A Transdiagnostic Review of Negative Symptom Phenomenology and Etiology

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In the DSM5, negative symptoms are 1 of the 5 core dimensions of psychopathology evaluated for schizophrenia. However, negative symptoms are not pathognomonic—they are also part of the diagnostic criteria for other schizophrenia-spectrum disorders, disorders that sometimes have comorbid psychosis, diagnoses not in the schizophrenia-spectrum, and the general “nonclinical” population. Although etiological models of negative symptoms have been developed for chronic schizophrenia, there has been little attention given to whether these models have transdiagnostic applicability. In the current review, we examine areas of commonality and divergence in the clinical presentation and etiology of negative symptoms across diagnostic categories. It was concluded that negative symptoms are relatively frequent across diagnostic categories, but individual disorders may differ in whether their negative symptoms are persistent/transient or primary/secondary. Evidence for separate dimensions of volitional and expressive symptoms exists, and there may be multiple mechanistic pathways to the same symptom phenomenon among DSM-5 disorders within and outside the schizophrenia-spectrum (ie, equifinality). Evidence for a novel transdiagnostic etiological model is presented based on the Research Domain Criteria (RDoC) constructs, which proposes the existence of 2 such pathways—a hedonic pathway and a cognitive pathway—that can both lead to expressive or volitional symptoms. To facilitate treatment breakthroughs, future transdiagnostic studies on negative symptoms are warranted that explore mechanisms underlying volitional and expressive pathology.

Key words: anhedonia/avolition/asociality/blunted affect/alogia

Introduction

Negative symptoms (ie, reductions in goal-directed activity, social behavior, pleasure, and the outward expression of emotion or speech) have long been considered a core feature of psychotic disorders.1,2 Modern empirical studies confirm these early clinical observations, indicating that negative symptoms are distinct from other domains of psychopathology (eg, psychosis, disorganization)3 and associated with a range of important clinical outcomes (eg, disease liability, quality of life, subjective well-being, recovery).4,5 Unfortunately, psychosocial and pharmacological interventions have yielded limited effectiveness for improving negative symptoms in schizophrenia.6 At present, treatment development is stunted due in large part to a poor understanding of the factors that cause and maintain negative symptoms.7 Thus, candidate mechanisms are unclear and treatment development remains a high priority.

Although traditionally described only in relation to schizophrenia, it has become clear that negative symptoms also occur in other schizophrenia-spectrum disorders, disorders that sometimes have comorbid psychosis, diagnoses not in the schizophrenia-spectrum, certain neurological disorders, and the general “nonclinical” population.10,11 Little attention has been paid to areas of commonality and divergence in the clinical presentation of negative symptoms across diagnostic boundaries. There has also yet to be systematic comparison of mechanisms underlying negative symptoms across diagnostic categories, despite recent advances in etiological models of negative symptoms that have been proposed for schizophrenia. These issues are of critical importance given the potential for research classification systems, such as the RDoC and HiTop, to answer questions related to equifinality and clarify the biological basis of negative symptoms.12

The current manuscript attempts to address these gaps in the literature by achieving 2 primary aims: (1) reviewing qualitative and quantitative differences in the clinical presentation of negative symptoms across DSM-5 diagnostic categories, as well as between clinical and healthy
Do Negative Symptoms Qualitatively Differ Across Diagnostic Categories?

Negative symptoms have been part of the diagnostic criteria for schizophrenia since the DSM-II. In the DSM-5, negative symptoms are 1 of 5 core dimensions of psychopathology evaluated for schizophrenia. There are 5 commonly agreed upon domains of negative symptoms, which separate into 2 dimensions—volition (anhedonia, avolition, asociality) and expression (blunted affect, alogia)—that are evaluated in the DSM-5 diagnostic criteria for schizophrenia. (see table 1 for definitions).

However, it is clear that negative symptoms are not pathognomonic—they are also part of the diagnostic criteria and associated features for other schizophrenia—spectrum disorders, including schizoaffective disorder, schizoaffective disorder, schizotypal personality disorder, schizoid personality disorder, and paranoid personality disorder. Negative symptoms are not part of the diagnostic criteria for delusional disorder and brief psychotic disorder.

Although not explicitly termed negative symptoms, several psychiatric disorders outside of the schizophrenia-spectrum have negative symptoms within their diagnostic criteria or associated features (eg, bipolar disorder, major depressive disorder, posttraumatic stress disorder), some of which are known to have comorbid psychosis. Other disorders that do not often have comorbid psychosis also include negative symptoms within their diagnostic criteria and associated features (eg, selective mutism, social anxiety disorder, neurocognitive disorders).

Negative symptoms also occur in several DSM-5 neurocognitive disorders, but are commonly referred to as apathy in this context. Neurological disorders where negative symptoms are frequently present include: Alzheimer’s disease, frontotemporal dementia, Huntington’s disease, multiple sclerosis, Parkinson’s disease, progressive supranuclear palsy, and traumatic brain injury. Apathy described in neurological disorders is qualitatively similar to avolition and asociality in schizophrenia. For example, the Apathy Evaluation Scale, a clinical scale commonly used to rate apathy, includes items that focus on the internal experience (eg, interest in activities) and overt behavior (eg, engaging in goal directed behavior) for social and goal-directed activities. Using such clinical rating scales, apathy is relatively frequent among the aforementioned neurological disorders, with prevalence estimates ranging from 32% to 80% depending on the disorder.

Terminology used to describe phenomenon that can be considered negative symptoms is highly variable across diagnostic categories. The terms blunted affect, alogia, anhedonia, avolition, and asociality are rarely used outside the schizophrenia-spectrum, even though symptoms described in other disorders fit definitions for these terms (table 1). Additionally, terminology within the schizophrenia-spectrum is not always consistent. For example, the terms “blunted affect” and “constricted affect” are used to refer to the same phenomenon in schizophrenia and schizotypal personality disorder, respectively. To illustrate these points, we carefully reviewed the “diagnostic criteria” and “associated features supporting diagnosis” sections of disorders included in the 20 DSM-5 sections and recorded terms/phrases that reflected any of the 5 NIMH negative symptom consensus domains. A total of 19 disorders were identified as having blunted affect, alogia, anhedonia, avolition, or asociality within their DSM-5 diagnostic criteria or associated features. Results of this process are presented visually in figure 1. As seen in the figure 1, there is not only a lack of consistency in terminology throughout the DSM-5, but also lack of specificity in descriptions and conflation of symptom domains.

