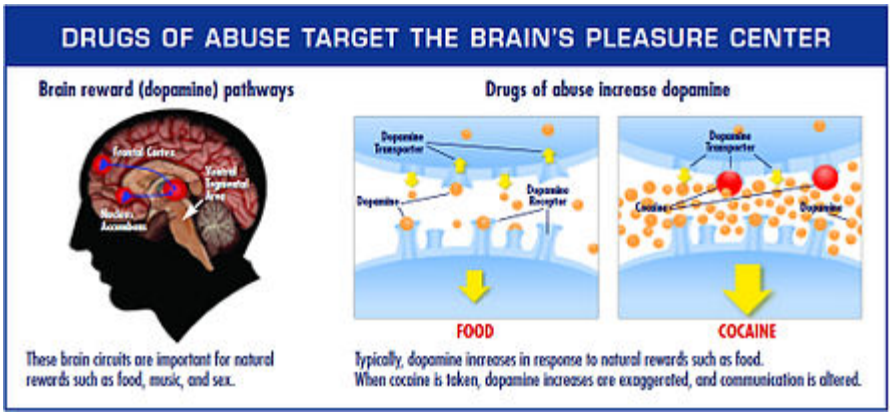
Reward system

Certain neural structures, called the **reward system**, are critically involved in mediating the effects of reinforcement. A reward is an appetitive stimulus given to a human or some other animal to alter its behavior. Rewards typically serve as reinforcers.



Drugs of abuse target the brain's pleasure center.[1]

A reinforcer is something that, when presented after a behavior, causes the probability of that behavior's occurrence to increase. Note that, just because something is labelled as a reward, it does not necessarily imply that it is a reinforcer. A reward can be defined as reinforcer only if its delivery increases the probability of a behavior.[1]

Reward or reinforcement is an objective way to describe the positive value that an individual ascribes to an object, behavioral act or an internal physical state. Primary rewards include those that are necessary for the survival of species, such as food and sexual contact.[2] Secondary rewards derive their value from primary rewards. Money is a good example. They can be produced experimentally by pairing a neutral stimulus with a known reward. Things such as pleasurable touch and beautiful music are often said to be secondary rewards, but such claims are questionable. For example, there is a good deal of evidence that physical contact, as in cuddling and grooming, is an unlearned or primary reward.[3] Rewards are generally considered more desirable than punishment in modifying behavior.[4]

Contents

Definition

In neuroscience, the reward system is a collection of brain structures that attempts to regulate and control behavior by inducing pleasurable effects. It is a brain circuit that, when activated, reinforces behaviors. The circuit includes the dopamine-containing neurons of the ventral tegmental area, the nucleus accumbens, and part of the prefrontal cortex.[5]

Anatomy of the reward system

The major neurochemical pathway of the reward system in the brain involves the mesolimbic and mesocortical pathways. Of these pathways, the mesolimbic pathway plays the major role, and goes from the ventral tegmental area (VTA) via the medial forebrain bundle to nucleus accumbens. The VTA is a source of many dopamine pathways in the brain, which use dopamine neurons transmit a signal to other structures.Dopamine acts on D1-like receptors or D2-like receptors to either stimulate (D1-like) or inhibit (D2-like) the production of cAMP.[6]

Humans and animals seem to have a similar sense of pleasure.[7] The human brain deciphers pleasant events and adds depth by changing the way humans pay attention and notice pleasures. The sense of pleasures differ in humans compared to animals because culture, life events, art, and other cognitive sources expand our understanding. This can make one realize how great a pleasure is or how displeasurable it may be.[8]

