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Avolition and expressive deficits capture negative symptom phenomenology: Implications for DSM-5 and schizophrenia research

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Abstract

The DSM-5 formulation presents an opportunity to refine the negative symptom assessments that are crucial for a schizophrenia diagnosis. This review traces the history of negative symptom constructs in neuropsychiatry from their earliest conceptualizations in the 19th century. It presents the relevant literature for distinguishing between different types of negative symptoms. Although a National Institute of Mental Health consensus initiative proposed that there are five separate negative symptom domains, our review of the individual items demonstrates no more than three negative symptom domains. Indeed, numerous factor analyses of separate negative symptom scales routinely identify only two domains: 1) *expressive deficits*, which include affective, linguistic and paralinguistic expressions, and 2) *avolition* for daily-life and social activities. We propose that a focus on *expressive deficits* and *avolition* will be of optimum utility for diagnosis, treatment-considerations, and research purposes compared to other negative symptom constructs. We recommend that these two domains should be assessed as separate dimensions in the DSM-5 criteria.

Keywords

schizophrenia; negative symptoms; emotional expression; asociality; avolition; diagnosis

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Introduction

Clinical symptoms are the only criteria for diagnosing schizophrenia, so a great deal of attention centers on which constellations of symptoms can best define the disease for research purposes, clinical care and prognosis. “Negative symptoms” are those that refer to the loss or diminution of normal functions, such as expressiveness and motivation. While their diagnostic utility was officially recognized in the *Diagnostic and Statistical Manual, Third Edition-Revised* (DSM-III-R; American Psychiatric Association, 1987), debate persists regarding the best specifications and definitions of negative symptoms.

The reformulation of diagnostic criteria in the DSM-5 has prompted an outpouring of commentary and research on negative symptoms. The *National Institute of Mental Health* (NIMH) *Measurement and Treatment Research to Improve Cognition in Schizophrenia* (MATRICS) consensus panel selected five negative symptoms for further study: blunted affect, alogia, asociality, avolition, and anhedonia (Kirkpatrick, Fenton, Carpenter, & Marder, 2006). They proposed that a two-factor model may best account for negative symptoms: *blunted affect-poverty of speech* and *anhedonia-asociality-avolition*, although others have argued against including anhedonia as a negative symptom (Foussias & Remington, 2008).

Herein we review historical perspectives of negative symptoms, as these early theoretical frameworks continue to influence current conceptualizations of the construct. We then integrate these early theories with current research in a systematic and comprehensive review of studies on individual negative symptoms, and further examine this construct through a review of factor analytic studies. We propose that a focus on *expressive deficits* and *avolition* will be of the greatest utility for diagnosis, treatment-planning, and research purposes compared to other negative symptoms formulations and that these two domains should be assessed as separate dimensions.

History of Negative Symptoms

Conceptual History of Negative (and Positive) Symptoms in Neuropsychiatry

The conceptual history of “negative symptoms,” versus “positive symptoms” in neuropsychiatry must be differentiated from our current conceptualization of “negative symptoms” in schizophrenia. The distinction was first introduced in the context of epilepsy by John Russell Reynolds (1861). He described negative symptoms as the loss of “vital properties” resulting in paralysis, anesthesia, or other defects, whereas positive symptoms were an excess of vital properties, such as spasms, pain, or convulsions. He conceptualized positive and negative symptoms as separate and independent manifestations of a single pathology (Berrios, 1985) (figure 1a).

Influenced by evolutionary theories of brain development, Hughlings Jackson (1958) proposed that negative and positive symptoms were separate inter-related neurological constructs. He theorized that complex neural systems evolved to inhibit lower level rudimentary neural activity for the purposes of higher level executive functioning. If higher systems were damaged then primitive responses could not be inhibited and positive symptoms would be released to dominate the clinical presentation (figure 1b). Following Jackson’s ideas, Henri Ey (1962) hypothesized that negative symptoms in psychiatry arose from organic pathology and he considered positive symptoms to be restitutive actions orchestrated by unaffected mental functions (figure 1c). There is a renewed focus on these early theoretical models concerning the biological basis for negative and positive symptoms in schizophrenia as investigators explore the relationship between neuropathology and symptom onset (e.g. Daniel Weinberger, 1987).

