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PSYCHOSIS (A AHMED, SECTION EDITOR)

Learning and Motivation for Rewards in Schizophrenia: Implications for Behavioral Rehabilitation

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Abstract

Purpose of Review Impaired reward processing and amotivation are well documented in schizophrenia. We aim to review the current state of neuroimaging and behavioral research addressing components of motivational deficits in this complex and impairing syndrome. Evidence will be integrated to inform the ongoing development of effective strategies for behavioral rehabilitation.

Recent Findings While striatal dopamine and aberrant reward prediction errors have been connected to amotivation in schizophrenia, they are not sufficiently full explanations of reward processing impairments. Frontal dysfunction and associated cognitive control deficits also have evidenced involvement in atypical reward prediction, learning, and valuation. Ongoing work supports the utility of interventions (e.g., cognitive remediation) for improved motivation for rewards.

Summary Within schizophrenia, greater negative symptoms (avolition and anhedonia) are associated with poorer functioning and more severely impaired reward processing. Utilizing behavioral interventions such as cognitive remediation and social cognition training hold promise for rehabilitation and increased community integration.

Keywords Schizophrenia · Psychosis · Reward processing · Motivation · Negative symptoms · Behavioral rehabilitation

Introduction

Impairments in motivation, associated with deficits in reward processing, are supported as a central component of schizophrenia (SZ) [1]. Reduced motivation and goal-oriented behavior is a main component of negative symptoms, which are associated with treatment-resistant functional impairment in people with schizophrenia (PSZ) [2–5]. Evidence concerning the latent structure of negative symptoms supports five core domains: anhedonia, avolition, asociality, blunted affect, and alogia [6]. Additional evidence of a higher-order dimension—motivation and pleasure (MAP)—which influences anhedonia and avolition suggests possible pathophysiological links between the two negative symptom dimensions [6]. Avolition and anhedonia are associated with reward processing deficits. Focusing on diminished expressivity (e.g., blunted affect, alogia) has been evidenced as comparatively less critical in recovery models [2, 7]. Research supports deficits in the anticipation of pleasure, rather than the capacity to experience pleasure, as a factor underlying motivational impairment in PSZ [2, 8–10]. While treating motivational abnormalities in PSZ has been evidenced to benefit functional outcomes, neural and behavioral mechanisms underlying these deficits have yet to be fully understood [11].

The construct of motivation is complex, incorporating direction (i.e., approach-avoidance) and degree of energy (i.e., persistence and vigor) in relation to the initiation (or absence) of goal-directed behavior [1]. Motivation to engage in or refrain from activity is related to the experience of salient rewards and aversive events. Heuristic models of motivation connect anticipated and experienced rewards with generated and maintained plans for action that are necessary to receive rewards [12]. Associations among rewarding stimuli, actions,

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and outcomes must be learned (i.e., reward learning) for behavior to be initiated. Additionally, the perceived degree of effort needed to attain rewards influences motivation to act.

The Research Domain Criteria Initiative (RDoC) includes a "positive valence" systems domain, encompassing constructs underlying impairments in motivation and hedonic capacity across psychopathology [11, 13]. The positive valence systems domain includes reward responsiveness, reward learning, and reward valuation [13]. Table 1 details components of the positive valence systems domain, along with associated brain regions and behavioral assessments utilized to capture performance relative to these constructs. Aberrant functioning in SZ has been evidenced across reward processing constructs [11]. Additionally, evidence supports the presence of impairment in reward processing and motivation in PSZ across incentive types (e.g., money, food) [23, 24].

Herein, recent neuroimaging and behavioral research focused on elucidating reward processing impairments in PSZ will be reviewed. Research examining positive valence systems constructs will be emphasized. Better understanding of the mechanisms underlying reward processing and motivation deficits can inform the composition of evidence-based intervention strategies for behavioral rehabilitation that hold promise for the improvement of daily functioning for PSZ. As the association between amotivation and impaired functioning in PSZ is well established [25], focus placed on translating evidence for mechanisms underlying reward processing deficits to inform behavioral rehabilitation could greatly improve the lives of PSZ.

