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Review Article

Reward Sensitivity: A Narrative Review

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Abstract

Behavioral effects of reward sensitivity include its increase across development, school burnout, internet gaming, internet addiction, cyber–aggression, depression and drinking. Physical effects include increased physical activity, decreased heart rate reactivity, blood pressure and connectivity as well as increased cortico-striatal function and a blunted relationship between substance use disorder and striatal activity. Potential underlying biological mechanisms are related to dopamine, the hippocampus, the cerebellum and connectivity between the dorsal motor nucleus and the striatum. Interventions have included behavioral activation, amplifying positivity, mindfulness, creating mental images, transcranial electrical stimulation, and administration of dopamine and naltrexone. Methodological limitations include prevalent sampling of university students, the cross-sectional design of the research, and the almost exclusively negative effects.

1. Introduction

Reward sensitivity has been defined as “how strongly a person notices, wants and enjoys positive rewards like food, money or praise. It’s a core part of personality driven by brain chemicals like dopamine”.

The literature search for this narrative review involved entering the terms reward sensitivity and the years 2024 to 2026 in PubMed and PsycINFO. Exclusion criteria included proposals, case studies and non-English language papers. This narrative review summarizes 28 papers on reward sensitivity. The current reward sensitivity literature can be categorized as effects of reward sensitivity, potential underlying biological mechanisms and interventions. Most of the papers are focused on behavioral and physical effects of reward sensitivity (14 papers), only a few researchers review potential underlying biological mechanisms (5 papers) and several researchers (9) have reported data on interventions to reduce reward sensitivity.

2. Effects of Reward Sensitivity

2.1. Behavioral Effects

The papers on behavioral effects of reward sensitivity include an increase in its development across childhood and adolescence, school burnout, internet gaming, internet addiction, cyber – aggression, depression and drinking. As suggested by these topics, most of the researchers have recruited young people.

In research measuring children's reward and punishment sensitivity, the Contingency Response Rating Scale was used for children 5 to 12 years of age (N= 195) [1]. These researchers showed that children's **reward and punishment sensitivity increased across childhood**. This increase may simply relate to the children’s increasing experience with reward and punishment that in turn contributed to their increased reward sensitivity.

Another research group suggested that reward sensitivity **rapidly increased across adolescence** (N= 166 participants 9–29 years-old) [2]. Both reward sensitivity and executive function increased rapidly in this sample and was dominant by mid-adolescence. The increase in executive function likely contributed to the increased reward sensitivity as increased executive function suggests greater control by the brain on working memory, enabling greater memory of rewards.

Internet problems have also been related to reward sensitivity. In a study **on internet gaming disorder**, those with the disorder (N=21) versus recreational game users (N= 23) had greater cravings for rewards [3]. These results were not surprising given that those with internet gaming disorder had experienced more rewards. The authors suggested that intrinsic reward sensitivity was driving internet gaming disorder through striatal-thalamic hyper-synchronization. Given that the striatum and the thalamus have both been involved in reward sensitivity, it is not surprising that they mediated the relationship between reward sensitivity and internet gaming disorder. However, these hyper-synchronization data were derived from a sample that was small likely because of the expense of fMRIs.

In research on **internet addiction symptoms** and reward sensitivity (N = 310 internet users, 18–65 years old), different mediators were noted for the relationship between reward sensitivity and internet addiction (Lyvers et al, 2025). These authors suggested that the relationship between reward sensitivity and internet addiction was mediated by frontal dysfunction, executive dysfunction, and negative mood. These multiple negative mediators were clearly interrelated, and the combination of their negative effects would significantly contribute to internet addiction.

Reward sensitivity has also been a risk factor for **depressive symptoms**. This relationship was noted in a sample of adolescents (N=320) [4]. A similar relationship was reported for a sample of adults who experienced a stroke (N=32) versus healthy controls (N= 22) [5]. In this study, those who had experienced a stroke had lower reward sensitivity, greater depression, and less connectivity. Because this was a cross – sectional study, it's not clear whether the stroke contributed to lower reward sensitivity, greater depression, and less connectivity or whether those factors contributed to lower reward sensitivity. Nonetheless, relationships between lower reward sensitivity, greater depression and less connectivity would be expected.