The Early Descriptions of Negative Symptoms in Schizophrenia: Kraepelin and Bleuler

Emil Kraepelin (1919/1971) noted “indifference,” “lack of interest,” and deficits in “higher [logical, moral, artistic and religious] feelings” in patients with dementia praecox. Kraepelin had been a student of Wilhelm Wundt who developed hierarchical and combinatory models to explain psychological functioning. In keeping with this teaching, Kraepelin proposed that these symptoms represented a simple pathological deficit in emotion. In this view, “dull appearance” (blunted affect) was secondary to the absence of feelings (Kraepelin, 1899/1990). A core defect in emotional experience, at a higher level, caused poor motivation, which was observable as indifference and lack of interest. The affective deficit drove an absence of pleasure (anhedonia) from activities and, more fundamentally, an absence of boredom, which together caused the “complete loss of volitional impulse.” The absence of a “desire for activity” in the patients produced their indifference to the external world and to themselves, including their physical health, which resulted in poor hygiene, decreased pain sensations and mental poverty.

Eugen Bleuler (1908/1950) adopted Kraepelin’s symptom descriptions but differentiated them into *Fundamental Symptoms*, which were necessary to make a schizophrenia diagnosis, and *Accessory Symptoms*, which were not specific to schizophrenia. At the psychopathological level, he distinguished *Primary Symptoms*, which were manifestations of the psychopathological process, from *Secondary Symptoms*, which were the reaction of intact psychological functions to the pathological processes. Bleuler hypothesized that the primary psychopathological process in schizophrenia was a loosening of associations (or disconnection) which operated at all psychic levels – from concrete ideas to complex psychic structures. Bleuler viewed the same signs differently for phenomenological versus psychopathological interpretations. Thus, while affect was impaired at the phenomenological level, giving the impression of indifference, Bleuler believed that affectivity as a mental function was intact: “Thus there can be no doubt at all that the psyche’s capacity to produce affects has not disappeared in schizophrenia” (Bleuler, 1908/1950, p 47). This foreshadowed recent findings that many patients with deficits in emotional expression have intact emotional experiences (Cohen & Minor, 2010; Horan, Green, Kring, & Nuechterlein, 2006; Trémeau, Antonius, Cacioppo, Ziwich, Butler et al., in press).

The Emergence of the Study of Negative Symptoms in Schizophrenia

Research and theory from the 1950’s through the 1970’s shifted to a focus on positive symptoms. Kurt Schneider (1959) referred to positive symptoms as “first-rank symptoms” and believed that they differentiated schizophrenia from all other psychoses. In addition, medications were identified that could treat positive symptoms and this focus on positive symptoms was reflected in the release of the DSM-III in 1980.

Studies of other symptoms continued nonetheless. Venables and Wing (1962) identified withdrawal as a dimension in schizophrenia, “defined by underactivity, slowness, lack of conversation, lack of friends, avoidance of others, poor personal hygiene, carelessness about appearance, and lack of interests” (p 115).

It was probably Snezhnevsky (1968) who first to applied the positive versus negative distinction to schizophrenia symptoms. Strauss, Carpenter, & Bartko (1974) consolidated this typology of schizophrenia into three dimensions: negative symptoms (blunting of affect, apathy and certain thought disorders), positive symptoms, and disorders of social relationships. This distinction was rapidly embraced by researchers as it helped to reduce and understand the heterogeneity of the illness (Andreasen, Flaum, Swayze, Tyrrell, & Arndt, 1990; Carpenter, Buchanan, & Kirkpatrick, 1991; Crow, 1980a, 1980b). These new

investigations set the stage for rehabilitating negative symptoms as important for the phenomenology of schizophrenia. In the 1987 DSM-III-R *flat affect* was listed as a distinct symptom and *avolition* was added to the prodromal/residual criteria of schizophrenia. In the 2000 DSM-IV-TR negative symptoms were listed as one of five characteristic schizophrenia symptoms, with *affective flattening, alogia*, and *avolition* provided as the examples.