Neural Underpinnings of Reward and Motivation in Schizophrenia

The frontal-striatal network has an evidenced role in reward processing, motivation, and associated deficits [26]. Brain regions implicated in reward processing deficits include the anterior cingulate cortex (ACC), dorsolateral prefrontal cortex (dlPFC), ventrolateral prefrontal cortex (vlPFC), orbitofrontal cortex (OFC), basal ganglia (BG), and caudate nucleus [11, 27]. Basic neuroscience supports the existence of two complimentary neural systems that underlie reward processing and utilize reward prediction errors (RPEs) in doing so [2, 28]. One such system underlies gradual reinforcement learning is mediated by the BG. Another system involves the updating of mental reward representations and associated possible

Table 1 Reward constructs, neural underpinnings of reward processing, and behavioral assessments

RDoC positive valence system constructs	Positive valence system subconstructs	Neural mechanisms	Behavioral assessments
Reward responsiveness	• Reward anticipation/prediction ("wanting," anticipatory pleasure) • Initial response to reward (reward receipt) • Reward satiation ("liking," consummatory pleasure)	• Ventral and dorsal striatum • Frontal striatal network • Basal ganglia (BG) • Anterior cingulate cortex (ACC) • Orbitofrontal cortex (OFC) • Ventral tegmental area (VTA) • Substantia nigra • Dopamine (DA) • Opioid signaling • GABA	• Monetary Incentive Delay Task (MID) [14] • Temporal Experience of Pleasure Scale (TEPS) [15] • Reward Representation Activation and Maintenance Task (RRAM) [16•]
Reward learning	• Probabilistic and reinforcement learning • Reward prediction error • Habit formation	• Ventral and dorsal striatum • Dorsolateral prefrontal cortex (dlPFC) • ACC • OFC • VTA • Substantia nigra • BG • DA	• Iowa Gambling Task (IGT) [17, 18] • Probabilistic Learning Task (PLT) [19•] • Progressive Ratio Breakpoint Task (PRBT) [19•] • Salience Attribution Test (SAT) [20]
Reward valuation	• Effort/cost computation • Reward representation (employing working memory)	• ACC • OFC • Nucleus accumbens • DA	• RRAM • SAT • Progressive Ratio Task (PRT) • PRBT • Effort-Cost Computation Task (ECCT) [21] • Effort-Expenditure for Rewards Task (EEfRT) [22]

responses relative to reinforcement contingencies. This system underlies rapid learning and is mediated by the PFC (specifically, the OFC). The dlPFC has been connected to the generation and completion of goal-directed behavior in effort to receive rewards.

An association between reward processing deficits in SZ and dysfunctional signaling of dopamine (DA) has been well documented [29]. DA neurons calculate RPEs themselves, playing an active role in assessing the difference between reward that is expected and reward that is received [30]. Typically, increases in DA are associated with positive RPEs (i.e., indicating better-than-expected outcomes), and decreases in DA are associated with negative RPEs (i.e., indicating worse-than-expected outcomes) [2]. DA neurons in the midbrain [e.g., substantia nigra, ventral tegmental area (VTA)] that project to the ventral- and dorsal-striatum mediate reward prediction and implicit reinforcement learning [11, 31]. Explicit reinforcement learning, involving value representations and cognitive control, has been associated with the OFC and the parietal and dorsal-frontal cortices [11]. Reward valuation (including cost/benefit analyses of rewards) involves the dorsal ACC (dACC) and DA activity in the nucleus accumbens. A non-clinical investigation evidenced a positive association between midbrain D_2 receptor availability and neural activity in the left ventral-striatum during a reward valuation task [32]. In a recent longitudinal investigation, first-episode PSZ determined to be treatment responders evidenced a correlation between BOLD signal improvement in the caudate nucleus captured during reward anticipation and D_2 receptor occupancy, suggesting that symptom reduction relates to improvement of reward system abnormalities via antipsychotics [33•].

While much research on reward processing has focused on DA, the influence of a range neuropeptides is being explored. Gamma-aminobutyric acid (GABA) and opioid signaling have also been implicated in reward processing and associated deficits [34, 35]. Additionally, support for the role of oxytocin in increasing motivation for social reward in PSZ has been evidenced [36]. Though, an investigation of the effects of oxytocin on vigor (i.e., motivation intensity) in PSZ did not find a significant association between oxytocin and increased vigor during social encouragement [37].