To facilitate qualitative transdiagnostic comparisons, we mapped descriptions used in DSM-5 diagnostic criteria and associated features onto the 5 domains identified in the 2005 NIMH negative symptom consensus conference. Table 2 depicts the presence/absence of these 5 symptoms across diagnoses. As can be seen in the table 2, asociality and avolition are the negative symptoms that occur most frequently within DSM-5 diagnostic criteria and associated features. Blunted affect and anhedonia occur with moderate frequency, and alogia is least frequent.

Table 1. Definitions for the 5 Symptom Domains Identified in the 2005 NIMH Consensus Meeting on Negative Symptoms

<table>
<thead>
<tr>
<th>Definition</th>
<th>Consensus Domain</th>
</tr>
</thead>
<tbody>
<tr>
<td>A decrease in the outward expression of emotion in terms of facial expression, vocal expression, or body gestures</td>
<td>Blunted affect</td>
</tr>
<tr>
<td>A reduction in the quantity of speech and amount of spontaneous elaboration</td>
<td>Alogia</td>
</tr>
<tr>
<td>A reduction in the initiation of and persistence in goal-directed activities and the desire to perform such activities</td>
<td>Avolition</td>
</tr>
<tr>
<td>A reduction in the intensity of positive emotion and/or a reduction in the frequency of engaging in pleasurable activities</td>
<td>Anhedonia</td>
</tr>
<tr>
<td>A reduction in the frequency of social interaction and the desire to form close relationships</td>
<td>Asociality</td>
</tr>
</tbody>
</table>
Although Table 2 makes the 5 domains appear qualitatively similar across diagnostic categories, there may be important phenomenological differences. It is possible for 2 different disorders to include the same negative symptom within their diagnostic criteria, but for these symptoms to be caused by very different processes. For example, asociality can occur in paranoid personality disorder because an individual is suspicious and preoccupied with whether others can be trusted, resulting in active social withdrawal to safeguard against the malicious intentions of others. In contrast, in schizophrenia, asociality can sometimes manifest as an apathetic social withdrawal, where an individual does not engage in social interactions because they have little desire to be around others and do not find relationships important or interesting.

The aforementioned example highlights the importance of making the primary/secondary distinction when performing transdiagnostic comparisons. Negative symptoms are considered primary or idiopathic if they are direct manifestations of the illness that cannot be attributed to secondary factors, such as anxiety, depression, disorganization, hallucinations, delusions, or medication effects. A minority of individuals diagnosed with schizophrenia (~15%–25%) display negative symptoms that are primary and persistent. This presentation, termed the “deficit syndrome,” reflects a separate disease within the broader diagnosis of schizophrenia. Deficit patients have unique signs and symptoms, course of illness, pathophysiology, neurocognition, functional outcome, risk factors, and treatment response compared to “nondeficit” patients. It is unknown whether negative symptoms that occur in disorders other than schizophrenia also display primary negative symptoms, or whether all of these manifestations can be considered secondary. By definition some could be considered secondary when negative symptoms are driven by depression, anxiety, delusions, or hallucinations. Negative symptoms seen in autism spectrum disorders, which have high genetic and diagnostic overlap with schizophrenia, may even be influenced by secondary factors (eg, asociality due to anxiety). In neurological disorders, negative symptoms can reflect secondary factors, as indicated by associations between clinical ratings of apathy and depression in Alzheimer’s disease, Parkinson’s disease, temporal lobe epilepsy, and Huntington’s disease, however, many neurological patients also display negative symptoms when depression is absent, suggesting that primary negative symptoms may exist. Some disorders also display negative symptoms that are more transient and...
Negative Symptoms

tied to acute exacerbations by secondary causes, which resolve when those symptoms are treated. Such instances are distinct from the more enduring, trait-like presentation of primary negative symptom schizophrenia patients who show little fluctuation in severity across the course of illness despite changes in other symptoms or medication regimen.32

The primary/secondary and persistent/transient distinctions should be carefully considered when conducting research on negative symptoms in transdiagnostic samples. The proportion of individuals whose negative symptoms are primary and persistent differs across diagnostic categories, and the success of research endeavors aiming to examine pathophysiological mechanisms of negative symptoms may depend on how homogeneous samples are in relation to these 2 factors. Similar to Clementz et al,33 a promising approach to reducing heterogeneity could be to identify “biotypes” that cut across diagnostic categories using data-driven methods, and then examine whether those biotypes travel with primary/secondary and persistent/transient clinical classifications.

Do Negative Symptoms Quantitatively Differ Across Diagnostic Categories?

Few studies have made direct quantitative comparisons of negative symptom severity across disorders within the schizophrenia-spectrum. This may be due in part to different scales being used to measure negative symptoms across disorders and phases of illness. For example, negative symptoms are typically assessed in schizotypy using trait self-reports,34 whereas studies examining psychotic disorders tend to use clinical interview-based rating scales.15,16,35,36 The few studies making direct quantitative comparisons suggest the presence of a continuum of severity within phases of illness: chronic (ie, multi-episode with extended illness duration) > first episode >clinical high-risk.37,38 There also appears to be a continuum across diagnostic categories within the schizophrenia-spectrum, with schizophrenia more severe than schizoaffective disorder39–42 which is more severe than schizotypy43; however, this may be more true of blunted affect and alogia than anhedonia, avolition, or asociality.44,45

Results of studies comparing negative symptom severity between schizophrenia and disorders outside the schizophrenia-spectrum are mixed, with some studies reporting greater severity in schizophrenia than bipolar disorder, depression, and anxiety disorders/neuroses44–46 and others reporting comparable severity and frequency.47,48 Although severity may not always differentiate schizophrenia from nonschizophrenia-spectrum disorders, there is evidence that negative symptoms may be more stable and trait-like in schizophrenia than other psychiatric disorders which can be more transient.47,48,51–53