These historical perspectives provide a frame of reference for understanding current research and assessment of negative symptoms. As we prepare for the anticipated DSM-5 diagnostic criteria it is important to remember that inclusion or omission of negative symptoms in previous editions of the DSM greatly impacted schizophrenia research and treatment approaches (Andreasen & Carpenter, 1993). Likewise, the complexity of research regarding negative symptoms is still largely driven by the symptom definitions in the DSM. As we review below, refinement of these definitions and our understanding of negative symptoms as a construct is necessary to enhance research and advance treatment of schizophrenia.

Current Research on Negative Symptoms

Primary Negative Symptoms and Deficit Symptoms

Carpenter and colleagues identified a subgroup of cases with *enduring* and *primary* negative symptoms which they categorized as the “deficit syndrome” (Carpenter, Heinrichs, & Wagman, 1988; Kirkpatrick, Buchanan, McKenney, Alphs, & Carpenter, 1989). This syndrome is found in approximately 15% of first-episode patients and 25–32% of chronic schizophrenia patients (Kirkpatrick, Buchanan, Ross, & Carpenter, 2001; Peralta & Cuesta, 2004). The identification of deficit symptoms requires distinguishing between primary and secondary negative symptoms (Carpenter et al., 1988). Primary negative symptoms are manifestations of core pathology, while secondary negative symptoms are consequences of the illness process (Carpenter et al., 1988; Kirkpatrick, Buchanan, Breier, & Carpenter, 1993). For example a lack of facial expression can be a negative symptom, a symptom of depression (Trémeau et al., 2005) or a side effect of antipsychotic medications (e.g. *extrapyramidal symptoms*). Similarly, a lack of social relationships can be a primary negative symptom (*asociality*); but if the same behavior is a consequence of paranoia or the lack of environmental stimulation, then low social interest is a secondary symptom. Differentiating primary from secondary negative symptoms is not straight forward as the procedures can be subjective and easily confounded by the accuracy of historical information.

The Individual Negative Symptoms

Affective Flattening—The definition of *flattened affect* includes diminished facial emotional expression. On many scales the affective flattening items also tap psychomotor and social deficits, including *poor eye contact, decreased spontaneous movement, and lack of spontaneity* (Abrams & Taylor, 1978; Alphs, Summerfelt, Lann, & Muller, 1989; Andreasen, 1989; Kay, Fiszbein, & Opler, 1987; Kirkpatrick et al., 1989; Trémeau et al., 2008). These items reflect the direct expressions of an emotion and other types of expression/behavior that are linked to emotions. Recent research shows that affective flattening captures *facial emotional expressions, coverbal behavior and prosody* (Kirkpatrick et al., 2006). Coverbal behavior includes the hand, head and facial movements that accompany speech (Hadar, Wenkert-Olenik, Krauss, & Soroker, 1998). Compared to nonpatient controls, subjects with schizophrenia show fewer body and facial movements and smiles, and less social and coverbal behavior (Brune et al., 2008; Davison, Frith, Harrison-Read, & Johnstone, 1996; Trémeau et al., 2005; Troisi, Spalletta, & Pasini, 1998). Coverbal gestures correlate with negative symptoms (Brune et al., 2008) and cluster with facial emotional expressions (Trémeau et al., 2008). Prosody refers to features of speech including

stress, intonation (speech melody), rhythm, tempo, loudness, voice quality and pausing. Prosody, like coverbal gestures and facial expressions, is a paralinguistic features that provides "extra" information that is not explicitly contained in the lexical and syntactic make-up of a sentence, such as cues to information structure, feedback, turn-taking, and emotional or attitudinal aspects of speech, including sarcasm (Grandjean, Banziger, & Scherer, 2006).

Alogia—*Alogia* is a decrease in verbal output or verbal expressiveness, often referred to as *poverty of speech*. The Scale for the Assessment of Negative Symptoms (Andreasen, 1989), groups alogia with such speech symptoms as *impoverished content of speech*, *blocked speech*, and *increased latency of response*. However, poverty of content of speech more consistently associates with disorganization than with negative symptoms. Increased latency of response also does not cluster with negative symptoms (Kelley, van Kammen, & Allen, 1999), demonstrating stronger associations with depressive symptoms (Knight & Valner, 1993). Finally, the clinical assessment of increased latency of response is not reliable (Alpert, Pouget, Sison, Yahia, & Allan, 1995; Alpert, Shaw, Pouget, & Lim, 2002).