Recent Updates in Neuroscience and Behavioral Research

Building off of identified neural circuitry underlying reward processing and motivation, ongoing research adds to our understanding of these systems and how they are impaired in SZ. As the literature on reward processing in SZ is vast, details of research conducted throughout the past 5 years are highlighted.

Overall Reward Processes

The negative symptom dimension of MAP (alternatively referred to as "apathy") has been differentiated from diminished expressivity and has been differentially associated with functional impairment. Additionally, research points to different neural mechanisms underlying these dimensions [7]. As striatal dysfunction is proposed to underly negative symptoms, a recent investigation examined resting-state cerebral blood flow (rCBF) in the striatum of PSZ. Severity of apathy, but not diminished expressivity, was found to be associated with increased rCBF in the ventral- and dorsal-striatum [7]. However, overall rCBF in the ventral- and dorsal-striatum was not found to significantly differ between PSZ and controls. Despite the evidenced association between apathy and striatal hypoactivation, the relationship between reduced striatal volume and apathy is less clear [4].

It is important to identify how components of reward processing function in at-risk populations. A recent fMRI examination evidenced no significant brain activation differences between clinical high risk (CHR) youth and controls during reward anticipation and receipt, suggesting motivation for reward may be spared at this stage [38]. However, CHR youth evidenced reward learning impairments and exhibited blunted RPE signals in the ventral-striatum, dACC, and ventromedial-PFC (vmPFC). Another study examining psychotic like experiences (PLEs) in a university sample evidenced a significant association between higher PLEs and greater effort expenditure regardless of reward probability and value, suggesting that hypersensitivity to reward may be present in less severe presentations of the psychotic continuum [39•].

Reward Anticipation/Prediction

Anticipation or prediction of reward refers to the "wanting" component of reward processing (i.e., anticipatory pleasure). Reduced activity in the ventral-striatal network of PSZ has been evidenced relative to reward-predictive cues [11, 40]. This reduction in ventral-striatal activity has been found in both medicated and unmedicated PSZ; though, some research suggests that these deficits are not seen in patients on atypical antipsychotics and those in the prodrome [41, 42]. Mechanisms underlying deficits in reward anticipation may extend beyond ventral striatal DA, implicating cognitive control mechanisms such as representation and maintenance of reward and working memory [43, 44]. A recent meta-analysis of fMRI studies examining appetitive anticipation signals revealed significant reduction in activation within the frontal-striatal network in PSZ compared with controls [45•]. Notably, reduced activation was evidenced in the cingulate/paracingulate gyri and left-striatum of PSZ, extending reward anticipation deficits beyond the ventral-striatum. These findings remained significant when controlling for psychotic

symptom severity and antipsychotics. An examination of reward representation activation and maintenance in PSZ was conducted in effort to assess mechanisms underlying anticipatory anhedonia [16•]. When anticipating rewards with high probability in situations wherein representation activation and maintenance were difficult to achieve, PSZ exhibited lower subjective arousal compared with controls, possibly accounting for anticipatory anhedonia. Another investigation evidenced attenuated anticipatory, but not consummatory, pleasure for rewards in PSZ but not in people with high schizotypy [8•]. This suggests that differences between anticipatory and consummatory pleasure may, in part, account for the anhedonia/emotion paradox in SZ.

Reduced activity in the ACC and insula in PSZ has also been evidenced during prediction of reward [46, 47]. Recent findings suggest that antipsychotics may normalize connectivity between the ACC and insula evident during reward prediction [48]. In a study of reward anticipation deficits relative to incentive type, PSZ evidenced increased reaction times compared with controls related to both monetary and social stimuli [49]. Though, PSZ exhibited improved performance relative to reward increases, suggesting that patients who are treated with atypical antipsychotics may have capacity to anticipate monetary and social awards which could be leveraged in intervention strategies.

Initial Response to Reward/Reward Receipt

Hypoactivation of the medial-frontal-cortex and ACC has been evidenced during expected reward receipt in PSZ [50]. Though, some evidence points to reward receipts being spared in motivational deficits [51]. Notably, this research has focused on predictable rewards. In a cross-diagnostic examination of unexpected reward receipt, reduced activation compared with controls was evidenced in the OFC, ventral-striatum, inferior-temporal gyrus, and occipital cortex in both SZ and depression [52]. Greater hypoactivation of the medial-PFC relative to receipt of unexpected reward was evidenced in PSZ. PSZ, but not depression, showed abnormal activation in the lateral parietal cortex. As these regions have been connected to the signaling of surprising outcomes and are mediated by DA, it is notable that dysfunction was evidenced in SZ but not depression [53, 54].

