Review Article

A Systematic Review on Reward Activation and its Effect on Brain Function

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ABSTRACT

Reward system consists of group of nerves regulates incentive salience (motivation /wanting, associative learning and positively-valanced emotions, particularly ones which is included with pleasure as a core component. Primary rewarding stimuli facilitate survival of itself /offspring, which include homeostatic (eating) and reproductive (sex) rewards. Intrinsic rewards are unconditioned rewards which are attractive and motivate behavior because they are inherently pleasurable. Extrinsic rewards (money) are conditioned rewards that are attractive and motivate behavior, but are not inherently pleasurable. Extrinsic rewards derive their motivational value resulted by learned association (conditioning) with intrinsic rewards and also causes pleasure (euphoria) after being classically conditioned with intrinsic rewards. Reward drives need to eat, drink and mate. Species brain allow them to get better reward makes to win in evolution. The core component of reward system is Pleasure, any action makes an individual return (positive reinforcement) and therefore becomes a reward. This review is an attempt to limelight research evidence to prove reward activation and its benefits.

Keywords: Reward centre, extrinsic, intrinsic, memory, learning.

INTRODUCTION

Humans and animals prefer a reward received after exerting a lot effort to obtain it compared to the same reward after a smaller amount of effort.[1] Reward system is responsible for incentive salience i.e, wanting, desire, associative learning and positively valanced emotions. Reward motivation can modulate episodic memory processes which can support future adaptive behaviour. Learning and memory are closely related concepts. Learning is the process or experience of gaining knowledge or skill. Memory encodes, stores, retains and subsequently recall informations and past experiences in the brain, uses past experience to affect/influence present behaviour.

Memory is of long-term memory and short-term memory. Long-term Memory is divided into 2 types: explicit/declarative memory and implicit/ procedural) memory. Declarative memory/ explicit memory consists of facts and events which is consciously recalled or declared, based on the concept on type of memory consists of information that can be explicitly stored and retrieved. Declarative memory is further classified as episodic memory and semantic memory, episodic memory is memory of experiences and specific events can reconstruct the actual events at any given point. It is the memory of autobiographical events (times, places, associated emotions and knowledge) that can be explicitly stated.

Semantic memory is more structured record of facts, meanings, concepts and knowledge about the external world which is acquired. It includes general factual knowledge, shared with others and independent of personal experience and of the spatial/temporal context in which it was acquired, these memories may have personal context, latter stored as simple knowledge, includes things like types of food, social customs, vocabulary etc. Declarative memory is sensitive to reward-related changes.

Reward signal usually broken down into information regarding the expected value of the reward, pleasure is the core component. Reward activation in brain can induce learning, approach behaviour, choices, and emotions. While exposing to a rewarding stimulus, brain releases dopamine, structures associated with reward system are major dopamine pathways of the brain, mesocorticolimbic dopamine pathway plays a primary role.

The Reward Pathway/ The Mesocorticolimbic Pathway

Mesocorticolimbic pathway has a primary role in reward which originates with dopaminergic cell bodies in Ventral Tegmental Area (VTA) (dopamine rich nucleus) located in the ventral portion of midbrain, dopaminergic axons project and primarily terminate in the nucleus accumbens (NAc) and olfactory tubercle in the ventral striatum, but also extends into the amygdala, lateral septal area and lateral hypothalamus.[2] Mesocortical pathway also originates from VTA and extends its fibres in the prefrontal, cingulate and perirhinal cortex.[3] VTA is in close proximity to the substantianigra, another dopamine rich nucleus. Substantianigra projects to the dorsal striatum where it mediates motor activity and the mesocorticolimbic pathway mediates reward.

Reward activation can cause associative learning (classical conditioning and operant reinforcement). Reward may be intrinsic those which can cause pleasure or externsic reward acts as a magnet elicits wanting but not liking once it is acquired. Experiencing reward activates mesolimbic dopaminergic pathway, increases extracellular concentrations of mesolimbic dopamine, DA released outside the synaptic cleft diffuses in the extracellular fluid from which it is slowly cleared as a result of reuptake and metabolism and activates its receptors[3].