Table 2. Qualitative Comparison of 5 Core Negative Symptom Domains Across Diagnostic Categories

<table>
<thead>
<tr>
<th>Negative Symptom Domain</th>
<th>Asociality</th>
<th>Alogia</th>
<th>Anhedonia</th>
<th>Avolition</th>
<th>Blunted Affect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorder</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Schizoaffective disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Schizophreniform disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Schizotypal personality disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Schizoid personality disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Avoidant personality disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Bipolar disorder (I and II)</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Persistent depressive disorder (dysthymia)</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Premenstrual dysphoric disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Selective mutism</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Social anxiety disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Separation anxiety disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Reactive attachment disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Posttraumatic stress disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Depersonalization/derealization disorder</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Autism spectrum disorder</td>
<td>18</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Neurocognitive disorders</td>
<td>19</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

Note: We reviewed the “diagnostic criteria,” “diagnostic features,” and “associated features supporting diagnosis” sections of disorders included in the 20 DSM-5 sections and recorded terms/phrases that reflected any of the 5 NIMH negative symptom consensus domains. A total of 19 disorders were identified as having blunted affect, alogia, anhedonia, avolition, or asociality within their DSM-5 diagnostic criteria or associated features. Not considered are disorders due to general medical condition, “other specified” disorders, unspecified disorders, and substance induced disorders. Symptoms displayed in neurocognitive disorders vary based on the neurological condition in question.
Notably, negative symptoms may not be as transient as one would expect in mood disorders. There is some evidence that depression and bipolar disorder are associated with attenuated negative symptoms that persist between mood episodes.46–48,51,52 When present in neurocognitive disorders, negative symptoms may be relatively severe, even comparable to that observed in chronic schizophrenia.54

Healthy controls with no diagnosable neurological or psychiatric conditions can also display negative symptoms.43,46,55 However, these cases tend to be infrequent and the severity is attenuated. Measures designed for clinical populations may not be ideal for evaluating negative symptoms in healthy populations, as they fail to capture nuances in the lower range of severity.

To directly illustrate quantitative differences in negative symptom severity across diagnostic categories and phases of illness, figure 2 and table 3 present archival data7,46,56–72 on the Scale for the Assessment of Negative Symptoms (SANS36) for the volitional dimension, expression dimension, and individual domain global scores. Groups include: chronic schizophrenia (n = 610), schizoaffective disorder (n = 197), bipolar disorder (n = 113), major depressive disorder (n = 23), ultra high-risk (ie, psychosis prodrome: n = 24), and healthy controls (n = 69). The frequency distributions suggest that negative symptoms were quite common across schizophrenia, clinical high-risk, schizoaffective disorder, major depression, and bipolar disorder groups. Moreover, the differences between the schizophrenia group and the schizoaffective, major depression, and ultra-high risk groups were modest. Compared to healthy controls, the magnitude of effect was largest in schizophrenia, followed by clinical high-risk, schizoaffective disorder, major depression, and bipolar disorder (table 3 and figure 2).

The Structure of Negative Symptoms Across Diagnostic Categories

Although figure 2 appears to provide support for a continuous distribution of negative symptoms across diagnostic categories, the structure of negative symptoms remains an open debate. Historically, there has been case for 2 conceptualizations: continuous (ie, individuals differ in degree or amount) and categorical (ie, individuals differ in status or kind). There is support for both perspectives.

![Figure 2](https://academic.oup.com/schizophreniabulletin/article/43/4/712/3868631)

**Fig. 2.** Transdiagnostic quantitative comparison of the 2 negative symptom dimensions and 5 consensus domains on the SANS. Note: Panels A and B represent frequency distributions of SANS scores on the expressive and volitional dimensions, respectively. Y-axis values reflect cumulative percentages of participants and X-axis scores denote the average SANS dimension score calculated by adding the 2 global items in each factor and dividing by 2. Exp, expression dimension; VOL, volitional dimension; Anhed/Asoc, global anhedonia/asociality item. Panels C and D reflect Z-scores for each group compared to healthy controls and schizophrenia patients, respectively. As can be seen in Panels A and B, there is variability in scores within each group, but both dimensions tend to be positively skewed. Z-scores indicated a continuum of severity scores ranging from: schizophrenia > ultra high-risk > schizoaffective > major depression > bipolar disorder > healthy control.
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Taxometric studies examining the structure of negative symptoms in schizophrenia have provided support for the categorical perspective, indicating the existence of a negative symptom taxon (~28%–36%) that differs from a non-negative symptom taxon on a number of external validators (eg, summer birth, male sex, premorbid adjustment, neurocognition, and social functioning). However, it may also be that a hybrid categorical-continuous structure best fits negative symptom data in schizophrenia. One article supports this conceptualization, indicating that a distinct class of schizophrenia patients exists when negative symptom scores exceed a certain threshold, and that the magnitude of severity beyond this threshold determines the strength of association with outcome variables.

In disorders other than schizophrenia, it is unclear whether negative symptoms are best viewed as categorical or continuous. When positive and negative symptoms are entered into taxometric analyses concurrently, evidence for a continuous structure emerges in paranoid.