Reward Learning and Reward Prediction Errors

Reward anticipation is theorized to be an underlying component of implicit and explicit reward (or reinforcement) learning [11, 55, 56]. Aberrations in reward learning mechanisms have been associated with abnormal RPEs [30]. Figure 1 offers a theoretical depiction of reward learning and RPEs. Reinforcement learning, involving integration of information regarding the probability of rewards and punishments, is mediated by dopaminergic projections in the midbrain to the dorsal and ventral BG [11, 57]. The strength of DA response to rewards has been connected to reward predictability. Typically, rewards received that are unexpected or greater than predicted are associated with greater DA firing (i.e., positive RPE) and the reduction or absence of predicted rewards is associated with reduced DA firing (i.e., negative RPE) [28, 31, 58]. RPEs in SZ have been associated with bidirectionally altered DA activity in the ventral- and dorsal-striatum [11, 59, 60]. Negative associations have been evidenced between RPE magnitude in the BG and clinical symptoms (i.e., avolition) [61]. It has been suggested that first-generation antipsychotics may reduce RPE responses [41]. Though, RPE reduction has also been evidenced in unmedicated PSZ [59, 60]. Recent evidence suggests that RPE signaling during reinforcement learning is intact in medicated PSZ, despite worse task performance compared with controls [62, 63].

While much research evidences reinforcement learning deficits in PSZ [64, 65], some have not found such associations. A recent examination differentiating deficits related to causal (action-outcome) learning and reinforcement learning evidenced insensitivity to reward-value in PSZ but did not evidence reinforcement learning deficits [66]. Additionally, evidence supports relatively intact reinforcement learning in PSZ during simple and implicit learning tasks [67]. This pattern of findings suggests that striatal DA deficits cannot sufficiently explain impairments in reward prediction. Beyond dopaminergic explanations, cognitive control has been implicated in explicit learning and reward representation impairments [56]. PSZ evidence deficits in explicit reinforcement learning during more complex tasks involving sustained representations of rewards [68, 69]. Impairment in explicit reinforcement learning has been associated with greater negative symptoms [67]. Working memory impairments in PSZ may contribute to explicit reinforcement learning deficits requiring representations of stimuli-reward relationships [67, 70]. A protocol designed to identify separate contributions of working memory and reinforcement learning on performance in medicated PSZ evidenced that impairments in learning were associated with working memory deficits, while reinforcement learning was spared [71].

It has been suggested that deficits in reinforcement learning are not specifically associated with RPE signaling, but rather the impact of RPE signaling on learning and behavior (i.e., learning rate modulation) due to dysfunction in the ACC and dorsomedial PFC (dmPFC) [62•]. An investigation utilizing a probabilistic learning paradigm with shifting contingencies evidenced an association between learning rate modulation and decreased connectivity between the ventral-striatum and dmPFC. This relationship was stronger in PSZ with more severe motivational deficits. This suggests that impaired learning rate modulation could reduce one's capacity to alter task-relevant behavior when met with unexpected outcomes.

