

REWARD DYSREGULATION AND ALCOHOL DEPENDENCE

Reward dysregulation predicts drinking outcomes:

The reward dysregulation P3 (or differential valuation of alcohol vs. natural rewards) consistently and robustly predicted drinking outcomes in a sample of nondependent, young adult drinkers.

CONCLUSIONS

Taken together, these findings suggest that the reward dysregulation reflecting the differential valuation of drug-related reward vs. naturally-occurring rewards is critical for better understanding the underlying causes of addictive behaviors and neurophysiological indexes of this reward dysregulation can be used as meaningful tools for vulnerability assessment, clinical diagnosis, and recovery evaluation.

Research on addiction should not focus exclusively on motivation for drug use (i.e., craving and withdrawal symptoms) but also place emphasis on the role of non-drug-related rewarding activities, which can be the key for novel and better treatments for individuals with SUDs.