Table 3. Quantitative Comparison of SANS Global Score Severity Across Diagnostic Categories

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Cohen's d Compared to Healthy Controls</th>
<th>Cohen's d Compared to Schizophrenia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Schizophrenia (n = 610)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>1.7 (1.3)</td>
<td>0</td>
<td>5</td>
<td>1.21</td>
<td></td>
</tr>
<tr>
<td>Alogia</td>
<td>1.2 (1.2)</td>
<td>0</td>
<td>5</td>
<td>0.97</td>
<td></td>
</tr>
<tr>
<td>Avolition</td>
<td>2.1 (1.4)</td>
<td>0</td>
<td>5</td>
<td>1.34</td>
<td></td>
</tr>
<tr>
<td>Anhedonia/asociality</td>
<td>2.1 (1.4)</td>
<td>0</td>
<td>5</td>
<td>1.34</td>
<td></td>
</tr>
<tr>
<td>EXP dimension</td>
<td>1.5 (1.1)</td>
<td>0</td>
<td>5</td>
<td>1.34</td>
<td></td>
</tr>
<tr>
<td>AVOL dimension</td>
<td>2.3 (1.2)</td>
<td>0</td>
<td>5</td>
<td>1.74</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1.8 (1.0)</td>
<td>0</td>
<td>4.5</td>
<td>1.68</td>
<td></td>
</tr>
<tr>
<td><strong>Schizoaffective disorder (n = 197)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>1.3 (1.3)</td>
<td>0</td>
<td>4</td>
<td>0.96</td>
<td>-0.31</td>
</tr>
<tr>
<td>Alogia</td>
<td>0.9 (1.0)</td>
<td>0</td>
<td>4</td>
<td>0.93</td>
<td>-0.26</td>
</tr>
<tr>
<td>Avolition</td>
<td>1.9 (1.3)</td>
<td>0</td>
<td>5</td>
<td>1.38</td>
<td>-0.15</td>
</tr>
<tr>
<td>Anhedonia/asociality</td>
<td>2.1 (1.4)</td>
<td>0</td>
<td>5</td>
<td>1.45</td>
<td>0.00</td>
</tr>
<tr>
<td>EXP dimension</td>
<td>1.1 (1.0)</td>
<td>0</td>
<td>4</td>
<td>1.15</td>
<td>-0.37</td>
</tr>
<tr>
<td>AVOL dimension</td>
<td>2.2 (1.1)</td>
<td>0</td>
<td>5</td>
<td>1.94</td>
<td>-0.09</td>
</tr>
<tr>
<td>Total</td>
<td>1.4 (0.9)</td>
<td>0</td>
<td>4.3</td>
<td>1.50</td>
<td>-0.41</td>
</tr>
<tr>
<td><strong>Major depressive disorder (n = 23)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>1.1 (1.3)</td>
<td>1</td>
<td>5</td>
<td>1.18</td>
<td>-0.46</td>
</tr>
<tr>
<td>Alogia</td>
<td>0.9 (1.5)</td>
<td>1</td>
<td>5</td>
<td>1.06</td>
<td>-0.25</td>
</tr>
<tr>
<td>Avolition</td>
<td>1.7 (1.4)</td>
<td>1</td>
<td>5</td>
<td>1.6</td>
<td>-0.29</td>
</tr>
<tr>
<td>Anhedonia/asociality</td>
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<td>5</td>
<td>1.4</td>
<td>-0.36</td>
</tr>
<tr>
<td>EXP dimension</td>
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<td>5</td>
<td>1.23</td>
<td>-0.45</td>
</tr>
<tr>
<td>AVOL dimension</td>
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<td>5</td>
<td>1.69</td>
<td>-0.58</td>
</tr>
<tr>
<td>Total</td>
<td>1.3 (1.0)</td>
<td>1</td>
<td>5</td>
<td>1.84</td>
<td>-0.50</td>
</tr>
<tr>
<td><strong>Bipolar disorder (n = 113)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>0.8 (1.1)</td>
<td>0</td>
<td>4</td>
<td>0.66</td>
<td>-0.71</td>
</tr>
<tr>
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<td>4</td>
<td>0.18</td>
<td>-0.88</td>
</tr>
<tr>
<td>Avolition</td>
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<td>5</td>
<td>0.59</td>
<td>-0.88</td>
</tr>
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<td>Anhedonia/asociality</td>
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<td>5</td>
<td>0.83</td>
<td>-0.65</td>
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<td>EXP dimension</td>
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<td>0</td>
<td>3.5</td>
<td>0.69</td>
<td>-0.96</td>
</tr>
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<td>AVOL dimension</td>
<td>1.2 (1.2)</td>
<td>0</td>
<td>4.5</td>
<td>0.91</td>
<td>-0.92</td>
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<tr>
<td>Total</td>
<td>1.0 (0.8)</td>
<td>0</td>
<td>3.8</td>
<td>1.19</td>
<td>-0.82</td>
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<tr>
<td><strong>Ultra high-risk (n = 24)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>1.5 (1.8)</td>
<td>0</td>
<td>5</td>
<td>1.31</td>
<td>-0.15</td>
</tr>
<tr>
<td>Alogia</td>
<td>0.8 (1.3)</td>
<td>0</td>
<td>4</td>
<td>1.05</td>
<td>-0.33</td>
</tr>
<tr>
<td>Avolition</td>
<td>1.5 (1.8)</td>
<td>0</td>
<td>5</td>
<td>1.16</td>
<td>-0.42</td>
</tr>
<tr>
<td>Anhedonia/asociality</td>
<td>2.3 (1.8)</td>
<td>0</td>
<td>5</td>
<td>1.93</td>
<td>0.14</td>
</tr>
<tr>
<td>EXP dimension</td>
<td>1.1 (1.4)</td>
<td>0</td>
<td>4.5</td>
<td>1.35</td>
<td>-0.36</td>
</tr>
<tr>
<td>AVOL dimension</td>
<td>1.9 (1.6)</td>
<td>0</td>
<td>5</td>
<td>1.78</td>
<td>-0.33</td>
</tr>
<tr>
<td>Total</td>
<td>1.5 (1.2)</td>
<td>0</td>
<td>4.8</td>
<td>1.89</td>
<td>-0.30</td>
</tr>
<tr>
<td><strong>Healthy control (n = 69)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blunted affect</td>
<td>0.2 (0.5)</td>
<td>0</td>
<td>2</td>
<td>—</td>
<td>-1.21</td>
</tr>
<tr>
<td>Alogia</td>
<td>0.1 (0.2)</td>
<td>0</td>
<td>1</td>
<td>—</td>
<td>-0.97</td>
</tr>
<tr>
<td>Avolition</td>
<td>0.3 (0.6)</td>
<td>0</td>
<td>2</td>
<td>—</td>
<td>-1.34</td>
</tr>
<tr>
<td>Anhedonia/asociality</td>
<td>0.3 (0.6)</td>
<td>0</td>
<td>2</td>
<td>—</td>
<td>-1.34</td>
</tr>
<tr>
<td>EXP dimension</td>
<td>0.1 (0.3)</td>
<td>0</td>
<td>1.5</td>
<td>—</td>
<td>-1.34</td>
</tr>
<tr>
<td>AVOL dimension</td>
<td>0.3 (0.5)</td>
<td>0</td>
<td>2</td>
<td>—</td>
<td>-1.74</td>
</tr>
<tr>
<td>Total</td>
<td>0.2 (0.4)</td>
<td>0</td>
<td>1.8</td>
<td>—</td>
<td>-1.68</td>
</tr>
</tbody>
</table>
When individual negative symptoms (e.g., anhedonia and asociality) are examined in isolation in schizotypy, there is some support for both categorical and continuous structures. In depression, anhedonia has been shown to have a continuous structure in adults.