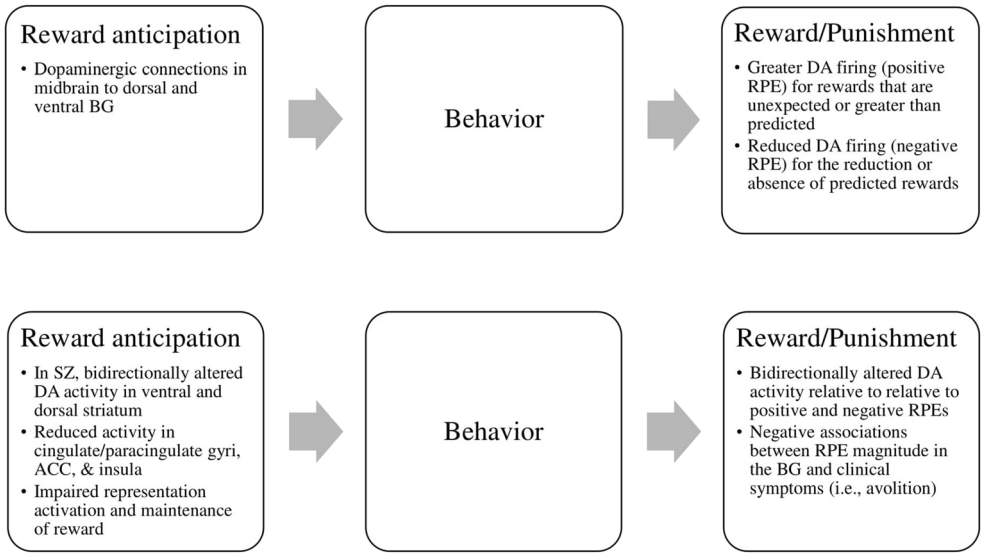

Fig. 1 Simplified theoretical diagrams of reward learning and prediction errors in neurotypical individuals (top) and individuals with schizophrenia (bottom)

Evidence suggests that PSZ are worse at learning to predict rewards than learning to avoid losses and have reduced capacity to alter behavior in response to rewards [56, 64, 72–74]. However, some investigations have not evidenced this pattern [67]. Notably, research concerning response to rewards and losses has largely been conducted with medicated patients, and it has been argued that this differential performance relative to reward prediction and loss avoidance could be influenced by the effects of D_2 receptor blockades. To assess learning of rewards and losses in PSZ without the potential confound of antipsychotics, an fMRI comparison of unmedicated PSZ and controls utilized a probabilistic learning task [75]. PSZ evidenced attenuated RPE response in regions including the striatum, medial temporal lobe, and medial PFC when learning to predict rewards but not when learning to avoid losses, suggesting that motivational context (reward vs. loss avoidance) modulates RPE response independent of medication effects. As many PSZ exhibit treatment refractory symptoms, mechanistic explanations beyond the effects of DA are suggested to underlie symptoms of SZ and associated deficits. In a fMRI comparison of treatment-refractory and non-refractory PSZ, reinforcement learning impairment was evidenced in both groups relative to controls [76]. Though, treatment-refractory and non-refractory PSZ may differ in patterns of neural mechanisms underlying reinforcement learning, with antipsychotic response related to initial DA dysfunction and treatment resistance related to dysfunction in other neurotransmitters (e.g., glutamate). Another fMRI investigation evidenced reduced learning from positive feedback by PSZ [65]. Though, reduced learning from positive feedback was not related to severity of anhedonia and avolition, and RPE activation was similar between PSZ and controls during task feedback. PSZ did evidence lesser cognitive control network activation during early learning compared with controls. PSZ with greater avolition and anhedonia exhibited reduced response in the dlPFC and caudate relative to positive feedback. This suggests that anhedonia and avolition are related to deficits in higher-level cortical learning and reduced cognitive control.

As studies have examined potential differences among incentive types in reward anticipation, others have assessed different incentive types in reward learning. An examination of social and non-social reward learning evidenced impairments relative to each incentive type in both PSZ and their first-degree relatives but not in controls [24•]. This suggests that PSZ and their first-degree relatives experience a general reduction in reward sensitivity reflective of familial vulnerability rather than illness process.

Reward Valuation

Reward valuation refers to assessing the benefits and probability of possible reward outcomes (i.e., effort-cost computation), which includes the activation and maintenance of reward representation, implicating working memory [14]. Reward valuation impairment in PSZ has been evidenced across incentive types [15, 16, 77]. Investigations utilizing measures that require participants to update representations of rewards and punishments (i.e., value representations), such as the Iowa Gambling Task (IGT), have evidenced impaired performance by PSZ [78]. A meta-analysis comparing performance on the IGT by PSZ and controls evidenced decision-making impairments in PSZ characterized by an underweighting of gain frequency and overweighting of net losses and immediate gains [79]. Impairments in cognitive control and representation of goals have been documented in PSZ and associated with abnormal activation in the dlPFC [11, 26, 56, 80]. The lateral and medial OFC are also theorized to underlie value computations involving updating reinforcement contingencies [11, 56]. Though, evidence supporting the