Anhedonia—Anhedonia is the inability to experience pleasure from positive stimuli. It has long been considered to be a core symptom of schizophrenia (Andreasen, 1982; Meehl, 1962) and self-report questionnaires consistently find anhedonia in schizophrenia (Horan, Kring, & Blanchard, 2006), including social (Blanchard, Horan, & Brown, 2001; Blanchard, Mueser, & Bellack, 1998; Katsanis, Iacono, & Beiser, 1990) and physical anhedonia (Burbridge & Barch, 2007). These are particularly endorsed by deficit syndrome cases (Kirkpatrick & Buchanan, 1990). Social anhedonia, moreover, may predict the development of a schizophrenia related disorder (Kwapil, 1998). However anhedonia findings have been challenged by laboratory evocative studies that take place in controlled environments and use standard stimuli for all participants. Surprisingly, patients with schizophrenia, including those with primary negative symptoms, report comparable levels of pleasure for positive/pleasant stimuli as the non-patient controls (Trémeau, Antonius, Cacioppo, Ziwich, Jalbrzikowski et al., 2009; see reviews in Cohen & Minor, 2010; Kring & Moran, 2008; Trémeau, 2006).

The validity of the anhedonia questionnaires commonly used in schizophrenia research has been questioned (Germans & Kring, 2000; Linscott, 2007; Trémeau, Antonius, Cacioppo, Ziwich, Jalbrzikowski et al., 2009). Perhaps the evocative studies are less cognitively demanding than self-report approaches, which often rely on complex cognitive processes, integrate various affective processes, and are subject to systematic biases (Barrett & Nelson-Goens, 1997; Robinson & Clore, 2002). It is also possible that self reported anhedonia reflects high rates of co-morbid depression (Kollias et al., 2008). Based on the laboratory data showing intact emotional reactivity, some have recommended that anhedonia not be included in the negative symptoms construct (Foussias & Remington, 2008). However, other impairments in the processing of emotions may still contribute to negative symptoms. For example, authors have shown a disconnect in the ability for schizophrenia subjects to generate behavioral motivation from pleasurable experiences (Heerey & Gold, 2007; Trémeau et al., in press). Other authors have distinguished anticipatory pleasure (the experience of pleasure related to future activities) from consummatory pleasure and hypothesized that anticipatory but not consummatory pleasure is impaired in schizophrenia (Gard, Kring, Gard, Horan, & Green, 2007). Although these preliminary findings and hypotheses do not warrant present consideration of anhedonia as a separate negative symptom dimension, they provide guidance for future research.

Asociality—Andreasen (1989) defined *asociality* as "lack of involvement in social relationships of various kinds" (p56). However, the limited social engagement observed in

schizophrenia may not necessarily reflect psychopathology (Carpenter et al., 1988). Compared to healthy control subjects, fewer individuals with schizophrenia have intact families or as much access to contemporary communicative devices such as internet and e-mails. They may be excluded from gatherings as victims of stigma and their economic status further limits their ability to participate in social activities. All these extrinsic factors can contribute to their constrained social lives. To increase its specificity, asociality should include both a reduction in social activity as well as decreased interest/desire in forming relationships with others (Kirkpatrick et al., 2006). Then asociality actually reflects an impaired motivation for social contact (avolition), and may be defined as the lack of self-initiated social interactions. Based on the above reports, asociality does not adequately constitute an independent subdomain, and avolition is a better term to capture this concept.

Avolition (Amotivation)—This includes both a subjective reduction in interests, desires and goals and a behavioral reduction of self-initiated and purposeful acts. Motivational deficits in schizophrenia have always been considered core symptoms, yet the optimal means to assess motivation is still debated (Barch, 2005), and the inclusion of the subjective aspect of motivation may have limited validity. In the general population, self-report measures of motives predict only immediate responses to specific situations (McClelland, Koestner, & Weinberger, 1989). In clinical populations, however, self-reported motivation has no predictive value; not even for immediate behaviors (Marin, Biedrzycki, & Firinciogullari, 1991). One self-report questionnaire study of intrinsic motivation (the interest in and enjoyment of activity for its own sake) could not differentiate schizophrenia patients from healthy participants (Barch, Yodkovik, Sypher-Locke, & Hanewinkel, 2008).

