

Anhedonia in Schizophrenia: A Review of Assessment Strategies

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Anhedonia, the diminished capacity to experience pleasant emotions, is a common, treatment-resistant feature of schizophrenia that is often included among the negative symptoms of this disorder. This selective review describes the 3 most commonly used approaches to assess anhedonia in schizophrenia: interview-based measures, self-report trait questionnaires, and laboratory-based assessments of emotional experience. For each assessment approach, psychometric properties, relationships to other symptoms and features of schizophrenia, and relationships with the other assessment approaches are evaluated. It is concluded that anhedonia can be reliably assessed and constitutes a distinctive, clinically important aspect of schizophrenia that should be included in a comprehensive evaluation of negative symptoms. Current efforts to define more precisely the nature of the hedonic deficit in schizophrenia are discussed, and recommendations for optimal assessment of anhedonia in clinical trials of novel treatments for negative symptoms are provided.

Introduction

Anhedonia is defined as a diminished capacity to experience pleasant emotions¹ and is commonly included among the negative symptoms of schizophrenia.² Since the writings of Kraepelin³ and Bleuler,⁴ anhedonia has figured prominently in clinical descriptions of the core deficits of schizophrenia. Major theorists such as Rado⁵ and Meehl⁶ assigned a central role to anhedonia in their etiological models of schizophrenia, positing that anhedonia is an indicator of genetic vulnerability to schizophrenia and a critical determinant of the debilitating social impairments associated with this disorder. Empirical research on anhedonia was facilitated in the late 1970s and early 1980s by the development of clinical

symptom rating scales⁷ with acceptable levels of reliability and psychometrically sound self-report trait questionnaires⁸ to assess decreases in the experience of pleasure in both schizophrenia patients and high-risk populations.

In recent years clinical research on anhedonia has benefited from the application of methods and theories derived from the burgeoning field of affective science, which has enabled researchers to investigate patterns of emotional responding in schizophrenia patients in controlled laboratory studies. Through the use of these various assessment strategies, a considerable body of research has amassed that attests to the clinical significance of anhedonia and its role within the broader construct of negative symptoms. It has also become clear that, like most negative symptoms, anhedonia is inadequately treated by currently available pharmacological and psychosocial interventions. The development and evaluation of new treatments will be most effectively accomplished through the use of methods that optimally assess anhedonia and are sensitive to potential treatment effects.

This selective review describes the 3 major approaches that have been used to assess anhedonia in schizophrenia: interview-based instruments, self-report trait measures, and laboratory-based assessments of emotional experience. For each assessment approach, we briefly describe the most commonly used instruments or paradigms, their psychometric characteristics, their relationships to other symptoms and features of schizophrenia, and their strengths and limitations for use in treatment studies. This is followed by an integration of findings across these 3 methods and a description of current research efforts aimed at specifying the precise nature of anhedonia in schizophrenia. Finally, we provide recommendations for optimal assessment of anhedonia in treatment studies that are aimed at ameliorating anhedonia and other negative symptoms.

Interview-Based Assessment

Available Instruments

Anhedonia is most commonly assessed in the context of a semi-structured interview. These clinical interviews are conducted by a researcher or clinician with extensive training in the careful probing that is required to elicit information about the patient's daily activities and subjective emotional experiences. Several frequently

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used interview-based measures of negative symptoms include items that are conceptually related to anhedonia,² including the Scale for the Assessment of Negative Symptoms (SANS),⁷ the Schedule for the Deficit Syndrome (SDS),⁹ the Positive and Negative Syndrome Scale (PANSS),¹⁰ and the Scale for Emotional Blunting (SEB).¹¹ Each of these instruments assesses how frequently patients engage in recreational and social activities of various kinds. However, the instruments differ considerably in terms of how patients' emotional experiences during these types of activities are assessed, how specifically these assessments focus on reductions in pleasant emotions, and how consideration of patients' emotional experiences figures into the overall ratings of the relevant items on these instruments.

Anhedonia is most directly and comprehensively assessed by the Anhedonia-Asociality subscale of the SANS.⁷ This subscale was designed to assess either difficulties or reductions in experiencing interest or pleasure, which may be expressed as a loss of interest in pleasurable activities, an inability to experience pleasure when participating in activities that are normally considered pleasurable, or a lack of involvement in social relationships of various kinds. The subscale consists of 4 items that cover recreational interests and activities, sexual interest and activities, ability to feel intimacy and closeness, and relationships with friends and peers. These 4 items, as well as a global summary score, are rated on a 0 (not at all) to 5 (extreme) Likert scale. The subscale also originally included an item to rate subjective awareness of Anhedonia-Asociality, although this item is typically excluded from analyses of this subscale. There is considerable variability across studies in the sources of information (eg, patient interview alone versus the addition of information from family members, medical records, etc) and the time period covered (ranging from 1 week to several months) in rating this subscale.

The SANS Anhedonia-Asociality subscale has several strengths in the assessment of anhedonia. Patients are queried not only about how frequently they engage in recreational and social activities, but they are also directly asked about how much they enjoy and are interested in those activities in which they do engage. The time frame of the assessment can be adjusted to cover relatively brief periods (eg, 1 month) that capture patients' characteristic experiences yet are also amenable to assessing change during the course of relatively brief treatment interventions. In addition, as detailed in this article, considerable evidence documents the good psychometric properties and clinical relevance of this subscale.

However, some conceptual and psychometric features of this subscale may limit its ability to validly assess the anhedonia construct. The chief limitation is that an individual item may actually reflect several conceptually distinct processes or domains. Thus, an item rating may reflect either frequency of engagement or pleasure de-

rived from or interest in various types of activities. However, it is not clear that anhedonia, interest, and asociality should be considered in a unitary rating, as these constructs do not necessarily measure the same thing. As a consequence, patients may receive high ratings on the items that comprise this subscale for reasons that have little or no relationship to anhedonia.

For example, limited engagement and/or interest in recreational, sexual, or social activities will almost inevitably result in increased ratings on the Anhedonia-Asociality subscale items. While limited interest and engagement in such activities are indeed possible consequences of anhedonia, they may also result from a variety of emotional, motivational, and social factors other than a decreased capacity to experience pleasure, and several of the anchors for ratings on this subscale appear to reflect these other potential causes. The anchors for the Ability to Feel Intimacy and Closeness item indicate that the highest rating may be given if the subject prefers no contact with or is hostile toward family or significant others. Similarly, the anchor for the highest score for the Relationships with Friends and Peers item indicates that the subject has no friends and is not interested in developing any social ties. However, limited engagement with friends, romantic partners, and family could reflect such factors as limited financial resources, social anxiety, paranoia, or intentional avoidance of stressful or uncomfortable social situations, rather than an inability to experience pleasure from interpersonal sources. In addition, basic emotion research suggests that interest and pleasure reflect relatively distinct psychological processes.¹² Thus, by conflating assessment of anhedonia with actual performance of and interest in recreational and social activities, SANS Anhedonia-Asociality ratings may often reflect a social "performance" deficit more than a fundamental hedonic "capacity" deficit.