role of the OFC in reward valuation impairment is wanting. In animal research, projections from the ventrolateral OFC to the basolateral amygdala have been found to be critical for encoding positive reward-value, and projections from the medial OFC to the basolateral amygdala have been found to be critical for value retrieval [81]. FMRI research has supported the importance of the cingulo-striatal network in amotivation and has evidenced the focal role of the ACC in translating the value of an action to its initiation [82]. Clinically stable PSZ evidenced reduced activation in the rostro-ventral ACC, in addition to altered striatal activity, during initiation of approach to reward. Reduced sensitivity to value changes in guiding decisions in PSZ has also been associated with greater self-reported aberrant salience traits, suggesting that altered reward processing closely relates to aberrantly salient attributions in SZ [83].

It has been proposed that PSZ experience difficulty in integrating the probability and magnitude of experienced outcomes when assessing reward-value. In an investigation examining mechanisms underlying deficits in probability-magnitude integration in PSZ, a learning paradigm was utilized wherein variations in reward-magnitude and probability established the expected value (EV) of each stimulus [84]. PSZ with motivational deficits did not accurately represent reward-magnitude, relying heavily on stimulus-response associations that were void of value rather than utilizing EV-based learning.

Effort-Allocation and Goal-directed Decision-making

PSZ evidence difficulty with integrating causal knowledge regarding changes in outcome values to guide decisions [85]. Additionally, an expanding body of research shows that PSZ exhibit less willingness to expend effort to receive rewards compared with controls, and much work exhibits associations among impaired effort-allocation, greater negative symptoms, and poorer functioning [86, 87]. Many investigations focused on effort-allocation in reward processing have utilized paradigms requiring physical effort expenditure (e.g., grip strength) [21, 22]. PSZ exhibit reduced allocation of physical effort relative to increases in reward-value or probability [11, 21, 22, 88]. Preliminary research on allocation of cognitive effort suggests that cognitive effort may be reduced in PSZ; though, potential confounding effects of physical effort need to be better accounted for [89]. Deficits in motivation for physical effort expenditure have recently been evidenced to significantly correlate with reduced global cognition, as well as reduced attention and processing speed [19•].

A recent examination of self-efficacy relative to effort-cost computation revealed associations among greater low-effort choices, greater negative symptoms, greater working memory deficits, poor self-efficacy, and reduced anticipatory pleasure [90]. Deficits in effort allocation were associated with greater impairment in completing skills of daily functioning. Evidenced trouble with effective effort allocation experienced by PSZ exhibiting greater negative symptoms could relate to the formation of negative beliefs about competency, further contributing to amotivation.

Implications for Behavioral Rehabilitation Treatment Strategies

It is important to utilize what is known regarding the mechanisms underlying impaired motivation for rewards to establish effective forms of behavioral rehabilitation that target these deficits. Addressing reward processing impairment in PSZ holds promise for improved symptoms, social engagement, and overall functioning. Table 2 depicts behavioral rehabilitation recommendations. As impaired cognitive control has been implicated in explicit learning deficits in PSZ, cognitive remediation interventions have been examined and have evidenced improvement in reward processing and negative symptoms in SZ [91, 92]. Cognitive remediation has also been associated with improvements in social functioning and negative symptoms in first-episode SZ [93]. A program designed to focus on improvement of cognitive skills and emotion regulation [Positive Emotions Programme for Schizophrenia (PEPS)] evidenced greater improvement in apathy and anhedonia scores for PSZ at post-treatment and follow-up compared with treatment as usual [94]. A real-world feasibility assessment of PEPS evidenced significant improvement of negative symptoms and social functioning post-treatment [95]. Cognitive training that integrates exercises aimed at improving social cognition holds promise for improving reward processing and daily functioning in PSZ [96•]. The development of real-time interventions (e.g., mobile applications) may also be of benefit, particularly for at-risk and first-episode populations [97]. Psychopharmacological intervention, particularly with atypical antipsychotics, has been associated with improvement in reward system abnormalities and can augment positive results of cognitive training via the maintenance of clinical stability [33•]. Notably, examination of first-generation antipsychotic treatment has not evidenced these same improvements [41]. Additionally, anticholinergic treatment has been associated with memory impairments in PSZ and can constrain the potential benefits of cognitive training and psychosocial interventions [98].

