**Schizophrenia Bulletin** vol. 43 no. 4 pp. 701–705, 2017
doi:10.1093/schbul/sbx063
Advance Access publication May 17, 2017

COMMENTARY

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

**Deanna M. Barch*[,1], James M. Gold[2], and Ann M. Kring[3]**

[1]Departments of Psychological & Brain Science and Psychiatry, Washington University, St. Louis, MO; [2]Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD; [3]Department of Psychology, University of California at Berkeley, Berkeley, CA

*To whom correspondence should be addressed; Departments of Psychological & Brain Science and Psychiatry, Washington University, Box 1125, One Brookings Drive, St. Louis, MO 63130, US; tel: 314-935-8729, fax: 314-935-8790, e-mail: dbarch@wustl.edu

**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 Cohen[1] 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 Scale[2–4] 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 transdiagnositically, 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 & Elis[14] 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 past[20] 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 lab[46] 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.

**Acknowledgment**

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

**References**

1. Strauss GP, Cohen AS. A transdiagnostic review of negative symptom phenomenology and etiology. *Schizophr Bull*. In press.
2. Kirkpatrick B, Strauss GP, Nguyen L, et al. The Brief Negative Symptom Scale: psychometric properties. *Schizophr Bull*. 2011;37:300–305.
3. Strauss GP, Hong LE, Gold JM, et al. Factor structure of the Brief Negative Symptom Scale. *Schizophr Res*. 2012;142:96–98.
4. Strauss GP, Keller WR, Buchanan RW, et al. Next-generation negative symptom assessment for clinical trials: validation of the Brief Negative Symptom Scale. *Schizophr Res*. 2012;142:88–92.
5. Blanchard JJ, Kring AM, Horan WP, Gur R. Toward the next generation of negative symptom assessments: the collaboration to advance negative symptom assessment in schizophrenia. *Schizophr Bull*. 2011;37:291–299.
6. Horan WP, Kring AM, Gur RE, Reise SP, Blanchard JJ. Development and psychometric validation of the Clinical Assessment Interview for Negative Symptoms (CAINS). *Schizophr Res* 2011.
7. Kring AM, Gur RE, Blanchard JJ, Horan WP, Reise SP. The Clinical Assessment Interview for Negative Symptoms (CAINS): final development and validation. *Am J Psychiatry*. 2013;170:165–172.
8. Pizzagalli DA, Jahn AL, O'Shea JP. Toward an objective characterization of an anhedonic phenotype: a signal-detection approach. *Biol Psychiatry*. 2005;57:319–327.
9. Gold JM, Waltz JA, Matveeva TM, et al. Negative symptoms and the failure to represent the expected reward value of actions: behavioral and computational modeling evidence. *Arch Gen Psychiatry*. 2012;69:129–138.
10. Cohen AS, Alpert M, Nienow TM, Dinzeo TJ, Docherty NM. Computerized measurement of negative symptoms in schizophrenia. *J Psychiatr Res*. 2008;42:827–836.
11. Gard DE, Kring AM, Gard MG, Horan WP, Green MF. Anhedonia in schizophrenia: distinctions between anticipatory and consummatory pleasure. *Schizophr Res*. 2007;93:253–260.