While other commonly used interview-based measures of negative symptoms include items that may be related to anhedonia, they also do not specifically assess reductions in the capacity to experience pleasant emotions. For example, the SDS⁹ includes items that assess primary, enduring deficits in the areas of Diminished Emotional Range and Diminished Social Drive. These items are assessed by directly asking patients about their emotional experiences in the context of social and other types of activities throughout the preceding 12 months. The Diminished Emotional Range item probes for reductions in the experience of pleasant emotions, but it also assesses the presence of a reduction of unpleasant emotions. While some patients may experience pervasive reductions in the experience of both pleasant and unpleasant emotions, this item appears to tap an emotional disturbance that is broader than the anhedonia construct, as reductions in the experience of pleasure do not necessarily involve accompanying decreases in unpleasant emotions (eg,¹³⁻¹⁵; discussed further below). Regarding Diminished Social

Drive, while an elevated score on this item certainly could reflect the patient's incapacity to experience pleasure from social contacts, low levels of interest or efforts to engage in social situations could also result from a variety of other factors. The SDS attempts to distinguish diminished social drive from a lack of social success, which might result from such secondary causes as social skill deficits, anxious withdrawal, or a lack of resources to engage in social activities. However, a primary lack of desire to engage in social activities could result from such factors as prolonged exposure to an unstimulating social environment or from having a limited social environment, rather than reflecting a fundamental inability to experience pleasure in social settings.

Similar issues apply to other available interview-based negative symptom rating scales that focus on more circumscribed time intervals. For example, the PANSS¹⁰ includes items that assess Emotional Withdrawal, which is described as decreased interest in, involvement with, and affective commitment to life events, and Passive/Apathetic Social Withdrawal, which assesses diminished interest and initiative in social interactions due to passivity, apathy, anergia, or avolition. Ratings typically cover 1 week to several weeks. According to the PANSS manual, these items are rated on the basis of reports provided by treatment providers or others (eg, family members) who know the patient, as well as on interpersonal behaviors observed during the course of the interview. While ratings on these items are based on the assumption that reduced frequency of engagement in various activities and certain types of observable behaviors are reflective of some underlying emotional disturbance, it is not clear that these behaviors reflect an incapacity to experience pleasure. A lack of social skills required for achieving satisfying relationships or effectively interacting with a clinical interviewer, overwhelming positive symptoms, social anxiety, or frequent exposure to hostile environments could also lead to elevated ratings on these scale items.

Likewise, the SEB¹¹ focuses on rating behaviors that are observed during a standard clinical interview and includes 3 items that measure indifference ("lack of affection for family," "unconcern for own present situation," "unconcern for own future"). Again, these items assess behaviors that are potential consequences of anhedonia, but they do not necessarily reflect incapacity to experience pleasant emotions. While a focus on observable behaviors may enable raters to more easily establish reliable negative symptom assessments, a valid interview-based assessment of anhedonia requires careful and direct questioning of patients about their emotional experiences.

Interview-based measures of negative symptoms vary considerably in their conceptualizations and assessments of anhedonia and disturbances in emotional experience. Among the currently available instruments, the SANS Anhedonia-Asociality subscale provides the most direct

and specific assessment of anhedonia. The remainder of this section focuses on the Anhedonia-Asociality subscale and reviews its psychometric properties, relationships to other clinical symptoms, and relationships to other features of schizophrenia.

Psychometric Properties, Temporal Stability, and Prevalence

Although research groups throughout the world have been trained in the administration of the entire SANS and dozens of studies report achieving acceptable levels of interrater reliability, only a few published reports have systematically evaluated the psychometric properties of the Anhedonia-Asociality subscale. For the most part, these studies were composed of relatively small samples that sometimes included psychiatric patients with diagnoses other than schizophrenia. Early reports of interrater reliability for the Anhedonia-Asociality subscale were very good, with Andreasen⁷ reporting an average intraclass correlation coefficient (ICC) of .85 for the individual items (.80-.90) and .86 for the global rating. Andreasen and Flaum¹⁶ reported similarly high interrater reliabilities across 4 samples collected by other research groups (representing a variety of cultural settings) during the mid-1980s.^{17,18}

Subsequent studies indicate somewhat lower and more variable levels of interrater reliability. Findings from a large, multisite trial indicated an average ICC of .56 (range .26-.85) across items and .70 for global ratings, based only on information gathered during clinical interviews that were conducted by trained raters and that covered the previous week.¹⁹ Norman et al.²⁰ reported a similar mean ICC of .61 (range .49-.65) across items with an ICC of .65 for global ratings in a study that used pairs of experienced raters who had access to the same supplemental materials (case notes, consultation with treating clinicians) for subsets of a total of 85 schizophrenia patients. However, somewhat higher levels of interrater reliability were reported in a more recent study of 30 inpatients with various psychotic disorders²¹ with a mean ICC of .85 (range .77-.92) based on a clinical interview, reports from significant others, and nurses' observations of ward behavior. Thus, interrater reliability for the Anhedonia-Asociality scale is moderate to good, though more recent studies report somewhat lower and more variable reliabilities across the items that comprise this scale. It is worth noting that the level of detail provided about the procedures that were used to train raters varies across studies, raising questions about the comparability of rater training.

Available evidence also indicates adequate internal consistency and test-retest reliability for the Anhedonia-Asociality subscale. Andreasen⁷ reported relatively high item-total subscale correlations, ranging from .49 to .75, with a mean correlation of .60. Alpha coefficients

ranging from .63⁷ to .77¹⁹ have been reported. As with most negative symptoms, SANS Anhedonia-Asociality appears to be relatively stable across various time intervals. For example, Mueser *et al.*¹⁹ reported a correlation of about .40 across a 6-month period from an initial acutely symptomatic state to a relatively remitted state among chronically ill patients. As compared to positive symptoms, several other groups have reported relatively stable mean levels of anhedonia ratings in both recent-onset and chronically ill patients.^{22–26} Thus, the limited available evidence indicates that the scale demonstrates moderate to good internal consistency and temporal stability.

Anhedonia appears to be a relatively common feature of schizophrenia, with similar or higher mean scores often reported on the Anhedonia-Asociality subscale as compared to other SANS subscales.^{26–28} For example, Fenton and McGlashan²⁹ found in a sample of 187 schizophrenia patients that 76 percent showed at least mild anhedonia, and 23 percent showed marked or severe anhedonia. Similarly, in 2 separate samples Andreasen and Flaum¹⁶ reported that about 80 percent showed at least moderate levels, with about 60 percent showing marked or severe anhedonia-asociality. Anhedonia is also prominent during the early course of the disorder,^{24,25,30} suggesting that this emotional disturbance is not merely secondary to chronicity or prolonged exposure to antipsychotic medications. In addition, several studies report that anhedonia-asociality is particularly severe among patients with enduring deficit symptoms.^{23,31,32}

Relationships With Other Symptoms

Three questions about the relationship between anhedonia and other symptoms of schizophrenia have been evaluated. First, is anhedonia related to other symptoms that are typically included in the negative symptom construct? Second, is anhedonia distinguishable from other symptom domains, including positive, disorganized, and mood symptoms? Third, within the domain of negative symptoms, is anhedonia distinguishable from other negative symptoms? In each case, the answer appears to be “yes,” but with some qualifications for the answer to the third question.

Regarding the first question of relationships with other negative symptoms, interview-rated Anhedonia-Asociality does indeed appear to be highly correlated with other symptoms that are typically conceptualized as components of the negative symptom construct. As detailed by Blanchard and Cohen (in this theme issue), factor analytic studies of the SANS alone or in conjunction with measures of positive symptoms such as the Scale for the Assessment of Positive Symptoms (SAPS)³³ indicate that Anhedonia-Asociality consistently and robustly correlates with other negative symptoms. These factor

analytic studies have also addressed the second question and indicate that anhedonia and other negative symptoms form a cohesive factor that is relatively independent from factors reflecting positive symptoms (ie, hallucinations and delusions) and disorganization.