The lack of self-initiated daily life activities and other associated signs of amotivation may best be assessed by instruments that measure overt behaviors. Individuals with schizophrenia show a reduction in their actions (reviewed in Morrens, Hulstijn, & Sabbe, 2007) that spans motor retardation/physical anergia (Jogems-Kosterman, Zitman, Van Hoof, & Hulstijn, 2001), decreased grooming and hygiene (Brewer, Edwards, Anderson, Robinson, & Pantelis, 1996), less involvement with work, school, vocational programs, or leisure activities (Vuksic-Mihaljevic, Mandic, Barkic, & Laufer, 1998), as well as less engagement in social activities (Vuksic-Mihaljevic et al., 1998).

Avolition in daily-life is distinct from poor social function, which can result from many psychopathological processes or environmental circumstances. A patient may not watch TV because he/she has no access to a television or thinks that a camera in the set is watching him/her. Another patient may have no regular contact with family or peers because they are not available or there is no easy way to communicate with them. Neither patient would be considered to have avolition, yet their activity level and social function are reduced. Avolition should be specified as reductions in self-initiated involvement in activities that are available to the patient and encompass more than just social activities.

Unfortunately, schizophrenia subjects are not very reliable in reporting their level of activity (Bowie et al., 2007; Trémeau et al., 2008). Objective observations can easily be done with inpatients and existing scales such as the Scale for the Assessment of Negative Symptoms (Andreasen, 1989) and the Motor-Affective-Social Scale (Trémeau et al., 2008) are quite suited for this approach. The major difficulty resides in the assessment of leisure, work, school and social activities with outpatients especially if no reliable informants are available. New and innovative instruments are needed with a focus on identifying which motivational processes are specifically impaired in schizophrenia.

Major Negative Symptom Rating Scales

In 1978 Abrams and Taylor published the Emotional Blunting Scale, the first scale to focus specifically on negative symptoms in schizophrenia. It contained 16 items that assessed affect, social behavior, and motivation. Shortly after, Andreasen (1982) developed the Scale for the Assessment of Negative Symptoms, which measured 25 negative symptoms, grouped into five sub-domains: *affective flattening, alogia, avolition/apathy, anhedonia/asociality*, and *attentional impairment*. It spurred considerable research on the construct validity of the negative symptom dimension and on the face validity of each item (see Earnst & Kring, 1997; Knight & Valner, 1993). Subsequently a modified version of the Scale for the Assessment of Negative Symptoms was published in which several items were dropped (Andreasen, 1989) and other new rating instruments were developed.

One such instrument, the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987) has been widely used in schizophrenia clinical trials. The PANSS is a 30-item symptom rating scale that was originally divided into three subscales: *positive, negative*, and *general psychopathology*. However, replication of the original PANSS factors has been problematic. Some studies support five PANSS symptom domains: *positive, negative, excited, depression/anxiety*, and *cognitive/disorganized* (Emsley, Rabinowitz, & Torreman, 2003; Kay & Sevy, 1990; Lancon, Aghababian, Llorca, & Auquier, 1998; Lindenmayer, Bernstein-Hyman, & Grochowski, 1994; Lindenmayer, Grochowski, & Hyman, 1995; Lykouras et al., 2000; Van den Oord et al., 2006; White, Harvey, Opler, & Lindenmayer, 1997).

Other rating scales developed during this time include the Negative Symptom Assessment (Alphs et al., 1989) and the Schedule for the Deficit Syndrome (Kirkpatrick et al., 1989). The Schedule for the Deficit Syndrome differs from the other negative symptom rating scales in that it was developed specifically to differentiate between primary and secondary negative symptoms and to assess the presence or absence of the Deficit Syndrome. A more recent measure, the aforementioned Motor-Affective-Social Scale (Trémeau et al., 2008), is specifically based on observable, well defined, and easily measurable behaviors such as coverbal hand gestures, spontaneous and voluntary smiles, and speech flow.

Because of disagreement among researchers regarding which symptoms to include in the negative symptom construct, none of these measures have complete overlap in symptoms assessed or in the language used to assess the symptom construct. Only *flat affect* is measured in all scales, followed by *alogia* (present in all but one rating scales), then *asociality, anhedonia* and *avolition* (for reviews see Earnst & Kring, 1997; Fenton & McGlashan, 1992; Foussias & Remington, 2008; Silk & Tandon, 1990).