12. Moran EK, Culbreth AJ, Barch DM. Ecological momentary assessment of negative symptoms in schizophrenia: relationships to effort based decision making and reinforcement learning. *J Abnorm Psychol*. In press.
13. Barch DM, Oquendo MA, Pacheco J, Morris S. *Behavioral Assessment Methods for RDoC Constructs: A Report by the National Advisory Mental Health Council Workgroup on Tasks and Measures for RDoC*. Washington, DC: National Insitutes of Mental Health; 2016.
14. Kring AM, Elis O. Emotion deficits in people with schizophrenia. *Annu Rev Clin Psychol*. 2013;9:409–433.
15. Cohen AS, Minor KS. Emotional experience in patients with schizophrenia revisited: meta-analysis of laboratory studies. *Schizophr Bull*. 2010;36:143–150.
16. Radua J, Schmidt A, Borgwardt S, et al. Ventral striatal activation during reward processing in psychosis: a neurofunctional meta-analysis. *JAMA Psychiatry*. 2015;72:1243–1251.
17. Belden AC, Irvin K, Hajcak G, et al. Neural correlates of reward processing in depressed and healthy preschool-age children. *J Am Acad Child Adolesc Psychiatry*. 2016;55:1081–1089.
18. Levinson AR, Speed BC, Infantolino ZP, Hajcak G. Reliability of the electrocortical response to gains and losses in the doors task. *Psychophysiology*. 2017;54:601–607.
19. Brinkmann K, Schüpbach L, Joye IA, Gendolla GH. Anhedonia and effort mobilization in dysphoria: reduced cardiovascular response to reward and punishment. *Int J Psychophysiol*. 2009;74:250–258.
20. Strauss GP, Gold JM. A new perspective on anhedonia in schizophrenia. *Am J Psychiatry*. 2012;169:364–373.
21. Barch DM, Dowd EC. Goal representations and motivational drive in schizophrenia: the role of prefrontal-striatal interactions. *Schizophr Bull*. 2010;36:919–934.
22. Gard DE, Germans M, Kring AM, John OP. Anticipatory and consummatory components of the experience of pleasure: a scale development study. *J Pers Res*. 2006;40:1086–1102.
23. Zhang B, Lin P, Shi H, et al. Mapping anhedonia-specific dysfunction in a transdiagnostic approach: an ALE meta-analysis. *Brain Imaging Behav*. 2016;10:920–939.
24. Zhang WN, Chang SH, Guo LY, Zhang KL, Wang J. The neural correlates of reward-related processing in major depressive disorder: a meta-analysis of functional magnetic resonance imaging studies. *J Affect Disord*. 2013;151:531–539.
25. Waltz JA, Frank MJ, Wiecki TV, Gold JM. Altered probabilistic learning and response biases in schizophrenia: behavioral evidence and neurocomputational modeling. *Neuropsychology*. 2011;25:86–97.
26. Culbreth AJ, Westbrook A, Daw ND, Botvinick M, Barch DM. Reduced model-based decision-making in schizophrenia. *J Abnorm Psychol*. 2016;125:777–787.
27. Chiew KS, Braver TS. Dissociable influences of reward motivation and positive emotion on cognitive control. *Cogn Affect Behav Neurosci*. 2014;14:509–529.
28. Chiew KS, Braver TS. Reward favors the prepared: incentive and task-informative cues interact to enhance attentional control. *J Exp Psychol Hum Percept Perform*. 2016;42:52–66.
29. Jimura K, Locke HS, Braver TS. Prefrontal cortex mediation of cognitive enhancement in rewarding motivational contexts. *Proc Natl Acad Sci USA*. 2010;107:8871–8876.
30. Padmala S, Pessoa L. Reward reduces conflict by enhancing attentional control and biasing visual cortical processing. *J Cogn Neurosci*. 2011;23:3419–3432.