Along these lines, there has also been considerable interest in whether Anhedonia-Asociality (and other negative symptoms) rated in schizophrenia patients are merely a secondary consequence of depression, which is common in schizophrenia and can also involve anhedonia.³⁴ Cross-sectional studies provide a mixed picture, with some reporting significant, though generally moderate, relationships between SANS Anhedonia-Asociality ratings and various indices of interviewer-rated or self-reported symptoms of depression^{35–39} and others failing to find significant correlations.^{26,28,40,41} It has been suggested that SANS Anhedonia-Asociality may be associated with only specific aspects of depression, including retardation, slowness, and lack of energy,^{37,42–44} although anhedonia is not consistently associated with behavioral measures of psychomotor retardation.⁴⁰ While anhedonia and depression may show some overlap and can be difficult to differentiate clinically (particularly in acutely symptomatic patients), several factor analytic studies indicate that anhedonia and depression form separate factors in schizophrenia patients,^{26,35,37,41} suggesting that Anhedonia-Asociality is not wholly redundant with symptoms of major depression in schizophrenia. Thus, findings concerning the first 2 questions raised above indicate that anhedonia does appear to be strongly related to the broader construct of negative symptoms and is distinguishable from psychotic, disorganized, and mood disorder symptoms.

Regarding the third question, the SANS Anhedonia-Asociality subscale appears to be distinguishable from at least some of the other negative symptoms. Factor analytic studies of the SANS (that do not also include the SAPS) rather consistently demonstrate that the Anhedonia-Asociality subscale loads on a separate factor from the Affective Flattening and Alogia subscales of the SANS.⁴⁵ These findings suggest that self-reports of social behavior and subjective experiences measured by the Anhedonia-Asociality subscale are separable from directly observable aspects of emotional expression and speech production rated during the interview.

However, these factor analytic studies have typically also found that the Anhedonia-Asociality subscale coheres with items from the SANS Avolition-Apathy subscale to form a latent factor that has been termed “social amotivation” or “diminished motivation.”^{36,46,47} The Avolition-Apathy subscale was designed to capture a subjective lack of energy, drive, and interest in the absence of sadness or depression.⁷ Similar to the Anhedonia-Asociality subscale, the Avolition-Apathy subscale ratings incorporate information that ranges from frequency of engagement in certain types of activities

(grooming and hygiene, work and/or school functioning) to subjective emotional and physical experiences. It is unclear if the apparent overlap between these subscales reflects the presence of a single underlying deficit. Instead, the high level of correlation may reflect the content overlap of the 2 subscales. For example, consideration of level of interest and productive community functioning is a feature of both subscales. More fine-grained psychometric assessments may be necessary to meaningfully differentiate between actual social behaviors and the various aspects of emotional experience that are tapped by these scales.

An important area for continued research is to determine whether conceptual and psychometric distinctions can be made among constructs such as hedonic capacity, hedonic experience, social affiliation, apathy, and anergia. This may be most productively accomplished through studies employing a classic construct validation approach.⁴⁸ Such studies would ideally evaluate the convergent and discriminant validity of measures of these putative constructs across multiple levels of analysis, including interviewer ratings, self-reports, performance-based laboratory measures, and measures of physiological and neural functioning. In addition, distinguishing among these constructs and identifying their underlying mechanisms would be a fruitful area for translational research. For example, theoretical and methodological advances from basic research in affective science⁴⁹ and social neuroscience⁵⁰ hold considerable promise for clarifying the nature of emotional, motivational, and social disturbances in schizophrenia.

Association With Functional Outcome and Neurocognition

The clinical significance of Anhedonia-Asociality is evident in its cross-sectional relationships with various measures of community functioning in the interpersonal, family, and recreational domains.^{26,47,51} Furthermore, higher levels of Anhedonia-Asociality are related to worse premorbid adjustment^{26,41,47,52} and lower levels of social competence^{52,53} and predict poor long-term outcome.^{23,29,47} Since SANS Anhedonia-Asociality ratings directly incorporate information about the frequency and quality of engagement in recreational and social activities, it is somewhat difficult to determine the extent to which these significant relationships with social functioning reflect overlapping item content rather than a direct link with the capacity to experience pleasure.

There has also been some interest in the question of whether ratings of Anhedonia-Asociality are separable from the pervasive neurocognitive deficits of schizophrenia. This issue emerges as an important topic because neurocognitive deficits are also strongly linked to poor social and community functioning.⁵⁴ Available evidence suggests that interview-rated Anhedonia-Asociality is not

significantly related to indices of general intellectual ability or generalized cognitive impairments. There is some evidence of moderate relationships between anhedonia and measures of executive or frontally mediated cognitive functions,^{55,56} as well as frontal functional^{57,58} and structural abnormalities.^{59,60} No consistent patterns have emerged for other specific aspects of neurocognition. Thus, SANS Anhedonia-Asociality ratings appear to be largely separable from generalized neurocognitive impairments.

Summary

Anhedonia can be reliably assessed using an interview-based format, with the SANS Anhedonia-Asociality subscale providing the currently best available assessment instrument. Anhedonia-Asociality ratings are strongly related to other negative symptoms and are distinguishable from psychotic, disorganized, and mood symptoms, as well as from neurocognitive deficits. It is noteworthy that although SANS Anhedonia-Asociality ratings tend to be relatively stable, several studies have reported that these ratings demonstrate sensitivity to treatment effects,^{22,61-63} suggesting that an interview format may be useful for detecting relatively short-term changes in anhedonia.

Although considerable evidence supports the utility of interview-based assessment of anhedonia, several additional factors should be considered. First, interview-based measures have several inherent limitations, including susceptibility to interviewer bias or drift, potential changes in the patient's ability to articulate his or her emotions with a change in medication status, differences in interviewer skill in establishing rapport and eliciting information about a patient's emotional experiences, differences in patients' abilities to recall and reflect on the experiences asked about, and interpersonal factors such as social skill deficits that may interfere with a patient's ability to communicate or changes in the patient-rater relationship over time. Second, since the studies reviewed in this section were based exclusively on the SANS Anhedonia-Asociality subscale, findings concerning the temporal stability, characteristics, and clinical correlates of interview-based assessment should be interpreted cautiously. Because only one measure of anhedonia was considered, it is difficult to determine the extent to which any of the reported findings reflect idiosyncrasies or imperfections of this particular subscale versus the true phenomenon of anhedonia that this subscale is meant to measure.

A third consideration involves the content overlap between the SANS Anhedonia-Asociality subscale and both the SANS Avolition-Apathy subscale and measures of actual community functioning. This overlap may reflect the fact that SANS Anhedonia-Asociality ratings are based not only on patients' capacity to experience

pleasant emotions but also on the frequency, quality, and level of interest and engagement in recreational and social activities. By incorporating information about actual functioning and level of interest into an assessment of anhedonia, measurement of the capacity to experience pleasure, as well as changes in pleasurable experience (and their independent contribution to social dysfunction), may become muddled or lost. A more refined and specific assessment of anhedonia would improve the validity of interview-based assessment of this construct and enhance the reliability of such assessments.

