

Paradigms for Assessing Hedonic Processing and Motivation in Humans: Relevance to Understanding Negative Symptoms in Psychopathology

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Clinicians and researchers have long known that one of the debilitating aspects of psychotic disorders is the presence of “negative symptoms,” which involve impairments in hedonic and motivational function, and/or alterations in expressive affect. We have a number of excellent clinical tools available for assessing the presence and severity of negative symptoms. However, to better understand the mechanisms that may give rise to negative symptoms, we need tools and methods that can help distinguish among different potential contributing causes, as a means to develop more targeted intervention pathways. Using such paradigms is particularly important if we wish to understand whether the causes are the same or different across disorders that may share surface features of negative symptoms. This approach is in line with the goals of the Research Diagnostic Criteria Initiative, which advocates understanding the nature of core dimensions of brain-behavior relationships transdiagnostically. Here we highlight some of the emerging measures and paradigms that may help us to parse the nature and causes of negative symptoms, illustrating both the research approaches from which they emerge and the types of constructs that they can help elucidate.

Key words: anhedonia/amotivation/assessment/negative symptoms/psychopathology/schizophrenia

Introduction

Psychotic disorders are associated with hallucinations, delusions, and disorganized speech and behavior. These are referred to as “positive symptoms,” as these are florid signs that are apparent to clinicians and family members. However, individuals with psychosis can also experience “negative symptoms,” which involve impairments in

hedonic and motivational function, and/or alterations in expressive affect. Negative symptoms can cause as much if not more disability and functional impairment than positive symptoms. In this issue, Strauss and Cohen¹ provide a cogent review of the phenomenology of negative symptoms transdiagnostically, illustrating the ways in which individuals across the psychotic disorder spectrum and those with other disorders (ie, bipolar disorder, depression, post-traumatic stress disorder) may experience seemingly similar negative symptoms. Clinically, there are several measures available for assessing the presence and severity of negative symptoms, including older measures such as the Schedule for the Assessment of Negative Symptoms, and newer measures such as the Brief Negative Symptom Scale²⁻⁴ and the Clinical Assessment Interview for Negative Symptoms.⁵⁻⁷

Critically, to understand the mechanisms that may give rise to negative symptoms, it is important to employ assessment approaches that distinguish contributing causes and potential intervention pathways. Using such paradigms is particularly important if we wish to understand whether the causes are the same or different across disorders that may share surface features of negative symptoms. This approach is in line with the goals of the Research Diagnostic Criteria (RDoC) Initiative, which advocates understanding the nature of brain-behavior relationships transdiagnostically. The goal of this review is to highlight some of the emerging measures and paradigms that may help us to parse the nature and causes of negative symptoms. We selectively review available tasks and paradigms for examining different components of negative symptoms. We do not review findings with these paradigms in regards to specific domains of negative symptoms transdiagnostically, as such a review would be

beyond the scope of this commentary, but rather highlight their potential utility for guiding further work on the mechanisms of negative symptoms.

There are several approaches for studying the mechanisms of negative symptoms. The first approach emerges from methods long-utilized in behavioral neuroscience work with animals, including a wide variety of reinforcement learning paradigms see ref.⁸ for an example. A second and related approach comes from computational modeling of reinforcement learning and decision-making.⁹ A third approach comes from the growing area of technology-enhanced methods, including computer-based analysis (eg, computerized speech analyses)¹⁰ and mobile technology.^{11,12} In discussing these approaches, it is useful to draw upon the empirically supported dimensions of negative symptoms. Specifically, as discussed by Strauss and Cohen (in press), several studies support the distinction between motivational/volitional and expressive dimensions of negative symptoms. The motivational/volitional dimension encompasses anhedonia (a putative reduction in the ability to experience pleasure), avolition (reduced initiation and persistence in goal-directed activities) and asociality (a reduction in the desire for and frequency of social interactions). The expressive dimension includes blunted affect (reduced expressiveness in facial, vocal or body gestures) and alogia (reductions in the amount of speech).

