Models of Addiction I



Models of Addiction

Addiction A maladaptive behavioural pattern of drug use, characterized by overwhelming involvement with the use of drug (compulsivity), the securing of supply, and high tendency to relapse after withdrawal, with disregard to negative consequences.

Models of Addiction

The Disease Model

- Until the mid-19th century addiction was considered a sin, a moral deficiency that should be treated by priests or the legal system
- The social changes of the late 19th century gave rise to the idea that addiction is actually a disease and addicts should be treated and not punished
- The susceptibility models (e.g., E.M. Jellinek): the problem is with the individual and not the drug
- The Exposure models: the drug changes the brain

Effects of Alcohol on the Individual: Review of the Literature of 1939.* E. Morton Jellinek, M.Ed., Sc.D. (Hon.) Executive Director, Study of the Effects of Alcohol on the Individual. Norman Jolliffe, M.D. Associate Professor of Medicine, New York University College of Medicine; Chief of the Medical Service of the Psychiatric Division, Bellevue Hospital. Received for publication April 10, 1940. INTRODUCTION WITHIN a century a jungle of 96,000,000 words has grown around the problem of alcohol. The lack of comprehensive periodic surveys of this dense growth has made itself felt in

the task of compiling a critical reference work on the effects of alcohol



Models of Addiction

Percent Using in Past Month

Addiction: a disorder of choice?



Gene Heyman

Illicit drug use (past month)



National Survey on Drug Use and Health, 2010 (U.S. data)

I. Negative Reinforcement Models

a. The Physical Dependence Model

- Addiction = Physical Dependence
- Drug is taken in order to alleviate and then to avoid the unpleasant effects of withdrawal
- In other words, the drug becomes a **negative reinforcer**
- Conditioned withdrawal might be triggered by the environment and result in relapse even in abstinent subjects

I. Negative Reinforcement Models

a. The Physical Dependence Model

Problems with the model:

- 1. Does not address the reason for developing drug use to dependence
- 2. Addiction can develop without physical withdrawal (e.g., <u>Bozart & Wise</u> <u>1984</u>)
- 3. Addicts rarely mention withdrawal as the reason for relapse:

"No, Doc, craving is when you want it – want it so bad you can almost taste it ... but you ain't sick ... sick is, well sick." (from A.R. Childress et al., NIDA Research Monographs, Vol. 84, 1988)

4. Self-reported craving for cocaine is higher AFTER taking the drug

I. Negative Reinforcement Models

b. The Self-medication hypothesis

- The drug is used to medicate an existing negative state (anxiety, stress, pain...)
- So, Valium is used to avoid anxiety, and alcohol to avoid stress
- This assumption was tested by de Wit et al., (1986):



Results:

- 1. No preference for "blue pill", even after screening for anxiety levels
- 2. Same results with depression and amphetamine
- 3. Exception: preference develops for opiates if pain is expected

I. Negative Reinforcement Models

- c. The opponent process and allostasis models
 - The opponent process model of affective equilibrium (Solomon & Corbit 1974)



- I. Negative Reinforcement Models
 - The Allostatic model (Koob & Le Moal 2001)

Addiction is the result of decreased function of the reward system and recruitment of "anti-reward" systems (similar to the "b" process or "counteradaptation")

The reward system: mesolimbic DA, opiate receptors and peptides, GABA receptors

Examples for decreased function in the reward system: reduce DA D2 receptors in the striatum of cocaine addicts; increased ICSS threshold in rats after chronic use



DA D2 receptors

Cocaine abuser (4 months abstinence)

Volkow 2006



Ahmed et al 2002

- I. Negative Reinforcement Models
 - The Allostatic model (Koob & Le Moal 2001)

However, Koob and Le moal emphasize the "DARK SIDE" of addiction, i.e., the adaptations in the anti-reward mechanisms



Opiates can be rewarding independently from their withdrawal alleviating properties (Bozarth & Wise, 1984)

- Morphine (100 ng/infusion) is selfadministered into the VTA but not other opiate receptor-rich areas (e.g., PVG – periventricular gray)
- Next, morphine 0.5 mg/hr/72 hr was infused into different regions using osmotic minipumps
- Rats were then tested with naloxone (5 mg/ kg) and withdrawal symptoms were recorded (escape from enclosure, wet-dog shakes, chattering teeth)





 The opponent process model of affective equilibrium: a "real-life" demonstration with cocaine smoking