Self-Report Trait Measures

Instruments and Psychometric Properties

The second method for assessing anhedonia is through the use of self-report questionnaires. By far, the most frequently used questionnaires have been the Revised Social Anhedonia Scale (RSAS)⁶⁴ and the Physical Anhedonia Scale (PAS).⁶⁵ Based on the theoretical work of Rado and Meehl, the Chapmans and colleagues developed these true-false questionnaires in the late 1970s to measure stable individual differences in the capacity to experience pleasure from social-interpersonal and physical-sensual sources. The 40-item RSAS includes items sampling interpersonal sources of pleasure such as talking, socializing, and being with people in other ways (eg, “A car ride is much more enjoyable if someone is with me” [keyed False]; “I could be happy living all alone in a cabin in the woods or mountains” [keyed True]). The 61-item PAS includes items concerning the experience of pleasures related to taste, sight, touch, sex, and smell (eg, “The beauty of sunsets is greatly overrated” [keyed True]; “I like playing with and petting soft little kittens or puppies” [keyed False]). The scales are sometimes supplemented with a brief validity scale to ensure that participants are not responding in a highly idiosyncratic or random fashion.

The Chapman anhedonia scales have been used in dozens of studies of individuals with schizophrenia or at genetic or psychometric high risk for the development of schizophrenia spectrum disorders. In both patient and nonclinical samples, internal consistency reliability for both scales is consistently very good, with alpha coefficients typically exceeding .80.^{13,66,67} As discussed further below, the scales also demonstrate good test-retest reliability in schizophrenia patients.

Two other self-report measures of anhedonia have been used in a small number of studies of schizophrenia and have provided mixed results. The Fawcett-Clark Pleasure Scale, which was designed to assess anhedonia in depressed patients, failed to discriminate schizophrenia patients from healthy controls^{68,69} but was found to be higher in deficit syndrome patients than healthy controls.⁷⁰ The Snaith-Hamilton Pleasure Scale,⁷¹

also originally designed to assess pleasure in depressed patients, was found to be elevated in one study of schizophrenia patients as compared with norms for healthy controls.⁷² The current review focuses on studies of the considerably more widely used Chapman scales. While the content validity of the Chapman anhedonia scales may be somewhat outdated, they remain the standard anhedonia questionnaires in the field.

Comparisons With Healthy Controls and Diagnostic Specificity

Elevated scores on the RSAS and the PAS have repeatedly been shown to be a characteristic of individuals with schizophrenia during both the early and chronic stages of illness.^{13,66,73–81} The magnitude of these between-group differences is typically large, with patient samples tending to score about 1 standard deviation higher than healthy controls. In the context of elevated mean levels of anhedonia, a substantial range of individual differences in anhedonia is typically found among individuals with schizophrenia. For example, Chapman, Chapman, and Raulin⁸ found that one-third of their schizophrenia sample ($N = 121$) scored greater than 2 standard deviations above normal standards. More recently, Schürhoff *et al.*⁸² reported that about 41% of a sample of 80 schizophrenia patients reported marked anhedonia. Similar to findings for interview-based anhedonia, self-reported trait anhedonia appears to be particularly elevated among schizophrenia patients who meet criteria for the deficit syndrome.^{83–85}

Regarding the diagnostic specificity of self-reported trait anhedonia, schizophrenia patients have been found to report higher trait anhedonia as compared to euthymic or recently manic bipolar patients.^{74,82} While cross-sectional studies indicate that scores on the RSAS and PAS do not discriminate between schizophrenia patients and patients with depressive disorders with or without psychotic symptoms,^{73,79,86} the correlates of elevated scores on these anhedonia scales appear to differ across groups. Specifically, anhedonia appears to covary with clinical state in patients with depression but instead reflects an enduring trait in schizophrenia.

The notion that the correlates of anhedonia differ between patients with depression and schizophrenia is supported by cross-sectional findings that anhedonia correlates with severity of depressed mood but not with premorbid adjustment among depressed patients, whereas scores correlate with premorbid adjustment but not with depressed mood in schizophrenia patients.^{87,88} These differences are also supported by findings from longitudinal studies. In a 12-month longitudinal study, Blanchard, Horan, and Brown⁸⁹ found that acutely symptomatic groups of depressed and schizophrenia patients showed similarly elevated RSAS scores during an initial inpatient psychiatric hospitalization as compared with healthy controls. However, both depressed

mood and scores on the RSAS significantly decreased to normal levels by the outpatient follow-up point in the depressed group, whereas RSAS scores remained stably elevated in the schizophrenia patients throughout the follow-up period, despite a significant reduction in symptoms. Similar reports of stable elevations in schizophrenia patients as compared with healthy controls have been found in other studies,^{13,78} with remarkable stability found among schizophrenia patients for periods of up to 20 years.^{66,90} These findings converge to suggest that anhedonia reflects an enduring trait in schizophrenia but appears to be an episode indicator in depressed patients.

It is noteworthy that the anhedonia reported by schizophrenia patients does not necessarily reflect a pervasive diminution of both pleasant and unpleasant emotions. Schizophrenia patients also frequently report elevated levels of trait negative affectivity and neuroticism as compared with healthy controls.^{13,15,89,91,92} It is important to note that trait negative affectivity and neuroticism refer to a substantially broader emotional characteristic than the mood disturbances associated with clinical depression that are assessed by commonly used clinical rating scales. While individuals with elevated trait negative affectivity do often report experiencing elevated levels of sadness and guilt, they also frequently report experiencing a variety of other unpleasant emotions such as anxiety, frustration, fear, anger, and heightened reactivity to stress.⁹³ Interestingly, Rado⁵ hypothesized that schizophrenia patients' anhedonia could actually contribute to an increase in the experience of negative emotions, since hedonic experiences (or "welfare" emotions) may serve to buffer against the experience of unpleasant emotions. Likewise, Meehl^{6,94} speculated that such a process could lead anhedonic individuals to experience "aversive drift," or a progressive tendency to experience interactions with the external environment in negative ways, that might ultimately lead to a preference for decreased contact with the social environment. Since neither of these 2 theoretical propositions have been empirically tested, this remains an important direction for future research.

In light of the cognitive deficits of schizophrenia, one might question whether patients' self-reports of elevated trait anhedonia and trait negative affectivity accurately reflect their typical emotional experience in daily life. The validity of this pattern of self-reported traits is supported by naturalistic studies that use the experience sampling method, in which patients are cued to complete ratings of their emotions at random intervals throughout the day for a week or more. These studies indicate that schizophrenia patients also report experiencing lower levels of pleasant emotions and higher levels of unpleasant emotions than controls in the course of their daily lives.^{95,96}

Trait anhedonia demonstrates a similar pattern of relationships with positive symptoms, neurocognitive

deficits, and social functioning as found for interview-based assessments of Anhedonia-Asociality. Trait anhedonia is not significantly related to positive or disorganized symptoms^{74,87,89} and is typically not related to clinical ratings of depression.^{8,74,87,89} Trait anhedonia is also not significantly correlated with general intellectual ability or generalized neurocognitive deficits.^{8,74,76,80} However, trait anhedonia has shown moderate relationships with measures of executive functions both in schizophrenia patients and their unaffected biological relatives,^{80,97} as well as decreased frontal activation while performing cognitive tasks and certain psychophysiological abnormalities among patients.⁹⁸⁻¹⁰⁰