31. Green MF, Satz P, Ganzell S, Vaclav JF. Wisconsin Card Sorting Test performance in schizophrenia: remediation of a stubborn deficit. *Am J Psychiatry*. 1992;149:62–67.
32. Hellman SG, Kern RS, Neilson LM, Green MF. Monetary reinforcement and Wisconsin Card Sorting performance in schizophrenia: why show me the money? *Schizophr Res*. 1998;34:67–75.
33. Vollema MG, Geurtsen GJ, van Voorst AJ. Durable improvements in Wisconsin Card Sorting Test performance in schizophrenic patients. *Schizophr Res*. 1995;16:209–215.
34. Roiser JP, Stephan KE, den Ouden HE, Barnes TR, Friston KJ, Joyce EM. Do patients with schizophrenia exhibit aberrant salience? *Psychol Med*. 2009;39:199–209.
35. Mann CL, Footer O, Chung YS, Driscoll LL, Barch DM. Spared and impaired aspects of motivated cognitive control in schizophrenia. *J Abnorm Psychol*. 2013;122:745–755.
36. Chung YS, Barch DM. Frontal-striatum dysfunction during reward processing: relationships to amotivation in schizophrenia. *J Abnorm Psychol*. 2016;125:453–469.
37. Treadway MT, Bossaller NA, Shelton RC, Zald DH. Effort-based decision-making in major depressive disorder: a translational model of motivational anhedonia. *J Abnorm Psychol*. 2012;121:553–558.
38. Gold JM, Strauss GP, Waltz JA, Robinson BM, Brown JK, Frank MJ. Negative symptoms of schizophrenia are associated with abnormal effort-cost computations. *Biol Psychiatry*. 2013;74:130–136.
39. Wolf DH, Satterthwaite TD, Kantrowitz JJ, et al. Amotivation in schizophrenia: integrated assessment with behavioral, clinical, and imaging measures. *Schizophr Bull*. 2014;40:1328–1337.
40. Gold JM, Kool W, Botvinick MM, Hubzin L, August S, Waltz JA. Cognitive effort avoidance and detection in people with schizophrenia. *Cogn Affect Behav Neurosci*. 2015;15:145–154.
41. Reddy LF, Horan WP, Barch DM, et al. Effort-based decision-making paradigms for clinical trials in schizophrenia: part 1—psychometric characteristics of 5 paradigms. *Schizophr Bull*. 2015;41:1045–1054.
42. Croxson PL, Walton ME, O'Reilly JX, Behrens TE, Rushworth MF. Effort-based cost-benefit valuation and the human brain. *J Neurosci*. 2009;29:4531–4541.
43. Granholm E, Ben-Zeev D, Fulford D, Swendsen J. Ecological Momentary Assessment of social functioning in schizophrenia: impact of performance appraisals and affect on social interactions. *Schizophr Res*. 2013;145:120–124.
44. Ben-Zeev D, Scherer EA, Wang R, Xie H, Campbell AT. Next-generation psychiatric assessment: using smartphone sensors to monitor behavior and mental health. *Psychiatr Rehabil J*. 2015;38:218–226.
45. Ben-Zeev D, Wang R, Abdullah S, et al. Mobile behavioral sensing for outpatients and inpatients with schizophrenia. *Psychiatr Serv*. 2016;67:558–561.
46. Aghevli MA, Blanchard JJ, Horan WP. The expression and experience of emotion in schizophrenia: a study of social interactions. *Psychiatry Res*. 2003;119:261–270.
47. van den Bos W, Talwar A, McClure SM. Neural correlates of reinforcement learning and social preferences in competitive bidding. *J Neurosci*. 2013;33:2137–2146.
48. Vernetti A, Smith TJ, Senju A. Gaze-contingent reinforcement learning reveals incentive value of social signals in young children and adults. *Proc Biol Sci Royal Soc*. 2017;284.
49. Lin A, Adolphs R, Rangel A. Social and monetary reward learning engage overlapping neural substrates. *Soc Cogn Affect Neurosci*. 2012;7:274–281.
50. Heerey EA. Learning from social rewards predicts individual differences in self-reported social ability. *J Exp Psychol Gen*. 2014;143:332–339.
51. Shore DM, Heerey EA. The value of genuine and polite smiles. *Emotion*. 2011;11:169–174.
52. Mote J, Stuart BK, Kring AM. Diminished emotion expressivity but not experience in men and women with schizophrenia. *J Abnorm Psychol*. 2014;123:796–801.
53. Kring AM, Sloan DM. The Facial Expression Coding System (FACES): development, validation, and utility. *Psychol Assess*. 2007;19:210–224.
54. Park S, Kim K. Physiological reactivity and facial expression to emotion-inducing films in patients with schizophrenia. *Arch Psychiatr Nurs*. 2011;25:e37–e47.
55. Kring AM, Kerr SL, Earnst KS. Schizophrenic patients show facial reactions to emotional facial expressions. *Psychophysiology*. 1999;36:186–192.
56. Cohen AS, Morrison SC, Callaway DA. Computerized facial analysis for understanding constricted/blunted affect: initial feasibility, reliability, and validity data. *Schizophr Res*. 2013;148:111–116.
57. Hoekert M, Kahn RS, Pijnenborg M, Aleman A. Impaired recognition and expression of emotional prosody in schizophrenia: review and meta-analysis. *Schizophr Res*. 2007;96:135–145.
58. Cohen AS, Mitchell KR, Elvevåg B. What do we really know about blunted vocal affect and alogia? A meta-analysis of objective assessments. *Schizophr Res*. 2014;159:533–538.
59. Martínez-Sánchez F, Muela-Martínez JA, Cortés-Soto P, et al. Can the acoustic analysis of expressive prosody discriminate schizophrenia? *Span J Psychol*. 2015;18:E86.
60. Lavelle M, Healey PG, McCabe R. Is nonverbal communication disrupted in interactions involving patients with schizophrenia? *Schizophr Bull*. 2013;39:1150–1158.
61. Barch DM, Berenbaum H. The effect of language production manipulations on negative thought disorder and discourse coherence disturbances in schizophrenia. *Psychiatry Res*. 1997;71:115–127.
62. Cohen AS, Mitchell KR, Docherty NM, Horan WP. Vocal expression in schizophrenia: less than meets the ear. *J Abnorm Psychol*. 2016;125:299–309.
63. Cohen AS, McGovern JE, Dinzeo TJ, Covington MA. Speech deficits in serious mental illness: a cognitive resource issue? *Schizophr Res*. 2014;160:173–179.
64. Barch D, Berenbaum H. The relationship between information processing and language production. *J Abnorm Psychol*. 1994;103:241–250.