Motivational Domain

Many processes relevant to assessing motivational deficits are those captured in the Positive Valence System (PVS) RDoC domain, which was recently modified.¹³ This RDoC domain includes constructs such as reward responsiveness, reward learning, and reward valuation. Critically, it may be important to understand how these constructs interact with constructs in the Cognitive Systems domain of RDoC, including measures related to goal-directed action.

Anhedonia

The paradigms most relevant to assessing anhedonia are those that examine responses to positive stimuli, including primary (eg, food) and secondary (eg, money) rewards. These approaches are captured in the Initial Response to Reward construct in the RDoC matrix. Several paradigms examine reported experience to such stimuli, using well-validated self-report measures or visual analog scales in the lab (see Kring & Elis¹⁴ for a review) and ecological momentary assessment responses in everyday life.^{11,12} Although such paradigms are useful and indeed are the best way to capture an individual's self-perceived experience, self-reports can be subject to biases, including experimental demand.¹⁵ Thus, many researchers also examine other indicators of responses to reward, including neural responses (eg, responses to

reward and loss outcomes on monetary incentive delay [MID] type tasks),¹⁶ evoked response potentials such as the RewP (eg, in response to outcomes on the “doors” task),^{17,18} or other physiological indicators such as skin conductance or heart rate.¹⁹ The ideal approach to assessing anhedonia is one that assesses multiple indicators (ie, self-report, behavior, brain activation). Importantly, these methods can help us understand whether clinical assessments of “anhedonia” actually reflect a diminished “in the moment” responsiveness to positive stimuli, or some other deficit that might yield misleading clinical ratings of anhedonia, such as diminished recall of the extent of pleasure that was actually experienced in the past²⁰ or difficulties imagining future pleasure.²¹

Avolition

Avolition involves reduced initiation of goal-directed activities and is assessed using paradigms that examine anticipation of future positive or negative outcomes, or how people change subsequent behavior based on the experience of outcomes. These approaches are incorporated in several constructs of the RDoC PVS matrix, including Reward Anticipation, Habit, Probabilistic and Reinforcement Learning, Reward Prediction Error, and Reward Valuation. Other than the Temporal Experience of Pleasure Scale,²² there are few self-report paradigms for Reward Anticipation. Of course, items querying about anticipation to cues in paradigms such as the MID task or other cued gambling tasks could provide useful data. Variations on the MID task have been used with fMRI to examine neural responses to cues that predict the likelihood of rewarding outcomes.^{19,23,24} Another method to assess reward anticipation is by assessing Reward Prediction Error. This is a physiological response to the occurrence of unexpected positive outcomes or the absence of expected positive outcomes. It is typically measured with fMRI or other neuroimaging methods in paradigms such as the MID task or other reward learning paradigms.^{19,23,24} Reward prediction errors are associated with activation in striatal regions of the brain.¹⁶

Habit Learning tasks assess the degree to which behavior is influenced by positive outcomes using behavioral or neuroimaging methods. Habit tasks have not been used frequently in negative symptom research, but could be an interesting approach to understanding more implicit or “automatic” components of reward learning that may be relevant to negative symptoms. In contrast, Probabilistic and Deterministic Reinforcement Learning paradigms have been used more frequently. These include those tasks that assess implicit reward learning,⁸ those which dissociate learning from rewards vs learning from punishments,²⁵ those which dissociate learning from rewards vs learning to avoid losses,⁹ and those which distinguish between model-based vs model-free reinforcement learning.²⁶ Many of these tasks arise from a computational modeling tradition that allows them to be analyzed in

terms of derived parameters such as learning rate or exploration, and most can be used with neuroimaging methods to assess neural correlates.

In addition to reinforcement learning paradigms, paradigms that examine how cognitive functions (eg, attention, working memory) can be modified by incentives can also be useful. Many of these paradigms involve cognitive control or working memory and provide both behavioral and neuroimaging metrics of these processes.^{27–30} Such tasks can tap how incentive information may drive the development and maintenance of goal-directed action plans, which may contribute to avolition.^{31–36}