Factor Analytic Studies of Negative Symptoms

Factor analytic approaches are a promising solution to the difficulty in measuring and conceptualizing negative symptoms. Overall, factor analytic studies of the different negative symptom rating scales support two distinct negative symptom domains: “Expressive Deficits,” which includes *flat affect* and *alogia*, and “Avolition,” which includes *amotivation, anhedonia* and *asociality*.

The Scale for the Assessment of Negative Symptoms has been the most widely studied negative symptom scale using factor analysis and the majority of these studies have likewise supported separate factors for “Expressive Deficits” and “Avolition” (table 1; Emsley et al., 2001;Keefe et al., 1992;Kelley et al., 1999;Malla et al., 2002;Minas, Klimidis, Stuart, Copolov, & Singh, 1994;Mueser, Sayers, Schooler, Mance, & Haas, 1994;Peralta & Cuesta, 1999;Sayers Curran, & Mueser, 1996;Toomey et al., 1997). The Scale for the Assessment of Negative Symptoms items *inappropriate affect, poverty of content of speech*, and

inattentiveness do not segregate with other negative symptoms and they form a third factor when they are entered in the analyses (Keefe et al., 1992;Malla et al., 2002;Mueser et al., 1994;Peralta & Cuesta, 1999;Sayers et al., 1996). Although Peralta and Cuesta (1995) found support for five separate factors as defined in the Scale for the Assessment of Negative Symptoms, high intercorrelations existed between "Affective Flattening" and "Alogia," and between "Anhedonia/Asociality" and "Avolition/Apathy." Many studies find *alogia* to load onto the "Expressive Deficits" factor (Emsley et al., 2001;Kelley et al., 1999;Malla et al., 2002) or have high interfactor correlations (Sayers et al., 1996) suggesting that alogia might be closely related to emotional expression, rather than a separate factor.

Fewer studies have examined the factor structure of negative symptoms in other rating scales, but the results are consistent with the Scale for the Assessment of Negative Symptoms studies. Factor analytic studies of the Schedule for the Deficit Syndrome (Kirkpatrick et al., 1989) support two separate factors for "Diminished Emotional Expression" and "Avolition" (table 1; Kimhy, Yale, Goetz, McFarr, & Malaspina, 2006; Nakaya & Ohmori, 2008). A comparable solution identified in the Motor-Affective-Social Scale showed two factors: a "Motor-Affective" factor (including spontaneous and voluntary smiles and coverbal gestures) and a "Motor-Social" factor (including motor retardation, personal hygiene, attendance at groups and activities, and verbal interaction), with *alogia* loading onto both factors (Trémeau et al., 2008).

Separate factors for "Expressive Deficits" and "Avolition" have been identified in heterogeneous groups of patients with psychotic disorders (table 1; Peralta & Cuesta, 1995,1999;Toomey et al., 1997) as well as schizophrenia spectrum patients (Emsley et al., 2001;Keefe et al., 1992;Kelley et al., 1999;Malla et al., 2002;Mueser et al., 1994;Sayers et al., 1996;Trémeau et al., 2008) and deficit syndrome patients (Kimhy et al., 2006;Nakaya & Ohmori, 2008). Furthermore, these two factors were found in patients who were on medication (Kimhy et al., 2006;Mueser et al., 1994;Sayers et al., 1996;Trémeau et al., 2008) and who were medication free (Kelley et al., 1999), and in first-episode (Malla et al., 2002) and chronic (Keefe et al., 1992) patients. Separate factors for "Expressive Deficits" and "Avolition" appear to hold up cross-culturally as well. Factor analyses of data collected from patients in the United States (Keefe et al., 1992;Kelley et al., 1999;Kimhy et al., 2006;Mueser et al., 1994;Sayers et al., 1996;Trémeau et al., 2008), Canada (Malla et al., 2002), Spain (Peralta & Cuesta, 1999), South Africa (Emsley et al., 2001), Australia (Minas et al., 1994), and Japan (Nakaya & Ohmori, 2008), all supported "Expressive Deficits" and "Avolition" as separate factors, providing further evidence that deficits in emotional expression and volition are two distinct symptom domains in schizophrenia that encompass all negative symptom variance.