It is also important to consider whether the internal structure of negative symptom scales is similar in schizophrenia and other disorders. As previously mentioned, when negative symptom scales are factor analyzed in samples of schizophrenia patients, there is consistent evidence for 2 independent factors reflecting volitional pathology (avolition, asociality, anhedonia) and expressive pathology (blunted affect, alogia). Much like schizophrenia, there is evidence that negative symptoms load on a factor that is separate from positive symptoms in schizotypy, and negative symptoms divide into 2 factors akin to expression (constricted affect) and volition (no close friends) on self-report questionnaires. In depression, anhedonia loads onto a factor that is distinct from psychomotor symptoms and negative affect, but there is no evidence for separate expression and volition dimensions. Factor analysis of apathy scales in neurological disorders most commonly produces one large factor; however, there is some evidence for minor factors representing interest, insight, and social function. In autism, blunted affect, alogia, and asociality related symptoms load onto a factor separate from motor, sensory, and verbal communication symptoms.

Future studies should examine the internal structure of negative symptom scales and the continuous vs categorical issue in transdiagnostic samples. It will be important to determine how primary/secondary causes influence the internal structure of negative symptom scales, as well as whether categorical, continuous, or hybrid conceptualizations best fit transdiagnostic samples.

The NIMH RDoC “positive valence system” offers a useful conceptual framework for addressing this question. This framework proposes that there are 5 interrelated constructs associated with the initiation of and persistence in goal-directed or reward-seeking activities: initial responsiveness to reward attainment, sustained responsiveness to reward attainment, reward learning, habit, and approach motivation. The approach motivation construct is further divided into 4 subconstructs: reward valuation, effort valuation, expectancy, and action selection. Figure 3 presents a schematic overview of the RDoC positive valence system constructs and how those constructs may interact. Table 4 provides definitions of each construct and their associated biological mechanisms as currently described on the NIMH RDoC website.

**Initial Responsiveness to Reward Attainment.** In chronic schizophrenia, there is consistent evidence for intact initial reward responsiveness at the group level. This evidence comes from laboratory-based studies examining self-reported valence and arousal to pleasant stimuli, as well as electrophysiological studies and neuroimaging studies examining striatal response to monetary reward outcomes; however, lower initial reward responsiveness has been associated with greater severity of volitional symptoms in some studies, indicating important individual differences and heterogeneity within the broader schizophrenia diagnosis.

At the group level, initial reward responsiveness may be diminished for other schizophrenia-spectrum disorders and earlier phases of illness. For example, high-risk, first episode, and schizotypy samples all show diminished self-reported response to positive stimuli, potentially driven by high rates of comorbid depression and anxiety. Neural response to reward outcomes may be intact in high-risk, first-episode, and schizotypal samples at
Table 4. RDoC Positive Valence System Constructs and Associated Biological Correlates

<table>
<thead>
<tr>
<th>Molecules</th>
<th>Circuits</th>
<th>NIMH Definition</th>
<th>RDoC Construct</th>
</tr>
</thead>
<tbody>
<tr>
<td>CREB, endocannabinoids, FosB, glutamate, Mu and delta opioid, orexin</td>
<td>Anterior insula, dorsal ACC, lateral hypothalamus, medial OFC, nucleus accumbens, ventral pallidum, ventromedial PFC, VTA</td>
<td>Mechanisms/processes associated with hedonic responses—as reflected in subjective experiences, behavioral responses, and/or engagement of the neural systems to a positive reinforcer—and culminination of reward seeking</td>
<td>Initial responsiveness to reward attainment</td>
</tr>
<tr>
<td>Dopamine, endocannabinoids, opioid, orexin, serotonin</td>
<td>Arcuate nucleus BA9/medial, medial preoptic area, OFC, paraventricular hypothalamus, ventromedial hypothalamus</td>
<td>Mechanisms/processes associated with the termination of reward seeking, eg, satisfaction, satiation, regulation of consummatory behavior</td>
<td>Sustained/longer-term responsiveness to reward attainment</td>
</tr>
<tr>
<td>Acetylcholine Co-released neuromodular glutamate, CREB, dopamine and dopamine-related molecules, FosB</td>
<td>Amygdala, dorsal striatum, medial prefrontal OFC, ventral striatum, VTA/SN</td>
<td>A process by which organisms acquire information about stimuli, actions, and contexts that predict positive outcomes, and by which behavior is modified when a novel reward occurs or outcomes are better than expected. Reward learning is a type of reinforcement learning, and similar processes may be involved in learning related to negative reinforcement</td>
<td>Reward learning</td>
</tr>
<tr>
<td>Acetylcholine Co-released neuromodular glutamate, CREB, dopamine and dopamine-related molecules, FosB</td>
<td>Dorsal striatum, medial prefrontal SN/VTA, ventral striatum</td>
<td>Sequential, repetitive, motor, or cognitive behaviors elicited by external or internal triggers that, once initiated, can go to completion without constant conscious oversight. Habits can be adaptive by virtue of freeing up cognitive resources. Habit formation is a frequent consequence of reward learning, but its expression can become resistant to changes in outcome value. Related behaviors could be pathological expression of a process that under normal circumstances subserves adaptive goals.</td>
<td>Habit</td>
</tr>
<tr>
<td>Dopamine, serotonin</td>
<td>Anterior medial OFC, cortico-limbic circuit, ventral limbic striatum (incl. ventral caudate), ventral tegmental area/substantia nigra</td>
<td>Processes by which the probability and benefits of a prospective outcome are computed and calibrated by reference to external information, social context (eg, group input, counterfactual comparisons), and/or prior experience. This calibration is influenced by pre-existing biases, learning, memory, stimulus characteristics, and deprivation states. Reward valuation may involve the assignment of incentive salience to stimuli.</td>
<td>Approach motivation Reward valuation</td>
</tr>
<tr>
<td>Adenosine, dopamine, GABA</td>
<td>Basolateral amygdale, dorsal ACC, ventral pallidum, ventral striatum (nACC), VTA</td>
<td>Processes by which the cost(s) of obtaining an outcome is computed; tendency to overcome response costs to obtain a reinforcer</td>
<td>Effort valuation</td>
</tr>
<tr>
<td>Dopamine, serotonin</td>
<td>Amygdala, basal ganglia, dorsal ACC, lateral habenula, orbitofrontal cortex, rostral medial tegmentum, substantia nigra/VTA, ventral striatum</td>
<td>A state triggered by exposure to internal or external stimuli, experiences or contexts that predict the possibility of reward. Reward expectation can alter the experience of an outcome and can influence the use of resources (eg, cognitive resources).</td>
<td>Reward expectancy</td>
</tr>
</tbody>
</table>