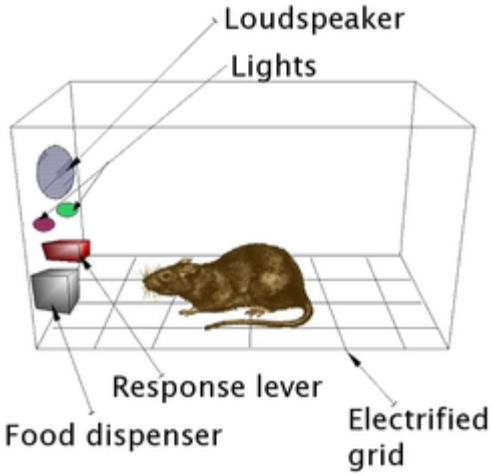
Animals vs humans

Based on data from Kent Berridge, the *liking* and *disliking* reaction involving taste shows similarities among human newborns, orangutans, and rats. Most neuroscience studies have shown that dopamine alterations change the level of likeliness toward a reward, which is called the hedonic impact. This is changed by how hard the reward is worked for. Experimenter Berridge modified testing a bit when working with reactions by recording the facial expressions

of *liking* and *disliking.* Berridge discovered that by blocking dopamine systems there did not seem to be a change of the positive reaction to something sweet; in other words, the hedonic impact remained the same even with this change. It is believed that dopamine is the brain's main pleasure neurotransmitter but, with these results, that did not seem to be the case. Even with more intense dopamine alterations, the data seemed to remain the same. This is when Berridge came up with the *incentive salience hypothesis* to explain why the dopamine seems to only sometimes control pleasure when in fact that does not prove to be happening at all. This hypothesis dealt with the *wanting* aspect of rewards. Scientists can use this study done by Berridge to further explain the reasoning of getting such strong urges when addicted to drugs. Some addicts respond to certain stimuli involving neural changes caused by drugs. This sensitization in the brain is similar to the effect of dopamine because *wanting* and *liking* reactions occur. Human and animal brains and behaviors experience similar changes regarding reward systems because they both are so prominent.[7]

History

James Olds and Peter Milner were researchers who found the reward system in 1954. They discovered, while trying to teach rats how to solve problems and run mazes, stimulation of certain regions of the brain. Where the stimulation was found seemed to give pleasure to the animals. They tried the same thing with humans and the results were similar.



Skinner box

In a fundamental discovery made in 1954, researchers James Olds and Peter Milner found that low-voltage electrical stimulation of certain regions of the brain of the rat acted as a reward in teaching the animals to run mazes and solve problems.[9][10] It seemed that stimulation of those parts of the brain gave the animals pleasure,[9] and in later work humans reported pleasurable sensations from such stimulation. When rats were tested in Skinner boxes where they could stimulate the reward system by pressing a lever, the rats pressed for hours.[10] Research in the next two decades established that dopamine is one of the main chemicals aiding neural signaling in these regions, and dopamine was suggested to be the brain's “pleasure chemical”.[11]

Substance and behavioral addictions

Main article: ΔFosB

The common factor among virtually all forms of addiction – behavioral addiction and drug addiction – that is necessary and sufficient for many types of addiction-related plasticity is the gene transcription factor ΔFosB (delta FosB); specifically, the overexpression of ΔFosB in the nucleus accumbens, a component of the reward system, occurs with and modulates many aspects of behavioral- and neuro-plasticity that occur in addiction. Examples of behavioral plasticity regulated by ΔFosB include sensitization and cross-sensitization effects to reinforcers. Examples of neuroplasticity modulated by ΔFosB include altered trafficking or density of NMDA receptors, AMPA receptors, and dopamine receptors in the striatum and nucleus accumbens.

Almost all drugs causing drug addiction increase dopamine release in the mesolimbic pathway,[12] e.g. opioids, nicotine, amphetamine, ethanol, and cocaine. After prolonged use, psychological drug tolerance and sensitization arises.[*citation needed*]

See also

- Anterior cingulate cortex#Reward-based learning theory
- Child grooming
- Classical conditioning
- Operant conditioning
- Brain stimulation reward
- Drug addiction#Reward circuit
- Decision making
- Motivation
- Addiction
- Power and control in abusive relationships
- Psychoactive drug
- Ventral tegmentum#Reward system
- Incentive salience
- Pleasure center
- Learned industriousness

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