Dopamine and Learning

Sudden and unexpected dopamine increases upon perceiving stimuli that predict rewards, a dominant mechanism of reward learning. Dopamine can achieve both invigorating and learning functions is counterintuitive, ex: in rats absence of dopamine in brain will not retrieve food a few inches away while starving. Dopamine is a teaching signal to encourage a future reward.

Dopamine was first identified to motivational function. Feeding and drinking deficit were observed when there was damage to nigrostriatal dopamine fibres, while forward locomotion reduced when damage was caused to the mesolimbic dopamine fibres,[4] this shows motivation to act and the satisfaction in finishing a work are the functions of dopamine[5].

Treating rats with neuroleptics did not learn or even if previously trained no longer pressed the lever or ran along an alley in search of rewards, this lead to the dopamine hypothesis of reinforcement, reward and hedonia[4].

Dopamine plays a major role in prefrontal cortex dependant learning and memory processing. Studies on primates, rodents and birds via fMRI (Functional magnetic resonance imaging) increasing or decreasing dopamine levels in prefrontal cortex of the brain effected the performance in working memory task (D1R)[6].

Reward and Declarative Memory Formation

Midbrain dopaminergic activity influences declarative memory formation. In a study of adults incentivised by money to remember visual scenes reported that anticipatory activation in the ventral tegmental area, nucleus accumbens, and hippocampus predicted remembering was greater with higher rewards. Activity in the hippocampus and ventral tegmental area correlated with participant's enhanced long-term memory for the subsequent scene. Dopamine secreted from midbrain plays an important role in learning associated with reward actions (declarative memory) formation.

Understanding of the reward-memory relationship can potentially inform education. Rewards may focus the attention of individuals more on some stimuli than others, which may make them more salient and so memorable[7].

Dopamine and Reward

In the brain, dopamine functions as a neurotransmitter brain has played a major role in the motivational component of reward-motivated behavior. Dopamine mediates pleasure in the brain. It is released during pleasurable situations and stimulates individual to do pleasurable activity or occupation, all goal directed behavior including seeking of food or water is learned, motivation is derived from the rewards experienced in the past[8]. Until the incentive motivational stimuli gets devalued through experience, the formed habit remains intact, this can result from continuous unrewarded trials, continued trials without appropriate drive state or in the presence of neuroleptics[9]. Dopamine boosts actions that motivates reward. It was found that increasing dopamine level by administering levodopa boosted striatal and substantia nigra/ventral tegmental representations associated with actions leading to reward[10].

Reward System and Food Intake

Reward system plays a major role in food intake, studies showed following the exposure to food, sweets in rats, there was an increase in the dopamine level in their nucleus accumbens, also found that the dopamine level was higher with increasing concentration of such stimuli. The hedonic properties of food can stimulate feeding even when energy requirements have been met, this causes weight gain and obesity.

Regulating food intake is complex which involves multiple levels of control through environmental cues and cognitive, sensory, metabolic, endocrine, and neural pathways. It involves a close interrelationship between two factors- homeostatic factor, which relates to the body's nutritional requirements and monitor available energy within the blood and fat stores and non-homeostatic factors, which is unrelated to nutritional or energy requirements. Imbalance in energy intake its expenditure leads to weight gain. The non-homeostatic mechanism is related to the brain's reward system and dopamine is found to be the major influencer of this factor over food intake. Addiction for food leads to overeating is determined by the subjective experience of an individual, certain amount of dopamine release and activation of the brain reward system are not necessary or sufficient conditions for addiction[11].

Dopamine and Neurodegenerative Disorders

Alzheimer's Disease is a neurodegenerative disorder with progressive cognitive decline and dementia. Cognitive decline during AD is due to pathogenic dysfunction of dopamine[12] its Pathophysiology is linked with the morphological and functional changes occurring in the monoaminergic system[13]. The degeneration of dopaminergic neurons deprives the hippocampus and cortex of its influence which could be the cause of cognitive decline symptoms in Alzheimer's disease. In addition extrapyramidal signs observed in 35-40% AD patients support the idea that there is dopamine neuron

degeneration[14]. Advancement in the age leads to changes in dopamine system which undergoes several physiological changes includes decreased release of dopamine, reduced expression of its receptors. Also, impairment of dopamine transmission which is observed in both elderly and AD patients causes apathy[15].