Higher levels of trait anhedonia are related to both poor premorbid and current functioning in the community^{8,13,86,101} and have also been found to correlate with social isolation in the offspring of individuals with schizophrenia.^{102,103} It is worth pointing out that scores on the PAS, which does not include items with overtly social content, have been found to correlate with social functioning, supporting a direct link between anhedonia and actual community functioning. Finally, the relationship between anhedonia and poor functional outcome appears to be remarkably stable across the course of the illness.⁹⁰

Association With Vulnerability

As noted above, Meehl⁶ originally proposed that anhedonia, particularly in the social domain, is an indicator of vulnerability to schizophrenia or schizotypy. Family studies and psychometric high-risk studies support Meehl's original theorizing.¹⁰⁴ Unaffected biological family members often show elevated levels of trait anhedonia.^{75,77,79,80,82,97,105,106} Individuals with markedly elevated trait anhedonia are characterized by higher ratings of DSM-IV schizoid, schizotypal, and paranoid personality disorder features than healthy controls.¹⁰⁷⁻¹⁰⁹ These individuals have also been found to show similar, though attenuated, impairments to those found in schizophrenia patients on a range of neurocognitive,¹¹⁰⁻¹¹³ psychophysiological,^{107,114} and electrophysiological^{115,116} tasks. In addition, social anhedonia has been found to predict the development of schizophrenia-spectrum disorders.^{117,118} Thus, family studies and psychometric high-risk studies suggest that anhedonia is a promising indicator of vulnerability to schizophrenia-spectrum disorders or psychotic disorders more generally.

Summary

The reliability and validity of self-reported trait anhedonia is supported by studies that have used the Chapmans' anhedonia scales over the past 25 years. Self-report questionnaires can be quite useful for assessing emotion in schizophrenia patients who may have difficulties articulating their subjective experiences, and they have the

advantage of not being affected by social skill deficits or other interpersonal factors that may influence the process of gathering information from patients within the context of a clinical interview.¹¹⁹ While the Chapman scales have been extremely useful in clarifying the significance of anhedonia in schizophrenia and in at-risk populations, their content is somewhat outdated, and more recent behavioral neuroscience research indicates that anhedonia consists of distinct components that are not reflected on these scales (discussed further below). Because these scales are relatively time-consuming to complete and are intended to measure enduring traits, trait anhedonia questionnaires may not be ideal for assessing changes in anhedonia during relatively brief clinical trials. However, the sensitivity of self-report anhedonia questionnaires has not yet been evaluated in the context of an effective clinical trial. Questionnaires that are specifically designed to assess short-term changes in emotional experience could be particularly useful for evaluating treatment-induced changes.

Laboratory-Based Assessment

Over the past decade, research on anhedonia and the experience of pleasant emotions has benefited from the theoretical and methodological advances that have occurred in basic emotion research. Contemporary models often conceptualize emotion as consisting of at least 3 components, including subjective experience, expression, and physiology.¹²⁰ A variety of emotion-induction paradigms have been developed to assess these components of emotional responding, which typically involve exposing participants to a standard set of evocative pleasant or unpleasant stimuli, such as emotional film clips, still photographs depicting emotional scenes, or different types of food. These paradigms have now been used in several studies of schizophrenia patients, and a few have supplemented assessments of emotional experience with simultaneous assessments of outward facial displays of emotion and/or aspects of physiological responding.

Assessment of emotional experience in these laboratory-based studies involves instructing subjects to rate pleasant or unpleasant emotion terms (eg, cheerful, happy, pleased) using Likert scales or bipolar scales that correspond to the valence and arousal dimensions of emotion. Among schizophrenia patients, consistently high levels of internal consistency have been found in these ratings of emotional experience, with alpha coefficients typically exceeding .80 in ratings of related emotion terms.^{67,74,121–123} These laboratory-based assessments of pleasant emotions have produced a fairly consistent pattern of results and have provided important insights about the boundaries of emotional disturbances in schizophrenia.

With few exceptions, laboratory-based assessments indicate that although patients express fewer outward displays of emotion, they self-report similar levels of pleasant

emotions as compared with healthy controls. These results have been replicated across a range of different types of stimuli, including emotional film clips,^{67,73,121–124} foods,^{67,68,73} simulated social interactions,¹²⁵ and briefly presented emotional pictures.^{81,126} but see 127,128 These relatively normal patterns of in-the-moment experience of pleasure have been demonstrated in both medicated and unmedicated patients^{122,123,129} and have been found to be stable in the same patients assessed across time and medication status.¹²¹ Patients' reports of unpleasant emotions have also been found to be similar, or in some cases elevated,^{121–123} as compared with healthy controls. These laboratory studies indicate that the expressive and experiential components of emotion do not appear to function in a coordinated manner in schizophrenia. However, they also suggest that patients are capable of experiencing a normal range and intensity of pleasant and unpleasant emotions when they are exposed to evocative stimuli.

Concerns have been raised about whether schizophrenia patients are capable of providing valid self-reports of emotional experience or whether, for example, patients are merely responding to demand characteristics associated with the evocative stimuli that are typically used in these laboratory-based studies. Converging evidence supports the validity of patients' self-reported emotions. For example, based on similarity ratings of pairs of emotional words, Kring, Feldman-Barrett, and Gard¹³⁰ found that schizophrenia patients and healthy controls both represent affect knowledge using a similar 2-dimensional (valence-arousal) structure. In addition, studies of emotional experience that simultaneously recorded physiological responding provide concurrent validation of patients' self-reported emotions. For example, Kring and Neale¹²² found that schizophrenia patients demonstrated greater skin conductance responding than nonpatients in response to emotionally evocative films, even though they displayed very few observable facial expressions.

Similarly, studies using facial electromyography indicate that despite diminished outwardly observable expressions of emotion, patients demonstrate valence-specific patterns of microexpressive responding to pictures of faces expressing different emotions.^{131–133} Schizophrenia patients also show the valence-specific linear pattern of emotion-modulated startle found in individuals, with startle potentiation in the context of unpleasant emotions and startle suppression in the context of pleasant emotions^{81,127} (but see⁸¹ for abnormal responsivity to unpleasant stimuli). These nonverbal, largely involuntary psychophysiological responses strongly suggest that patients with schizophrenia do experience emotional states, at least in terms of behaviors mediated by subcortical circuitry.

Summary

Laboratory-based assessments of pleasant emotions in schizophrenia have provided important information

about the boundaries of disturbances in the experience of emotion. Specifically, schizophrenia patients appear to be capable of experiencing a full range and intensity of pleasant emotions, despite diminished outward expressions of pleasant emotions and reports of diminished experiences of pleasure in the course of their daily lives. These normal reports of emotional experience indicate that laboratory assessments may not be useful as primary measures for assessing emotional changes during clinical trials, at least for the types of paradigms and evocative stimuli described above. However, these findings do have important implications for conceptualizing and investigating the precise nature of anhedonia in schizophrenia, which in turn will have implications for optimal assessment of the anhedonia construct.

Integration Across the 3 Methods of Assessing Anhedonia

While the 3 methods that have been used to assess anhedonia have each been found to demonstrate good psychometric properties and convergent validity, studies using these different methods have produced a somewhat paradoxical set of findings, raising questions about the precise nature of hedonic deficit in schizophrenia. On the one hand, individuals with schizophrenia typically report experiencing lower levels of pleasure in general than nonpatients on interview-based and self-report measures of trait social and physical anhedonia. The few studies that have directly examined the relationship between interview-based and self-reported trait anhedonia indicate moderate convergence between these methods.^{67,134} While one study failed to find a significant relationship between trait and interview-based anhedonia,⁷⁴ it is noteworthy that SANS ratings covered only a 1-week period in acutely ill patients and the limited range of experiences during hospitalization may have failed to accurately reflect patients' characteristic levels of functioning and experience.