Given the relationship between reward sensitivity and depression, it's not surprising that reward sensitivity has also been associated with the **frequency and quantity of drinking** in young adults (N= 150, 18–20-year-old) [6]. Depression and drinking have been highly correlated in many studies.

Reward addiction (a more chronic form of reward sensitivity) has been studied in Polish and Turkish students [7]. The students' reward sensitivity, not surprisingly, led to **school burnout and cyber – aggression**. School burnout likely led to increased use of the internet for completing assignments which would result in more internet contact with classmates as well as conflict with them and aggression toward them. The relationship between school burnout and cyber-aggression was observed more frequently in the Polish versus the Turkish students, possibly because of less strict parenting in the Polish culture.

3. Physical Effects

Several physical effects have been reported in this current literature on reward sensitivity. They include **increased physical activity** as well as decreased heart rate reactivity, blood pressure and connectivity, increased cortico-striatal function, and a blunted relationship between substance use disorder and striatal activity.

In a study entitled " Parsing reward sensitivity reveals distinct relationships with energy intake, metabolic markers, physical activity, and fitness", a sample of young people was recruited (N=1238 individuals who were 15, 18 and 25 years-old) [8]. Openness to reward was associated with increased physical activity as well as **decreased blood pressure, glucose levels, insulin and cholesterol**. In contrast, insatiability from reward was positively associated with triglycerides and negatively related to energy intake and cardiorespiratory fitness. Increased physical activity may relate to more active physical pursuit of reward. The increased physical activity would, not surprisingly, lead to decreased blood pressure, glucose levels, insulin and cholesterol which has been reported in many studies. A similar reward sensitivity/cardiovascular relationship was reported for another sample in which those with high reward sensitivity had **lower heart rate reactivity,** suggesting again that reward sensitivity was having negative effects [9].

Reward sensitivity has also been related to **greater connectivity** in a study already described [5]. Further, reward sensitivity has **enhanced cortico-striatal function** (reward processing) based on fMRIs of adults playing a card guessing game in which correct or incorrect guesses resulted in monetary gains and losses (N= 45) [10]. Increased reward sensitivity and increased ventral striatal activation occurred when experiencing the game with a close friend. The incentives in this research were likely the potential monetary gains that were in turn reinforced by experiencing them with a close friend.

4. Potential Underlying Biological Mechanisms

Several potential underlying biological mechanisms have been the focus of research in this current literature on reward sensitivity. The mechanisms involve **dopamine**, the hippocampus, the cerebellum and **connectivity between the dorsal motor nucleus and the striatum.**

In a systematic review and meta-analysis entitled "Differential association of dopamine and serotonin with reward and punishment", dopamine was associated with reward sensitivity and serotonin was related to punishment learning (N=2291 receiving dopamine and 1491 receiving serotonin) [11]. Dopamine, being an activating neurotransmitter, not surprisingly, was related to reward sensitivity. In contrast, serotonin, as an antidepressant neurotransmitter, might be expected to relate to a negative experience like punishment avoidance. In another paper, the results are also given in the title "In vivo structural connectivity of the reward system along the hippocampal long axis" (M=628 fMRIs) [12]. Reward sensitivity would naturally relate to the hippocampus that is involved with emotions.

In a paper entitled "Consensus paper: **cerebellum** and reward", the cerebellum was said to be "a master – piece of reward mechanisms together with the striatum ventral tegmental area and prefrontal cortex" (Mayo et al, 2024). These three areas are intrinsically related to reward sensitivity. Recent research suggests that both the prefrontal cortex and the cerebellum are involved with emotional control. The ventral striatum and ventral tegmental area have been noted to "work together as the core engine of the brain's reward circuit".

In a study entitled " Trait reward sensitivity and behavioral motivation are associated with connectivity between the dorsal motor nucleus and the striatum during anticipation", the participants (N=46) completed the Monetary Incentive Delay Task and fMRIs were recorded [13]. In this research, reward sensitivity, as the title suggests, was associated with connectivity between the dorsal motor nucleus and the striatum. The striatum has been involved in reward processing, as already noted, and the dorsal motor nucleus influences heart rate which slows during reward sensitivity.

5. Intervention Studies

Several intervention studies have appeared in this literature on reward sensitivity. They include behavioral activation, amplifying positivity, mindfulness, creating mental images, transcranial electrical stimulation, and administration of dopamine and **naltrexone**.