the group level; however, greater severity of negative and depressive symptoms is associated with reduced activation of the ventral striatum and orbitofrontal cortex, respectively.116,117

Outside of the schizophrenia-spectrum, individuals with major depression, anxiety disorders, neurocognitive disorders, and autism also display diminished self-reported and neurophysiological response to reward outcomes in areas like the ventral striatum, caudate, putamen, orbitofrontal cortex, ventromedial prefrontal cortex, anterior cingulate cortex, and insula.106-113

Thus, there is an important discrepancy in the area of initial reward responsiveness, which appears to be intact in chronic schizophrenia, but reduced in other schizophrenia-spectrum disorders and disorders outside the schizophrenia-spectrum that display motivational abnormalities.

Sustained Responsiveness to Reward Attainment. Although initial reward responsiveness may be intact in chronic schizophrenia, the ability to sustain that response after a reward outcome has terminated may be impaired. For example, while directly viewing pleasant stimuli, the affect modulated startle response is comparable between chronic schizophrenia patients and controls; however, after stimulus offset, only controls continue to show potentiation of this response.114 Similarly, when directly viewing pleasant photographs, chronic schizophrenia patients and controls show similar magnitude of activation in the dorsolateral prefrontal cortex, orbitofrontal cortex, ventromedial prefrontal cortex, basal ganglia, and amygdala; however, in the delay period following stimulus offset, there is reduced activation in the dorsolateral prefrontal cortex that predicts clinically rated anhedonia.115 Diminished ability to sustain affective response after stimulus offset has also been observed in bipolar disorder, but not depression.116,117

Since schizophrenia and bipolar disorder have more severe cognitive control deficits than depression, it is possible that prefrontally mediated deficits in sustaining reward response reflect a cognitive control impairment rather than hedonics.

Habit and Reward Learning. On implicit learning tasks that measure striatum-mediated habit-based learning that occurs over a number of trials, individuals with chronic schizophrenia typically show normal striatal/model-free learning rates.118-124 In contrast, individuals with major depression and Parkinson's disease evidence reduced striatal activation and retarded learning rates on such tasks.125-129

In contrast to habit learning, chronic schizophrenia has consistently been associated with impairments in making rapid, trial-by-trial behavioral adjustment on probabilistic reinforcement learning tasks that require forming more explicit top-down representations using the OFC.115,130-132 Greater severity of motivational symptoms is associated with more impairment in rapid/early learning, with some evidence that this association may be more severe for deficits in learning from positive than negative outcome feedback.60,70,92,118,130-135 Mechanisms underlying the deficit in learning from positive outcome feedback are unclear. Some computational modeling and neuroimaging studies implicate OFC-driven deficits in value representation, whereas others implicate positive prediction error signaling in the ventral striatum.67,70,131,132,135-138 Basic working memory impairments may moderate reinforcement learning deficits in chronic schizophrenia.139 Reinforcement learning deficits are also present in high-risk, first episode psychosis, and schizotypal populations, but in milder form than chronic schizophrenia.140,141 Like chronic schizophrenia, there is greater impairment for positive than negative outcome feedback and aberrant fronto-striatal activation during prediction error signaling may underlie reinforcement learning abnormalities in earlier phases of psychosis.140-145 Parkinson's disease is also associated with intact learning from negative feedback and impaired learning from positive feedback, and this trend is reversed by dopamine agonists.146 In contrast, in depression there is surprisingly intact performance on probabilistic reinforcement learning tasks that require explicit representations.147-150

Important discrepancies therefore exist across disorders in the areas of implicit and explicit reinforcement learning. In disorders with true hedonic deficits, impaired striatal activation may impede implicit reinforcement learning, but intact prefrontal function may overcome this deficit and allow more normal performance on probabilistic learning tasks that require explicit representations. In contrast, disorders with intact hedonics and more severe PFC-driven cognitive control deficits may be impaired at probabilistic reinforcement learning tasks due to trouble forming explicit value representations, but can perform more implicit reinforcement tasks that rely on the basal ganglia normally because they do not rely on top-down representations.

Approach Motivation. The next 4 sections evaluate subcomponents of the RDoC “approach motivation” construct.