There are exceptions and not all negative symptom scales reveal two primary factors. A principal components analysis of the Negative Symptom Assessment identified seven factors (Alphs et al., 1989) and a later study determined that a six-factor solution was superior (table 1; Axelrod, Goldman, Woodard, & Alphs, 1994). One possible explanation for the failure to replicate a two factor model is that most of the items in the "Communication" factor (e.g. *prolonged time to respond*, *impoverished speech content*, *blocked speech*) are not supported as negative symptoms (reviewed above). Additionally, other items (e.g. *disorientation*, *poor memory*) are more closely related to disorganization and cognition, which have also not been supported as negative symptoms. Despite the results from the Negative Symptom Assessment studies, in accord with a previous review on the Scale for the Assessment of Negative Symptoms (Blanchard & Cohen, 2006), it can be concluded that negative symptoms, though multidimensional, can be collapsed across two main factors, "Expressive Deficits" and "Avolition."

Discussion and Implications for DSM-5

From our review of the history of negative symptoms, as well as current research on this construct, we have shown that negative symptoms in schizophrenia can be mapped onto two phenomenological domains: “Expressive Deficits,” which represents a constellation of expressive gestures including speech, facial expression and other non-verbal signs, and “Avolition,” which encompasses *amotivation* and *asociality*.

The concept of “negative symptoms” has a long history in neuroscience and the delineation of negative symptoms in schizophrenia was based on observation and theory long before any empirical studies found evidence for their clustering. Kraepelin identified most of the currently studied negative symptoms and grouped them according to their common psychopathology, a deficit in the process of emotions. He believed negative symptoms were the result of a disconnect between cognition and emotion. When negative symptoms were united in the 1970’s and grouped in opposition to the “favored” positive symptoms, what was a disconnection between cognitions and emotions became a loss of normal function, a deficit. Whereas Bleuler thought that affectivity as a mental function was intact, psychiatrists in the 1980’s thought of negative symptoms (and negative schizophrenia) as permanent and inaccessible to treatment.

However, current understanding of cognitive processes in schizophrenia suggests that defining negative symptoms as a loss of function is problematic. Some investigators have shown that positive symptoms such as hallucinations and delusions are linked to impairments that are the absence of normal cognitive functions such as deficits in source monitoring, poor executive functioning, and jumping to conclusions (Bentall et al., 2009; Brunelin et al., 2007). Despite these findings that hallucinations and delusions may result from the loss of normal functions, these psychotic symptoms are clearly different from negative symptoms.

We argue that negative symptoms are heterogeneous and distinct from other symptoms of schizophrenia. Redefining negative symptoms as specific and separate impairments in emotional expressiveness and volition offers the best approach to this construct for both research and clinical treatment. This is supported by our review of factor analytic studies of negative symptoms, which consistently show “Expressive Deficits” and “Avolition” as two separate negative symptom domains.

These two domains differ in their assessment and our knowledge of their etiology. Expressive deficits can be observed during a clinical interview, whereas the diagnosis of avolition needs specific inquiries to be ascertained. Similarly, expressiveness is a constellation of observable behaviors, whose common or unique psychopathologies remain to be clarified, whereas avolition refers to a specific psychopathological process, whose behavioral manifestations need clarification.

We propose that impairments in expressiveness and volition are distinct domains and should be assessed separately in DSM-5. Separating these concepts provides a more focused approach for research on negative symptoms. Likewise, distinguishing between these two domains offers the best hope for developing new treatment approaches that target these specific core phenomena. Based on our review of research on negative symptoms, we recommend that ratings only consider behavioral manifestations of these two dimensions which may be exhibited as follows:

a. For expressiveness: Four types of behavior are the most relevant and their impairments can be easily observed by clinicians and researchers: Communicative facial expressions, prosody, hand coverbal gestures, and language output.

b. For volition: Reduction in self-initiated and maintained behaviors can be observed in four categories: spontaneous motor activity, grooming/hygiene, work/recreation/leisure, and social engagement.