Evidence supports the utility of incorporating extrinsic rewards in promoting volitional extrinsic motivation in PSZ [57, 99]. Patients with low intrinsic motivation, common in SZ, have been evidenced to benefit more from such interventions compared with patients with greater intrinsic motivation [57]. Low-baseline intrinsic motivation may itself be improved in PSZ; a recent examination of intrinsic motivation in SZ found that during the completion of cognitive games, PSZ who received informationally administered rewards (i.e., positive

Table 2 Recommendations for behavioral rehabilitation in schizophrenia

1. Personalized cognitive remediation training
2. Utilization of social learning theory and token economy
3. Clarity and consistency in reward provision
4. Collaborative-supportive treatment orientation
5. Incorporation of social cognition and emotion regulation training
6. Development of real-time interventions
7. Concurrent continuation of standard treatment (e.g., antipsychotics, CBT)

feedback) reported and evidenced greater intrinsic motivation than patients in a no-feedback condition [100•]. Saperstein and Medalia detail components of self-determination theory (SDT) as determinants of the development of intrinsic motivation to attain rewards [101]. Through the lens of SDT, it is recommended that interventions (e.g., cognitive remediation) are personalized according to individuals' recovery goals, incorporate opportunities for patients to exercise choice (fostering autonomy and competency), and provide for contextualization of learning to practical, everyday circumstances.

The Second Chance program at the Westchester Division of New York Presbyterian Hospital is an inpatient rehabilitation unit which serves as an example of a program utilizing a social learning framework which incorporates the provision of extrinsic rewards in effort to improve intrinsic motivation and functional impairment in treatment-resistant PSZ [102, 103]. Second Chance utilizes a token economy aimed at the acquisition and improvement of skills of independent living within a collaborative-supportive framework wherein patients' perceived competency is fostered by the treatment team. The attainment of immediate rewards (e.g., milieu-based positive feedback, tickets that can be traded in for prizes) serves as reminders for the ability to meet overarching, distal goals (e.g., time spent off the unit). This is of benefit as PSZ evidence deficits in orienting toward long-term rewards [104].

Integrating what is known regarding impairments in motivation and reward processing in PSZ with preliminary evidence regarding behavioral rehabilitation, recommendations for treatment strategies are as follows:

1. Disseminate cognitive remediation targeting working memory impairment as this has been associated with improvements in negative symptoms and social functioning, and personalize training to individual patients' needs. Additionally, provide cognitive supports and training for the use of compensatory cognitive strategies. Gains in ability to maintain reward value representations may underlie the connection between cognitive training and reward processing improvement.
2. Establish a token economy wherein extrinsic rewards are provided for adaptive behaviors.
3. Provide rewards in real-time with consistency and clarity.
4. Utilize a collaborative-supportive orientation, reinforcing the development of competency and intrinsic motivation.
5. Incorporate social cognition and emotion regulation training in group and individual psychotherapy for PSZ.
6. Foster the development and examination of real-time intervention strategies (e.g., mobile applications) aimed at improving cognition and motivation in at-risk and early-illness populations.
7. Implement behavioral rehabilitation for the improvement of motivation in tandem with pharmacological treatment as usual.

Conclusions

Reward processing deficits are common in SZ and are associated with greater negative symptoms, particularly avolition and anhedonia. Dysfunctional dopaminergic signaling in the striatum, underlying aberrant reward prediction errors, is associated with amotivation. Additionally, the frontal-striatal network and impaired cognitive control have been associated with altered reward prediction (anticipatory pleasure), reward receipt, explicit reward learning, and reward valuation as PSZ evidence difficulty with activating and maintaining mental representations of rewards. As apathy and aberrant reward processing are associated with greater functional impairment and poorer quality of life for PSZ, incorporating behavioral rehabilitation strategies tailored to the improvement of these deficits stands to greatly improve illness course and daily living. Cognitive remediation, social cognition, and emotion regulation training should be incorporated in treatment protocols for PSZ with low intrinsic motivation and greater avolition and anhedonia. Additionally, utilizing a social learning framework wherein consistent extrinsic rewards are provided for adaptive behaviors within a supportive and collaborative environment holds promise for the development of motivational and reward processing gains in SZ.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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