Reward Expectancy. There is mixed evidence for group differences in ventral striatum activation in response to reward predictive cues among studies comparing schizophrenia and healthy control groups; however, there is consistent evidence for an association between greater blunting of the ventral striatum and volitional pathology.43,102 Similar to chronic schizophrenia, youth at ultra-high-risk for psychosis, first episode psychosis patients, and those with schizotypy show reduced activation of the ventral striatum during reward anticipation that predicts severity of negative symptoms.43,105,151-154 Consistent with a common mechanism for impairments in reward...
tions. Although reward incentives typically increase that result from poor maintenance of goal representa-

Action Selection. Schizophrenia patients display DLPFC-mediated impairment on cognitive control tasks that result from poor maintenance of goal representa-
tions at tasks where value representations must be generated, maintained, or updated, even when cognitive demands of tasks are minimal. However, the OFC has not been implicated, as would be expected; other mechanisms appear to contribute, including decreased deactivation of the default network, cognitive control regions, and prediction error signaling. First episode psychosis patients and youth at-risk for psychosis also demonstrate impairments in reversal learning and the Iowa Gambling task that are associated with negative symptoms. Similar reversal learning deficits are observed in major depression, PTSD, bipolar disorder, and autism spectrum disorders. Like schizophrenia, there is evidence that mechanisms other than the OFC contribute to these deficits (eg, striatal response).

Effort Valuation. Numerous studies have found impairment in physical or cognitive effort-cost computation in schizophrenia, with most reporting a significant association between willingness to work for rewards and voli-
tional symptoms. Similar to chronic schizophrenia, effort-cost computation is impaired in individuals with major depressive disorder who show poor integration of probability and magnitude information when deciding whether to work for rewards. However, effort-cost computation is spared or even superior in schizotypy and autism-spectrum samples that show an increased willingness to work for rewards. At present, mechanisms underlying effort valuation impairments in disorders are unclear because neuroimaging results have yet to be published.

Action Selection. Schizophrenia patients display DLPFC-mediated impairment on cognitive control tasks that result from poor maintenance of goal representa-
tions. Although reward incentives typically increase DLPFC activation and cause a shift toward less reactive and more proactive cognitive control strategies in healthy individuals, incentives fail to shift schizophrenia patients away from reliance on reactive control mechanisms. Although schizophrenia patients show similar activation of the DLPFC to controls in the context of sustained reward incentives that can be obtained for successful cognitive control task performance, individual differences in volitional symptoms are associated with reduced DLPFC activation as a function of reward incentives. General cognitive control deficits are also observed in depression, bipolar disorder, autism, and neurocognitive disorders; however, magnitude of impairment is typically not as severe as schizophrenia. Incentives modulate cognitive control normally in Parkinson’s disease, despite significant general control deficits. In depression, anxiety disorders, and bipolar disorder reward incentives have reduced impact on cognitive control processes; however, neuroimaging has yet to be conducted to determine whether mechanisms underlying action selection impairment are similar to schizophrenia.

Summary. Across DSM-5 disorders, there appear to be multiple pathways to the same common endpoints of motivational pathology (ie, equifinality). For example, in chronic schizophrenia, hedonic response is intact and mechanisms contributing to volitional pathology are subsequent to initial reward responsiveness; impairments in cortically mediated cognitive control processes may underlie abnormalities in several aspects of reward processing (eg, effort-cost computation, action selec-
tion, reward anticipation, rapid learning) that prevent intact hedonic responses from translating into moti-

Expressive Dimension

Fewer models have attempted to explain the pathophysi-
ology of expressive symptoms in chronic schizophrenia. When attempting to understand these expressive deficits, it is important to consider that they are more compli-
icated and potentially more subtle than what clinical rat-
ings suggest. A recent review found that clinical ratings of expressive deficits were profoundly different in patients with schizophrenia compared to nonpsychiatric controls (eg, on the order of 4–7 standard deviations), whereas objective analysis of their verbal and nonverbal behavior revealed much more benign anomalies (k = 13 studies, n = 480 patients; d = .80 for alogia, d = .36 for blunted vocal affect). In contrast, in disorders other than chronic schizophrenia that have hedonic deficits, diminished initial reward responsiveness may propagate forward, leading to down-stream impair-
ments in other aspects of reward processing (eg, implicit reinforcement learning, reward anticipation) that impede motivated behavior.

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we consider how expressive deficits may wax and wane as a function of hedonic, cognitive, and social systems.

It is worth noting that computational approaches to objectifying expressive behaviors have been applied to disorders outside of schizophrenia, and in many cases, have generally shown modest (at best) levels of expressive deficits. For example, measuring natural vocal expression with acoustic analysis has revealed modest, or at best, inconsistent abnormalities associated with autism, depression, mania, and schizotypal personality traits. On the other hand, there are domains of psychopathology that have yielded relatively consistent findings regarding expressive deficits. First, acoustic vocal anomalies have been consistently observed in neurodegenerative disorders, such as Parkinson’s and Alzheimer’s diseases. Second, efforts to identify acoustic correlates of depression and dementia using machine learning with large feature sessions comprising hundreds or thousands of variables, have demonstrated promising results. To our knowledge however, no machine learning studies have yielded algorithms that replicate or generalize across studies, samples and speaking tasks, so this remains an emerging field in need of further work.

Hedonic Systems. Historically, expressive deficits have been considered an extension of hedonic and volitional deficits in schizophrenia. With increased empirical efforts to isolate and objectify these expressive behaviors in schizophrenia, a more complicated picture is emerging, and it seems unlikely that expressive deficits wholly reflect disruptions in hedonics in chronic schizophrenia. As noted above, factor analysis of “next-generation” negative symptom clinical scales suggests that expressive symptoms are statistically independent of volitional symptoms. This finding is largely consistent with those from laboratory studies. For example, measures of computationally derived verbal production (eg, words and utterances produced) and vocal affect in patients with schizophrenia are largely uncorrelated with measures of affective experience. In contrast, in disorders where hedonic deficits are prominent (eg, depression, neurocognitive disorders), expression and experience are more tightly coupled.