To be classified as negative symptoms, and to have a diagnostic value, deficits in expressiveness and volition should be separated from other phenomenological components of psychotic disorders. To achieve this, the symptoms must be distinct from or present in the absence of clinically-significant depressed mood or anhedonia, serious extrapyramidal side-effects, and active avoidant behaviors (i.e. due to anxiety or paranoia).

Initially negative symptom classification was developed within the fields of psychopathology and clinical phenomenology. Current research in schizophrenia, however, is driven by cognitive, affective and social neurosciences that influence and enhance our symptom classification paradigms. Expressive deficits can be considered a specific deficit in social communication, overlapping with affective processes. Avolition can be seen as a deficit in functional outcomes until we can further identify which motivational processes are impaired in schizophrenia. Although, clarification is still needed regarding the nature of anhedonia and other affective processes in schizophrenia, at this time we do not consider anhedonia to fall under either domain. We recommend additional research on anticipatory anhedonia as it relates to avolition in schizophrenia. Future studies are also needed to clarify whether expressive deficits and avolition are merely separate observable dimensions or whether they have different neurobiological underpinnings requiring separate treatment approaches.

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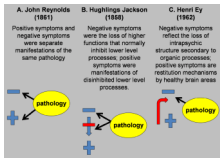

Figure 1.
Early conceptualizations of positive and negative symptoms in neuropsychiatry

Table 1

Summary of factor analytic studies of negative symptoms.

Author	Measure	Participants	Negative Symptom Factors
Alphs, et al. (1989)	NSA	100 schizophrenia spectrum patients	1) Affect/Emotion; 2) External Involvement; 3) Retardation; 4) Personal Presentation; 5) Thinking; 6) Interpersonal Interest; 7) Blocking
Keefe, et al. (1992)	SANS	130 chronic male schizophrenia inpatients	1) Diminished Expression; 2) Social Amotivation; 3) Disorganization*
Axelrod, et al. (1994)	NSA	233 schizophrenia inpatients	1) Communication; 2) Emotion/Affect; 3) Social Involvement; 4) Motivation; 5) Gross Cognition; 6) Retardation
Minas, et al. (1994)	SANS/SAPS	114 psychotic disorder patients	1) Negative Signs; 2) Social Dysfunctions
Mueser, et al. (1994)	SANS	207 schizophrenia patients	1) Affective Flattening or Blunting, 2) Avolition-Apathy and Anhedonia-Asociality, 3) Alogia and Inattention*
Peralta and Cuesta (1995)	SANS	253 schizophrenia inpatients	1) Affective Flattening; 2) Alogia; 3) Avolition-Apathy; 4) Anhedonia-Asociality, 5) Attention*
Sayers, et al. (1996)	SANS	437 schizophrenia spectrum inpatients	1) Diminished Expression; 2) Social Amotivation; 3) Inattention-Alogia*
Toomey, et al. (1997)	SANS/SAPS	549 psychotic disorder and mood disorder patients	1) Diminished Expression; 2) Disordered Relating
Peralta & Cuesta (1999)	SANS/SAPS	660 psychotic inpatients	1) Poverty of Affect/Speech; 2) Social Dysfunction; 3) Attention*
Kelley, et al. (1999)	SANS	93 male schizophrenia patients	1) Diminished Motivation; 2) Affective Flattening
Emsley, et al. (2001)	SANS/SAPS	422 schizophrenia patients	1) Diminished Expression; 2) Disordered Relating
Malla, et al. (2002)	SANS	110 first-episode schizophrenia spectrum patients	1) Flat Affect/Alogia; 2) Avolition/Anhedonia; 3) Attention*
Kimhy, et al. (2006)	SDS	52 deficit syndrome schizophrenia inpatients	1) Avolition; 2) Emotional Expression
Nakaya and Ohmori (2008)	SDS	70 deficit syndrome schizophrenia inpatients	1) Avolition; 2) Poor Emotional Expression
Trémeau et al. (2008)	MASS	101 schizophrenia inpatients	1) Motor-Affective; 2) Motor-Social

Notes: * Attention deficits and inappropriate affect are not negative symptoms; therefore the studies that included them in their factor analyses introduced a bias. NSA = Negative Symptom Assessment SANS/SAPS = Scale for the Assessment of Negative Symptoms/Scale for the Assessment of Positive Symptoms; SDS = Schedule for the Deficit Syndrome