Behavioral activation as an intervention for improving reward sensitivity has appeared in two studies by the same research group. In the first study (N=126), two weeks of online intervention comparing **behavioral activation versus mindfulness and gratitude** resulted in positive changes for depression expectations and a long-term increase occurred in reward sensitivity [14]. Decreased anhedonia and depression were stable over four months.

In the other behavioral activation study, behavioral activation was compared to mindfulness and gratitude, and a combination of both interventions [14]. In this randomized controlled trial (N=336), videos and daily exercises were given for the three groups. All three groups experienced a decrease in depression and an increase in reward sensitivity.

In a study entitled "Amplification of positivity treatment for anxiety and depression: a randomized experimental therapeutic trial targeting social reward sensitivity to enhance social connectedness", the results are given in the title [15]. The sample of adults who endorsed anxiety or depression (N= 68), received 5 or 10 sessions of **amplified positivity**, or participated in a waitlist control group. The intervention was comprised of increasing positive thoughts, behaviors, and emotions. Surprisingly, greater striatal and social connectedness increases were noted for 5 versus 10 sessions. The participants may have become bored after 5 sessions.

Mental images were provided in another intervention study [16]. In this research, participants who were screened for reward sensitivity (N = 95) were asked to create mental images of rewarding events for one week. A compliance rate of 85% was reported. In this sample, decreases were noted in both stress and depression.

In a paper entitled "Cerebellar stimulation modulates reward processing", **transcranial direct current stimulation** was provided for 20-minutes [17]. The participants (N=63) were given the Monetary Incentive Delay Scale, the Effort Expenditure for Reward Scale, and the Probabilistic Stimulus Selection Task. Active stimulation led to increased reward sensitivity.

Dopamine treatment is usually provided patients with Parkinson's. However, if they have impaired reward sensitivity, they are unresponsive to dopamine treatment, and in this sample (N= 62 patients), the participants with Parkinson's required non-dopamine treatment [18].

In a review of 36 studies on the efficacy of naltrexone as the treatment for alcohol dependent patients, **naltrexone** decreased reward sensitivity in 9 of 10 studies [19]. Naltrexone effectively decreased sweet-liking and alcohol use disorder.

6. Methodological Limitations of this Literature

Several methodological limitations can be cited for this current literature on reward sensitivity. They include the prevalent sampling of young people, the cross-sectional design of the research, and the almost exclusively negative effects.

Most of the samples were comprised of young people which have limited the **generalizability of the data** to other age groups. Most of the assessments were self-report/surveys that were conducted in **cross-sectional rather than longitudinal studies**. This study design and recruitment have limited the data analyses to correlations rather than determining causal relationships. Regression analyses or structural equation modeling would have suggested the relative importance of the predictor variables.

Exclusively negative effects were reported, and those were typically behavioral and physical effects. Only two studies focused on **potential underlying biological mechanisms** and they were focused on different parts of the brain and different functions. Most frequently the results were given in the titles of the studies. Only 7 intervention studies appeared in this current literature on reward sensitivity which was surprising given the negative reward sensitivity effects. Despite these methodological limitations, this current literature on reward sensitivity was informative and has suggested future directions for research on reward sensitivity.

Effects	First Authors
Increase in reward sensitivity across development	Waschbusch, Wun
Internet gaming disorder	Li
Internet addiction symptoms	Lyers
Depressive symptoms	Maddox, Sanchez-Kuhn
Frequent drinking	Lopez-Vergara
Reward addiction	Katarzyna

Table 1: Behavioral Effects of Reward Sensitivity (and First Authors)

Effects	First Authors
Increased physical activity	Kiive
Lower heart rate reactivity	Sozer
Greater connectivity	Sanchez-Kuhn
Enhanced cortico-striatal function	Smith

Table 2: Physical Effects of Reward Sensitivity (and First Authors)

Mechanisms	First Authors
Dopamine	Mkrtchian
Cerebellum	Mayo
Connectivity between dorsal motor nucleus & striatum	Wyngaarden

Table 3: Potential Underlying Biological Mechanisms (and First Authors)

Interventions	First Authors
Behavioral activation	Potsch
Amplification of positivity	Taylor
Mental images	Marciniak
Transcranial direct current stimulation	Wang
Dopamine	Costello
Naltrexone	Amsterdam

Table 4: Intervention Studies (and First Authors)

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