Dopamine is also associated with Parkinson's Disease (PD), it is reported that progressive loss of neuromelanin containing dopamine neurons in substantia nigra compacta of the ventral midbrain, dopamine depletion in the striatum induces motor symptoms like bradykinesia, tremor, rigidity and loss of postural control[16].

An accurate understanding of changes occurring in reward processing during illnesses. Abnormalities may occur in processing primary rewards (primary sensation), secondary rewards (or a change in the relative value of primary and secondary rewards, e.g., food may be more valued than money), reward-based learning, or reward-based decision-making. Impairments in reward during illnesses requires more direct assessment. Measures of reward-based learning and decision-making (gambling tasks) often require a broad range of intact functions and have variable results in degenerative illness, illustrated in Parkinson's disease, by testing simple sensory rewards, abstract rewards, anticipation of reward, and sensitivity to the timing of reward as well as corresponding punishments, understanding behavioral changes in each illness and the function of each component of the reward circuit will increase, inform the approach to address abnormal reward processing behaviors in neurodegenerative disease and application of this knowledge to other illnesses with aberrant sensitivity to reward or punishment, including gambling, drug and alcohol addiction.[17]

Reward and Depression

Reduced reward function was first mentioned as a diagnostic criterion for depression. There is a significant contribution of anhedonia to depression with reward processing being considered as a broad psychological construct, impaired behavior in depression such as lack of motivation, disinterest in learning, anhedonia has been characterized as dysfunction of the brain rewarding process. Preclinical and neuroimaging evidence suggested reward-related psychological processes are supported by dissociable brain systems.

Anhedonia diminished pleasure and/or reactivity to pleasurable stimuli is a core feature of depression that frequently persists after treatment, as a result, extensive effort has been directed towards characterizing the psychological and biological processes that mediate dysfunctional reward processing in depression. Reward processing can be parsed into sub-components that include motivation, reinforcement learning, and hedonic capacity, which, according to preclinical and neuroimaging evidence, involve partially dissociable brain systems. Recent findings indicate that behavioral impairments and neural abnormalities in depression vary across distinct reward-related constructs[18].

Reward System and Drug Addiction

Drugs of abuse acts by activating reward systems leading to experience an urgent need or powerful desire for drugs or addictive activities, trigger the release of dopamine which produces a pleasant sensation, serves to motivate to repeat these harmful behaviors. Characteristic addictive behaviors arise from brain's pleasure and rewards centers. All addictive drugs activities release varying amounts dopamine into the nucleus accumbens. However, Stimulant drugs include drugs such as cocaine and methamphetamine. Drugs such as alcohol or heroin, the brain's own opiate system (endorphins) also gets involved release the most. Although different addictions have different effects in the nucleus accumbens, they all activate the reward system. This is turn motivates to repeat those behaviors, even though they may be harmful. Dopamine involved in drug-seeking (craving) component of addiction. The opiate (endorphin), GABA, or glutamatergic systems may be more involved in pleasure-seeking aspect of addiction[19].

Effects of short term drug exposure on extracellular DA concentrations in human brain studied using PET and D2 DA receptor radioactive ligands, reveled the relationship between the effects of drugs on DA and their reinforcing properties in human brain (assessed by self-reports of "high" and "euphoria") was studied for the stimulant drugs methylphenidate and amphetamine. Methylphenidate, like cocaine, increases DA by blocking DA transporters, whereas amphetamine, like methamphetamine, increases DA by releasing it from the terminal via DA transporters. Imaging studies have corroborated the role of DA in the reinforcing effects of drugs of abuse in human beings and have extended traditional views of DA involvement in drug addiction. These findings suggest multicomponent strategies for the treatment of drug addiction that include strategies to decrease the reward value of the drug of choice and increase the reward value of nondrug reinforcers, weaken conditioned drug behaviors, weaken the motivational drive to take the drug, and strengthen frontal inhibitory and executive control[20].

CONCLUSION

Understanding human reward processes, requires much more integration of information at the molecular, cellular, systems and behavioral level. It is well known that dopamine along with other neurotransmitters play an important role in regulating hedonic state and also in reward related learning. Rewards produce hedonic consequences which initiates learning, motivational states such as hunger, sexual arousal increase the incentive salience of reward-related cues and reward itself, greater the hunger, greater will be the likelihood to show behavior that leads to obtaining memory despite all the obstacles that may arise.

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