On the other hand, individuals with schizophrenia have repeatedly reported experiencing levels of pleasant emotions that are similar to nonpatients in laboratory studies using emotionally evocative stimuli. Interview-based and self-report trait anhedonia demonstrate either nonsignificant or small associations with in-the-moment pleasant emotional responses to evocative stimuli.^{67,73,74} The overall pattern of findings suggests that individuals with schizophrenia are in fact capable of experiencing a normal range and intensity of pleasant emotions in response to evocative stimuli but for some reason report experiencing little pleasure more generally. Recent efforts to reconcile this discrepancy between trait and state assessments of pleasant emotions have focused on 2 possible explanations.

One possible explanation is that this trait-state discrepancy reflects the basic neurocognitive deficits of schizo-

phrenia. Self-report and interview-based measures of anhedonia require one to reflect on specific occurrences in one's life and provide aggregate ratings of the frequency and intensity of emotional experiences, which may be difficult in light of the declarative memory deficits that characterize schizophrenia.^{135,136} A recent study examined the hypothesis that anhedonia reflects faulty memory for subjectively experienced pleasant emotions, such that deficits in the encoding and/or retention of emotional information might lead patients to recall pleasurable experiences as less pleasurable than they actually were in the moment.⁶⁷ Patients first provided reports of their emotional experiences during exposure to a variety of pleasant and neutral foods and film clips and then completed a surprise recall task for their emotions after a 4-hour delay. Results indicated that patients did not significantly differ from controls in either their initial levels of reported pleasant emotional responses to the stimuli or in delayed recall for these experiences. Encoding and short-term retention for pleasurable experiences thus appear to be intact in schizophrenia, suggesting that trait anhedonia is not secondary to deficiencies in these memory processes. However, it remains possible that memory deficits for pleasant emotional experiences might be detectable at longer delay intervals.

An alternative explanation is that the pleasure deficit is circumscribed to a specific component of hedonic experience. Neurobehavioral models of hedonic experience distinguish between "appetitive pleasure," which refers to pleasure derived from anticipating that an activity will be enjoyable, and "consummatory pleasure," which refers to pleasure derived from engaging in an enjoyable activity.¹³⁷⁻¹³⁹ The validity of this distinction is supported by animal models,^{140,141} as well as by human neuroimaging studies,^{142, 143} which indicate that these aspects of hedonic experience rely on distinct neural circuits. Kring and colleagues^{144,145} have proposed that schizophrenia may be characterized by intact consummatory or "in the moment" pleasure when directly confronted with evocative stimuli, but impaired appetitive or anticipatory pleasure.

To evaluate this distinction in schizophrenia, a preliminary study administered the Temporal Experience of Pleasure Scale (TEPS),¹⁴⁶ a recently developed self-report trait measure that distinguishes between appetitive and consummatory pleasure, to a sample of stabilized schizophrenia outpatients ($n = 46$) and healthy controls ($n = 40$).¹⁴⁷ Schizophrenia patients reported lower appetitive pleasure, but similar consummatory pleasure, as compared with the controls. Furthermore, among patients, scores on the appetitive pleasure scale significantly correlated with clinical ratings of anhedonia and impaired community functioning, whereas no significant correlations were found for consummatory pleasure. These results were bolstered by an experience sampling study, which found that schizophrenia patients anticipated

experiencing less pleasure from future activities than controls during the course of daily life, providing additional support for the presence of an appetitive pleasure deficit in schizophrenia.

The distinction between appetitive and consummatory pleasure appears to have considerable explanatory value for reconciling the divergent findings across assessment methods. Further evaluation of whether schizophrenia is characterized by impaired appetitive or anticipatory pleasurable experiences is an important avenue for future research, particularly in light of the potential consequences for functional outcome. If patients are impaired in their ability to anticipate that potentially rewarding experiences will be enjoyable, they would be much less likely to seek out opportunities to engage in such activities, thereby contributing to the social withdrawal and lack of activity that many patients experience in their daily lives. Consistent with this possibility, an experience sampling study by Delespaul¹⁴⁸ found that schizophrenia patients reported “doing nothing” 5 times more frequently during the course of their daily lives. While these findings support the notion that anhedonia is linked to a failure to engage in pleasurable activities, it is difficult to determine whether these lower levels of engagement in activities that are typically considered to be pleasurable are a cause or a consequence of hedonic deficit.

Yet another explanation is that schizophrenia patients do have an impaired ability to experience pleasure that manifests only in certain contexts or in response to certain classes of stimuli. For example, according to Meehl,⁶ the pleasure deficit of schizophrenia is not a pan-deficit in emotional experience but is primarily social/interpersonal in nature. The nonaffiliative stimuli that have typically been used in laboratory studies of emotion in schizophrenia (eg, film clips, photographs, foods) may have limited generalizability to social situations that are encountered in daily life and potentially related to social anhedonia.⁷⁴ It is also worth noting that different types of pleasurable activities appear to be associated with different pleasurable feelings (eg, social activities appear to be differentially associated with cheerfulness¹⁴⁹), suggesting that more fine-grained analyses of different pleasant emotions across different activities may be informative. Along these lines, some studies have reported that schizophrenia patients show an impairment in the experience of pleasant odors,^{150,151} which may suggest that patients' experience of pleasure is impaired for certain types of stimuli.

Conclusions and Implications for Assessment

Empirical research using multiple assessment methods over the past 25 years indicates that anhedonia is a common, stable, and currently treatment-resistant feature of schizophrenia that is linked to the debilitating deficits in social functioning that so frequently characterize this disorder. Anhedonia is strongly related to other indicators

of the negative symptom construct, yet it also appears to be distinguishable from some negative symptoms, particularly those that reflect directly observable decreases in emotional expression and speech production. Thus, a comprehensive evaluation of negative symptoms should include an assessment of anhedonia.

The research reviewed in this article provides substantial evidence that emotional experience in patients with schizophrenia can be reliably and validly assessed through clinical interviews, self-report trait questionnaires, and laboratory-based assessments. Findings from these different assessment strategies have provided important insights into the precise nature of the hedonic deficit in schizophrenia and also have several implications for optimal assessment of anhedonia in clinical trials. First, since schizophrenia patients report generally normal levels of pleasant emotions in laboratory-based assessments of anhedonia and emotional experience, laboratory assessments using the types of evocative stimuli and paradigms employed to date would likely be less useful as primary measures of treatment-induced changes in anhedonia than other assessment strategies. However, no studies have employed these assessments in the context of an effective intervention. Thus, it remains unclear how patients' responses to emotionally evocative stimuli may or may not change following a successful intervention.

Furthermore, interview-based and self-report measures can be limited by the patient's ability to recall and relate particular experiences. Patients may report that they do not derive pleasure from pleasant events, such as movies, dinners out, or social interactions, because they have difficulty recalling these experiences, anticipating that they will be pleasurable, or organizing their thoughts in a manner that is required to answer questions about their emotional experiences. Yet, when presented with these opportunities, patients may well report experiencing pleasant emotions. Laboratory measures may serve as useful adjunct measures in the context of clinical trials using a pre-post assessment design, and they may help specify which aspect(s) of emotional experience are improved by novel treatments.

