A Meta-Analysis of Reward Function and Childhood Obesity

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Abstract: Obesity is a prevailing illness among children around the world. In the past decades, to unveil the truth about obesity, many researchers have conducted different studies on the influence of the reward function system on eating behaviors and obesity. Their reports all have similar statements that the dysfunctional neuronal reward function system has influenced many children worldwide and led to obesity. In addition, they generally state that the reward function brings more lousy eating behaviors and increases chances of obesity in children. In this case, the reward function system functions through the food reward, external reward, and the brain regions responsible for the reward processes. This review focuses on past research papers and analysis reviews related to the topics of reward function systems, obesity, and their relationships. Then, the conclusion can be made that the dysfunctional reward function system can affect childhood obesity in terms of the changing rewards, externalities, and brain functions. However, the brain is too complex for humans to learn all its facts. Thus, the existing limitations of technology and ethics limit the truth researchers can find about the reward function. As a result, more studies, even repeated studies, on different brain regions possibly responsible for the reward function should be conducted. Also, the development of human neurotechnology is urgently needed.

Keywords: Reward Function, Obesity, Food Reward, External Reward, Brain Regions, Prefrontal Cortex, Orbitofrontal Cortex.

1. Introduction

Obesity has become a serious problem among children worldwide, leading to long-term damage to both children’s physical and psychological states and having adverse effects on social and economic outcomes [1]. In the preceding decades, many researchers dedicated themselves to seeking the reasons for becoming obese. Among all the efforts around the topic, the emerging evidence of the effects of the reward function in the brain on children’s eating behaviors and obesity is essential and referential. In this case, many studies have investigated the reward function system. Therefore, many technologies are used, including MRI and fMRI, to take a deep look at human brain regions responsible for the reward function, such as the amygdala, prefrontal cortex, and orbitofrontal cortex. Although there are many restrictions to the studies and findings, they help unveil the facts about the reasons for obesity, the reward function, and the human brain.

This review describes how the reward function system influences children’s eating behaviors and obesity. In addition, it discusses the possible problems with past and current research on this topic, and parts of the most important and related studies will be analyzed. This review also proposes future
research directions and future improvements, which can help develop further and deeper investigations into the related topics of the reward function system and childhood obesity. Finally, it suggests some helpful information for patients with obesity, especially children and researchers, to conduct further research and obtain a more comprehensive understanding. In general, this review works as an analytical resource for people in need to address facts and problems within the topics of the reward function system and childhood obesity.

2. Literature Review

Based on Powers’s theory of the reward function system, the feedback function is crucial in processing the information [2]. The rewards are the environmental inputs that the sensory system receives and processes. Then the central comparator compares the reference signal with the sensor signal to form the error signal. As a result, the functioning of the reward influenced by the system is produced (Figure 1). In the case of eating and obesity, the reward in the system usually refers to the food people eat. Usually, the palatability of food is the critical fact that affects the information in the reward function system. Here, the “palatability” represents the hedonic reward provided by foods or fluids that are agreeable to the state of experiencing pleasure judged by the brain [3], which is not only the sweet and bitter taste but also the experiences of people who eat.

![Figure 1: Basic control-system unit of behavioral organization [4]](image)

According to some studies about reward and obesity, people who have obesity tend to see palatable food as more rewarding than those who do not have obesity [4]. In recent years, many research papers and reviews have proved that the reward function system and related brain regions affect eating behaviors and obesity. For instance, during adolescence, the development of limbic regions is faster than that of the prefrontal cortex [5]. Thus, the time difference leads to an imbalance between reward-
driven behaviors and top-down cognitive regulation [5]. In this case, there is enhanced sensitivity toward rewards and a reduced regulation of reward-related behaviors. As a result, there will be more overeating by the motivation of food rewards, including palatable foods linked with emotions and bingeing [5]. Moreover, the Shapiro et al. study proves that the “hedonically motivated disinhibited eating behaviors” are presented among children, and some neural regions have relationships with the disinhibited eating behaviors among children [6]. Thus, eating behaviors can change with neuronal changes, such as reward and connectivity network circuits, and this brings the risk of becoming obese [6].

3. How does the Reward Function System Influence Children’s Eating Behaviors and Obesity?

3.1. The Food Reward is Functioning

Regarding eating and obesity, the reward function system's reward refers to food and food-related information, dependent on the concept of "palatability." Moreover, according to Schachter's theory, eating habits respond to food taste and the external environment [7]. So, food is crucial to the reward function system in the brain.

When mentioning food, the first common concept should be the tastes—sweet and bitter. As a basis of the human reward function system, to achieve homeostasis inside the body, humans tend to eat sweet food other than bitter food, as sweet food is always associated with energy intake, such as carbohydrates [8]. As a result, one key factor in obesity development is that highly palatable food usually lacks nutrients [9]. According to related studies, highly palatable foods with high sugar and fat content increase food consumption as palatable foods promote eating behaviors and allow for the formation of new eating habits [10]. In addition, as proposed by Berridge, the reward function in the brain has two components: the liking system and the wanting system [3]. There is a famous study method called sham-feeding, which peels satiety away feedback away from the experiment subjects. In this case, the researchers can focus on subjects' initial taste affection and feedback in response to the tastes without the influence of satiety. It turns out that the subjects are sensitive to the taste stimuli and the effects of satiety are small in the study [11]. In this case, the hedonically positive taste (sweet) can increase the ingestion of food time, and the hedonically negative taste (bitter) can decrease the time of consistent eating [11]. When eating behaviors and time are increased, the energy intake is also increased to a level beyond the homeostatic need, resulting in overweight and obesity.