Cognitive Systems (ie, Attention, Working Memory, Cognitive Control). If expressivity deficits do not reflect disruptions in positive valence systems in schizophrenia, and they are relatively benign much of the time, what could cause them? There is evidence to suggest that at least some expressive deficits may reflect disruptions in basic cognitive abilities. This is not surprising, in that effective verbal and nonverbal expression require coordinating a host of attentional, memory, language, social cognitive, and other functions, and that disruption in these abilities due to neuropathy, stress, or attentional distraction correlates with less effective expression in various patient populations. In patients with schizophrenia, measures of natural verbal production have been correlated with more global cognitive deficits. Moreover, using experimental “dual-task” designs, involving participants speaking while simultaneously conducting various working memory tasks, patients show reductions in verbal production at a greater magnitude than for nonpatients. Where pause durations nearly doubled in patients with schizophrenia compared to a mild increase in nonpsychiatric patients. Importantly, neither nonverbal expression (eg, vocal prosody) nor speech content (eg, identity, filler word use) was impacted by the cognitive load manipulation. This suggests that blunted vocal affect may not require attentional resources in the same manner as alogia, or that vocal expression is multi-faceted such that individuals can differentially allocate resources to specific functions to compensate for limitations in cognitive resources.

Cognitive-based mechanisms of expressivity deficits have been examined outside of schizophrenia. In the case of affective disorders (ie, depression, mania), both correlational and experimental studies suggest that pause times are associated with attentional dysfunctions, and that the magnitude of this effect is not statistically different as a function of schizophrenia, depression or mania diagnosis. In the case of schizotypal personality traits however, the results are more varied. Correlational studies have demonstrated relationships between acoustic speech features and cognitive performance on a standardized battery. However, experimental manipulation of attentional load has not led to abnormalities in pause times or any other speech features. This has been demonstrated in 3 studies to date, one of which found that the speech of individuals with schizotypy was abnormally resilient to attentional load. Collectively, these studies complement a large literature examining expressive deficits within the context of neurodevelopmental and neurodegenerative disorders, for which a number of expressive features have been identified and tied to specific motor control and other cognitive mechanisms. To our knowledge, however, factors potentially contributing to expressivity deficits outside of cognition, for example, involving emotion, social, temporal, diurnal, and other factors, have received limited empirical attention.

Expressivity as a Social Process. Within the RDoC framework, the constructs of “production of facial communication” and “production of nonfacial communication” overlap conceptually with diminished expressivity, and thus warrant mention here. However, we are aware of little research relating expressivity deficits to specific social-based mechanisms in psychopathology despite the fact that expressive behaviors arise from, and modulate in response to a wide range of social and emotional factors. Behavior that is considered typical in one context (eg, silence while
Negative Symptoms

watching TV with a friend) may be considered pathologi-
cal in another (eg, silence after asked about delusional
thought content). Thus, it stands to reason that
expressive behavior must be understood within its socio-
contextual circumstances, and that consequently, a broad
range of contextual factors may potentially contribute
to expressive deficits. Within schizophrenia, eg, reduced facial
expression could potentially reflect a myriad of “primary”
and “secondary” sources, including paranoia, confu-
sion during a social exchange, and trait social anhedonia.
Similar behavior could reflect an emotion regulation strat-
egy in depression to attenuate emotional experience during
social interactions, or even a social cognition deficit as in
autism-spectrum disorders.214,215 Moreover, given comor-
bidity across these disorders, expressive deficits could
reflect different sources within an individual, eg, as an
emotion regulation in one context and a social cognitive
in another. From this perspective, expressive deficits may
be best viewed as an equifinality reflecting a wide range of
potential socially-related mechanisms.

Conclusions

Based on the present review, we believe it is clear that
negative symptoms are a transdiagnostic construct
that occurs within and outside of the schizophrenia-
spectrum. There are no domains or features of nega-
tive symptoms that are pathognomonic, ie, specific to
schizophrenia as the prototypical disorder and not pres-
ent in other diagnoses. Rather, expressive and volitional
negative symptoms are common in several disorders
and form a continuous distribution of scores rang-
ing from healthy, to high-risk, to clinical populations.
Complicating comparisons across diagnostic categories,
there is considerable heterogeneity in terminology used
to describe negative symptom phenomenology through-
out the DSM-5. Standardization of negative symptom
terminology and assessments that can be used across
disorders will be an important step to take before progres-
sion can be made in determining candidate mechanisms
for treatment. The success of transdiagnostic research
endeavors may depend on whether diagnostic groups
included in a study are similar with respect to the pres-
ence of primary and secondary negative symptoms and
whether measures are administered to capture the pri-
mary/secondary and persistent/transient distinctions.

New transdiagnostic models of the etiology of expres-
sive and volitional pathology are also needed for treatment
progress to be made. Models developed for schizophrenia
have led to significant breakthroughs; however, these
models may not apply to other schizophrenia-spectrum
disorders or disorders outside of the schizophrenia-spect-
rum that display negative symptoms. There is clear evi-
dence for equifinality in both volitional and expressive
dimensions of negative symptoms across DSM-5 disor-
ders, ie, the same behavioral outcome/symptom is reached
via different mechanisms. Figure 4 presents a new model
depicting how 2 mechanistic pathways, a hedonic pathway
and a cognitive pathway, can lead to both expressive and
volitional negative symptoms. In disorders where hedonic
deficits are prominent (eg, depression), impaired initial
reward responsiveness may propagate forward, leading to
reductions in expressing emotion and engaging in goal-
directed behaviors. In contrast, in disorders where hedon-
ics are intact (eg, schizophrenia), but cognitive deficits are
prominent, expressive deficits may result from difficulty
coordinating the many cognitive processes needed to pro-
duce contextually appropriate verbal and nonverbal com-
munication. Similarly, motivational deficits may result
from higher-order cognitive impairments that impact the
ability to generate, update, and maintain explicit value
representations needed to make effort-cost decisions and
form action plans. Transdiagnostic studies are needed to
test hypotheses such as these about common and distinct
pathways to motivational and expressive negative symp-
toms. These studies should take the structure of negative
symptoms into account, making study design and analytic
decisions based on whether dimensional, categorical, or
hybrid conceptualizations best fit the selected sample.

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interest in relation to the subject of this study.

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