Another area of recent research relevant to understanding negative symptoms is the construct of Effort valuation/Willingness to work in the RDoC PVS. Effort valuation refers to the computations that individuals perform to estimate the amount of “work” required to obtain a reward. Several paradigms assess effort valuation, whether in physical effort,^{37–39} cognitive effort,⁴⁰ or perceptual effort.⁴¹ These paradigms have been used most frequently to assess behavioral indicators of effort valuation, but can also be used with neuroimaging methods to assess neural responses.⁴²

Asociality

This symptom refers to a reduction in the desire for and frequency of social interactions. Newer mobile technology approaches have been developed to measure the self-reported frequency of social interactions in daily life using ecological momentary assessments,⁴³ assessments of texts and phone calls,^{44,45} and information about geolocation that may reflect social mobility.^{44,45} There are also self-report measures to assess enjoyment of social interactions, either in the lab⁴⁶ or in everyday life.⁴³ Although there are fewer experimental paradigms to assess the reward value of social interactions, there are recently developed reinforcement learning paradigms that use social instead of monetary incentives.^{47–51} Such paradigms may help distinguish between diminished social pleasure, difficulties in anticipating social interactions, and using such information to guide social-goal directed behavior.

Expressive Deficits

Some of the processes and paradigms that may be relevant for understanding expressive deficits are captured in the Social Processes domain of RDoC. For example, blunted affect may tap into the constructs of facial and non-facial communication. Alogia has a less clear mapping to the RDoC, in part because added work is needed on incorporating language related processes into the RDoC.

Blunted Affect

Many clinical scales for assessing negative symptoms rely on clinician or experimenter ratings of facial, vocal or

gestural expression. Although these can be useful, they are sometimes collected in the absence of emotionally evocative situations, which make them difficult to interpret. Thus, some researchers utilize detailed facial coding systems to assess facial expressions in response to evocative stimuli or situations,⁵² such as the Facial Expression Coding System.⁵³ Another approach is to use electromyography to measure the movement of facial muscles that might correspond to emotional expressions.^{54,55} More recent computerized facial analysis software to measure facial expression is also being used.⁵⁶ Detailed coding systems for assessing vocal expression have also been developed.⁵⁷ Another promising approach to assessing expressive deficits involves computerized acoustic analyses of speech to capture elements of prosody and intonation that may be indicative of emotional expressiveness in speech.^{10,57–59}

Gesture expression has received the least focus to date, but novel recent work using 3D motion capture to measure expressive gestures during social interactions shows that these measures are correlated with clinical assessments of negative symptoms.⁶⁰ Together, these rating and computerized analysis approaches are useful methods to measure the nature and severity of expressive deficits, and could be useful for characterizing the similarities and differences in the type of expressive deficits transdiagnostically. Further, they have also long been used to identify dissociations between experience and expression of emotion, and more recently between channels of expression.

Alogia

This symptom refers to reductions in the amount and rate of speech. Clinical rating scales that rely on clinician or experimenter judgment of speech reduction are typically used. Other methods include more time intensive interview based approaches. Here, interviewers use standard prompts, recording and transcribing speech, and then computing, eg, numbers of words and pauses in speech.⁶¹ More recently, computerized approaches have been developed to assess amount of speech, pace of speech, and pauses.^{10,62} Paradigms have also been developed for testing hypotheses about the mechanisms of alogia. For example, dual-task paradigms, where individuals speak while doing another task, can assess whether reduced cognitive resources or working memory may contribute to alogia.^{61,63,64} Such paradigms have been shown to reduce speech amount and rate, and to increase frequency and duration of pausing,^{61,63,64} suggesting that cognitive mechanisms (eg, working memory) may be contributing to alogia.

Summary

This brief selective review provides an overview of the types of paradigms and measures available for assessing psychological and neural mechanisms that might be associated with negative symptoms. Indeed, there are a

several available options for assessing motivational and expressive impairments, including paradigms derived from animal and computational work, as well as an increasing emphasis on mobile and computerized technologies. Importantly, these paradigms can be used transdiagnostically. Although these options are promising, there is still work to be done in terms of assessing the psychometrics of these paradigm, including being able to use them to establish differential deficits in specific processes. Importantly, as reviewed in Young et al, in this issue, the paradigms in humans inspired by animal work help to promote the use of homologous paradigm across species, which can further illuminate mechanisms and pathways to novel interventions.

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