However, there are not enough sham feeding studies on reward function systems and obesity, and the data for relationships between food reward and obesity is highly limited. Similarly, the studies on relationships between other food rewards and obesity are insufficient. This field of study is still in an early stage, and more future studies will help form a more substantial understanding of the topics. With the existing studies, the ideas will have more discussion and better proof in the future.

3.2. The External Reward Information is Functioning

Powers’s theory points out that the reward function system’s reward refers to the tastes and external factors related to the food, including the environment and experiences [2].

The environment and experiences work together with the tastes to form the palatability of food. For example, food consumption depends on the state when eating, like stress and anxiety level, and the convenience of obtaining the food determines the level of wanting the food [12]. Additionally, the affection for food can be learned through positive intake stimulations, such as classical conditioning. Classical conditioning offers a change in behaviors in response to specific external stimuli. In this case, under the condition of a flavor pairing with intragastric glucose infusions,
experiment subjects can even develop their preference for hedonically negative foods, such as bitter foods [13].

Moreover, the advertisements and product names can influence eating in associative learning. They can change their eating habits and allow more eating behaviors without the effect of satiety [14]. Therefore, when eating behaviors are influenced by external reward information, obesity can develop.

These existing studies provide a comprehensive overview of how external factors affect obesity. Nonetheless, massive external factors might affect the reward function system, which is challenging to manage and manipulate. Thus, though there is much evidence pointing to the significant influence of external rewards on obesity, future research still needs a deeper understanding of the reward function system.

3.3. The Brain is Functioning

In addition to the food and environmental rewards affecting the reward function system, the brain is also a critical factor as it processes all the information.

In order to verify the Berridge theory of the liking and wanting system, selectively activating or lesioning the brain regions provides a way of better analyzing the system [3]. The crucial lesion-induced aversion sites in the brain are the lateral hypothalamus (LH) and ventral pallidum (VP), which are responsible for the aphagia and aversion reactions to rewards [3]. When the damage is on the LH, it leads to aphagia but not necessarily aversion. Instead, aversion forms only when the damage is to the VP [3]. Then, with electric stimulation of the lateral hypothalamus (EHLS), the brain shows excessive wanting behaviors and largely reduced liking behaviors [3]. Moreover, the 6-OHDA lesions of the mesotelencephalic dopamine system eliminate all wanting aspects, but the liking system remains normal [3].

Furthermore, with knowledge of how the brain is functioning in the reward function system, further research on the brain regions affecting obesity suggests that the prefrontal cortex (PFC) has many contributions [15]. For instance, weak relationships between the dorsolateral PFC (dlPFC) and ventromedial PFC (vmPFC) lead to a reduction in the ability of the dlPFC to downregulate taste attributes in the vmPFC [15]. In this case, individuals will tend to select foods based on taste instead of healthiness [16]. Successful dietary self-regulation also relates to the dlPFC and vmPFC, that is, the lower regional grey matter volumes will reduce the regulation success [17]. Furthermore, eating food can interfere with behavioral and cognitive regulation. Animal studies point out that rats fed with high-sucrose or high-fat show a significant reduction in GABAergic interneurons and parvalbumin neurons in the PFC and hippocampus [18]. Therefore, the regulation of behavioral and cognitive functions is damaged as those neurons are essential in those regulation functions. As a result, eating behaviors can be out of control, leading to obesity [18].

Similarly, other dysfunctional reward system brain regions can also lead to obesity. For instance, when weaker functional connectivity occurs between the ventral striatum and the orbitofrontal cortex, the higher BMI the individual will get [19]. Moreover, the rising integration of the sensorimotor cortex into superior parietal areas will also lead to higher BMI as the corresponding bodily self-consciousness reduction [19]. Furthermore, obese children’s orbitofrontal cortex and amygdala region change after becoming obese [19].

With existing studies, it is essential to test the validity of the results repeatedly to find out the exact trigger and location in the brain corresponding to the reward function system and obesity. As the brain is too complex for technology nowadays to find out all the truths, more research and remarkable neurotechnology improvement are eagerly required. Moreover, animal studies, especially of rodents like rats, can provide valuable and instructive information for future research in understanding the brain. However, human beings and animals are different. The data from animal research can only be referenced. In the future, better neurotechnology and more research will help build a more substantial
base for this research area. Finally, some deep-brain technologies cannot be applied to human beings due to ethical issues. Therefore, the study of the human brain is restricted to the extent of the understanding of animal brains.

4. Conclusion

In general, this topic has progressed over several decades. This review analyzes part of the research on reward function systems and obesity in terms of different functioning factors, including food reward, external reward, and brain regions. Therefore, the conclusion can be made that obesity is due to the dysfunctional reward function system. Nonetheless, the research cited in this review has many limitations, including ethical issues, restricted technology, and generalizability. In this case, more research and reviews should be conducted in the future to perfect the understanding of this topic and even human brains. In addition, better neurotechnology is needed to investigate the brain’s facts for animals and humans.

References