Second, widely used self-report trait anhedonia questionnaires may be impractical as primary assessment measures in clinical trials due to their length and their putative focus on enduring traits, which may limit their utility for demonstrating relatively short-term changes in anhedonia. However, these measures may be sensitive to changes in the context of an effective intervention, which the field has yet to identify. Nevertheless, the item content of these scales is somewhat outdated, which thus limits their content validity, and the scales do not assess the multiple components of hedonic experience, including appetitive and consummatory pleasure, which appear to be relevant to the pleasure deficit in schizophrenia. The recently developed TEPS¹⁴⁶ does distinguish between appetitive and consummatory pleasures, and

it is a promising tool for measuring anhedonia in both patients and at-risk samples. This scale was also designed to assess enduring individual differences, and thus it remains to be seen whether it will be sensitive to change in response to effective interventions.

The corpus of evidence that schizophrenia patients are capable of providing reliable and valid self-reports of their emotional experience does, however, suggest that self-report measures that are specifically designed to assess short-term changes in emotional experience could be of use in clinical trials. Further, redesigning the currently available measures, including the Chapman scales and TEPS, to focus on states rather than traits might also prove useful. This has been done successfully with other measures of emotion experience, such as the Positive and Negative Affect Schedule (PANAS).¹⁵²

The third conclusion from this review is that interview-based assessments appear currently to be the most well suited method for use in clinical trials. This is due primarily to their relative time-efficiency and demonstrated sensitivity to treatment-induced changes. While the 4 items that comprise the SANS Anhedonia-Asociality subscale have provided a useful tool for assessing anhedonia in an interview format, this subscale demonstrates considerable conceptual and content overlap with the SANS Avolition-Apathy subscale, as well as with measures of actual daily functioning. Limited interest and engagement in social and recreational activities are possible consequences of anhedonia, but they may also result from a variety of emotional, motivational, and social factors other than a decreased capacity to experience pleasure. Thus, interview-based assessment of anhedonia would benefit from a more refined and specific focus on patients' subjective experience of pleasant emotions, as differentiated from actual social functioning and from other subjective experiences such as decreased interest, energy, and will.

Future interview-based assessments of anhedonia would benefit from an expanded number of items, which would not only bolster psychometric properties but also allow for greater coverage of the anhedonia construct. For example, it would be useful to assess patients' emotional experiences across a wider range of pleasure-eliciting activities, including the various social activities and interactions, hobbies and pastimes, intellectual pursuits, aesthetic and religious experiences, and physical experiences, ranging from satiation of hunger to sexual gratification, in which pleasant emotions have been investigated.¹⁴⁹ While interview-based assessment of anhedonia should include careful probing of patients' emotional experiences during those activities in which they do engage, it can be quite challenging to assess pleasure capacity in patients who demonstrate only limited involvement in recreational or social activities. It would also appear to be useful to ask patients about how much pleasure they would anticipate experiencing if they were to engage in activities that are typically considered

enjoyable by others with similar sociodemographic characteristics. A more specific and differentiated assessment of anhedonia will be useful in both research aimed at identifying the underlying causes of anhedonia and evaluating the effectiveness of novel treatments for enhancing the experience of pleasant emotions in schizophrenia patients.

Several fundamental questions remain about the nature of anhedonia in schizophrenia. For example, interview-based and questionnaire assessments suggest that anhedonia may be particularly common among a subgroup of schizophrenia patients with enduring negative or deficit symptoms. It is not yet clear whether anhedonia is most appropriately conceptualized as a dimensional individual difference across individuals with schizophrenia or instead as an indicator of a specific subtype. Studies using appropriate statistical methods and research designs can help address this issue.¹⁵³ In addition, it is possible that the causes of anhedonia may differ across schizophrenia patients. For example, some patients may report anhedonia with accompanying dysphoric mood that is a reflection of a concurrent episode of depression or attributable to co-occurring suspiciousness. Other patients may experience anhedonia in the context of a reduction in the experience of both pleasant and unpleasant emotions. The utility of this distinction is an open question that could be addressed in longitudinal research that includes comprehensive symptom assessments across different clinical states.

In sum, interview-based measures are certainly the starting point for assessing anhedonia in the context of clinical trials. However, there is room for both laboratory and self-report measures as well. As is true for any assessment, a broad assessment will likely yield the most useful information from both a clinical and research standpoint.

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References

1. Kring AM, Germans MK, Anhedonia. In: Kazdin AE, ed. *Encyclopedia of Psychology*. New York: Oxford University Press; 2000:174–175.
2. Earnst KS, Kring AM. Construct validity of negative symptoms: an empirical and conceptual review. *Clin Psychol Rev*. 1997;17:167–190.
3. Kraepelin E. *Dementia Praecox and Paraphrenia*. Bradley, RM, trans. Huntington, NY: Robert E Krieger Publishing Co; [1917]; 1971.
4. Bleuler E. *Dementia Praecox or the Group of Schizophrenias*. Zinkin J, trans. New York: International Universities Press; [1911] 1950.

5. Rado S. *Psychoanalysis of Behavior: The Collected Papers of Sandor Rado*. Vol 2. New York: Grune and Stratton; 1962.
6. Meehl PE. Schizotaxia, schizotypy, schizophrenia. *Am Psychol*. 1962;17:827–838.
7. Andreasen NC. Negative symptoms in schizophrenia: definition and reliability. *Arch Gen Psychiatry*. 1982;39:784–788.
8. Chapman LJ, Chapman JP, Raulin ML. Scales for physical and social anhedonia. *J Abnorm Psychol*. 1976;85:374–382.
9. Kirkpatrick B, Buchanan RW, McKenney P, Alphas LD, Carpenter WR. The Schedule for the Deficit Syndrome: an instrument for research in schizophrenia. *Psychiatry Res*. 1989;30:119–123.
10. Kay S, Fiszbein A, Opler L. The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophr Bull*. 1987;13:261–276.
11. Abrams R, Taylor MA. A rating scale for emotional blunting. *Am J Psychiatry*. 1978;135:226–229.
12. Silvia PJ. What is interesting? exploring the appraisal structure of interest. *Emotion*. 2005;5:89–102.
13. Blanchard JJ, Mueser KT, Bellack AS. Anhedonia, positive and negative affect, and social functioning in schizophrenia. *Schizophr Bull*. 1998;24:413–424.
14. Blanchard JJ, Horan WP, Brown SA. Diagnostic differences in social anhedonia: a longitudinal study of schizophrenia and major depressive disorder. *J Abnorm Psychol*. 2001;110:363–371.
15. Horan WP, Blanchard JJ. Emotional responses to psychosocial stress in schizophrenia: the role of individual differences in affective traits and coping. *Schizophr Res*. 2003;60:271–283.
16. Andreasen NC, Flaum M. Schizophrenia: the characteristic symptoms. *Schizophr Bull*. 1982;17:27–49.
17. Lewine RRJ. A discriminant validity study of negative symptoms with a special focus on depression and antipsychotic medication. *Am J Psychiatry*. 1990;147:1463–1466.
18. Schulberg D, Quinlan DM, Morgenstern H, Glazer W. Positive and negative symptoms in chronic psychiatric outpatients: reliability, stability, and factor structure. *Psychol Assess*. 1990;2:262–268.
19. Mueser KT, Sayers SL, Schooler NR, Mance RM, Haas GL. A multisite investigation of the reliability of the scale for the assessment of negative symptoms. *Am J Psychiatry*. 1994;151:1453–1462.
20. Norman RMG, Malla AK, Cortese L, Diaz F. A study of the interrelationship between and comparative interrater reliability of the SAPS, SANS, and PANSS. *Schizophr Res*. 1996;19:73–85.
21. Peralta V, Cuesta MJ. Dimensional structure of psychotic symptoms: an item-level analysis of SAPS and SANS symptoms in psychotic disorders. *Schizophr Res*. 1999;38:13–26.
22. Addington J, Addington D. Positive and negative symptoms of schizophrenia: their course and relationship over time. *Schizophr Res*. 1991;5:51–59.
23. Mueser KT, Douglas MS, Bellack AS, Morrison RL. Assessment of enduring deficit and negative symptom subtypes in schizophrenia. *Schizophr Bull*. 1991;17:565–582.
24. Arndt S, Andreasen NC, Flaum M, Miller D. A longitudinal study of symptom dimensions in schizophrenia: prediction and patterns of change. *Arch Gen Psychiatry*. 1995;52:352–360.
25. Gur RE, Cowell P, Turetsky BI, et al. A follow-up magnetic resonance imaging study of schizophrenia: relationship of neuroanatomical changes to clinical and neurobehavioral measures. *Arch Gen Psychiatry*. 1998;55:145–152.
26. Rey ER, Bailer J, Bräuer W, Händel M. Stability trends and longitudinal correlations of negative and positive syndromes within a three-year follow-up of initially hospitalized schizophrenics. *Acta Psychiatr Scand*. 1994;90:405–412.
27. Andreasen NC, Arndt S, Alliger R, Miller D. Symptoms of schizophrenia: methods, meanings, and mechanisms. *Arch Gen Psychiatry*. 1995;52:341–351.
28. Gur RE, Mozley PD, Resnick SM, Levick S. Relations among clinical scales in schizophrenia. *Am J Psychiatry*. 1991;48:472–478.
29. Fenton WS, McGlashan TH. Natural history of schizophrenia subtypes: II. positive and negative symptoms and long-term course. *Arch Gen Psychiatry*. 1991;48:978–986.
30. Gelber EI, Kohler CG, Bilker WB, et al. Symptom and demographic profiles in first-episode schizophrenia. *Schizophr Res*. 2004;67:185–194.
31. Fenton WS, McGlashan TH. Antecedents, symptoms progression, and long-term outcome of the deficit syndrome in schizophrenia. *Am J Psychiatry*. 1994;151:351–356.
32. Möller H, Bottlender R, Groß A, et al. The Kraepelinian dichotomy: preliminary results of a 15-year follow-up study on functional psychoses: focus on negative symptoms. *Schizophr Res*. 2002;56:87–94.
33. Andreasen NC, Olsen SA. Negative v positive schizophrenia: definition and validation. *Arch Gen Psychiatry*. 1982;39:789–794.
34. Siris SG. Managing depression in schizophrenia. *Psychiatr Ann*. 2005;35:60–69.
35. Häfner J, Löffler W, Maurer K, Hambrecht M, an der Heiden W. Depression, negative symptoms, social stagnation and social decline in the early course of schizophrenia. *Acta Psychiatr Scand*. 1999;100:105–118.
36. Kelley ME, van Kammen DP, Allen DN. Empirical validation of primary negative symptoms: independence from effects of medication and psychosis. *Am J Psychiatry*. 1999;156:406–411.
37. Kitamura T, Suga R. Depressive and negative symptoms in major psychiatric disorders. *Compr Psychiatry*. 1991;32:88–94.
38. Sax KW, Strakowski SM, Keck PEJ, Upadhyaya VH. Relationships among negative, positive, and depressive symptoms in schizophrenia and psychotic depression. *Br J Psychiatry*. 1996;168:68–71.
39. Whiteford HA, Riney SJ, Csernansky JG. Distinguishing depressive and negative symptoms in chronic schizophrenia. *Psychopathology*. 1987;20:234–236.
40. Brébion G, Amador X, Smith M, Malaspina D, Sharif Z, Gorman JM. Depression, psychomotor retardation, negative symptoms, and memory in schizophrenia. *Neuropsychiatry Neuropsychol Behav Neurol*. 2000;13:177–183.
41. Kulhara P, Avasthi A. Influence of depressive symptoms and premorbid adjustment on factor structure of phenomenology of schizophrenia: a study from India. *Eur Psychiatry*. 2003;18:226–232.
42. Kulhara P, Avasthi A, Chadda R, Chandiramani K. Negative and depressive symptoms in schizophrenia. *Br J Psychiatry*. 1989;154:207–211.
43. Goldman RS, Tandon R, Liberzon I, Greden JF. Measurement of depression and negative symptoms in schizophrenia. *Psychopathology*. 1992;25:49–56.
44. Müller M, Szegedi A, Wetzel H, Benkert O. Depressive factors and their relationships with other symptom domains in schizophrenia, schizoaffective disorder, and psychotic depression. *Schizophr Bull*. 2001;27:19–28.

45. Blanchard JJ, Cohen AS. The structure of negative symptoms in schizophrenia: implications for assessment. *Schizophr Bull.* In press.
46. Keefe RS, Harvey PD, Lenzenweger MF, Davidson M. Empirical assessment of the factorial structure of clinical symptoms in schizophrenia: negative symptoms. *Psychiatry Res.* 1992;44:153–165.
47. Sayers SL, Curran PJ, Mueser KT. Factor structure and construct validity of the scale for the assessment of negative symptoms. *Psychol Assess.* 1996;8:269–280.
48. Cronbach LJ, Meehl PE. Construct validity in psychological tests. *Psychol Bull.* 1955;52:281–302.
49. Davidson RJ, Scherer KR, Goldsmith HH, eds. *Handbook of Affective Sciences.* London: Oxford University Press; 2003.
50. Ochsner KN. Current directions in social cognitive neuroscience. *Curr Opin Neurobiol.* 2004;14:254–258.
51. Ho B, Nopoulos P, Flaum M, Arndt S, Andreasen NC. Two-year outcome in first-episode schizophrenia: predictive value of symptoms for quality of life. *Am J Psychiatry.* 1998;155:1196–1201.
52. Mueser KT, Bellack AS, Morrison RL, Wixted JT. Social competence in schizophrenia: premorbid adjustment, social skill, and domains of functioning. *J Psychiatr Res.* 1990;24:51–63.
53. Bellack AS, Morrison RL, Wixted JT, Mueser KT. An analysis of social competence in schizophrenia. *Br J Psychiatry.* 1990;156:809–818.
54. Green MF, Kern RS, Braff DL, Mintz J. Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the “right stuff”? *Schizophr Bull.* 2000;26:119–136.
55. Hammer MA, Katsanis J, Iacono WG. The relationship between negative symptoms and neuropsychological performance. *Bio Psychiatry.* 1995;37:828–830.
56. Sanfilipo M, Lafargue T, Rusinek H, et al. Cognitive performance in schizophrenia: relationship to regional brain volumes and psychiatric symptoms. *Psychiatry Res: Neuroimaging.* 2002;116:1–23.
57. Eikmeier G, Lodemann E, Olbrich HM, Pach J. Altered fronto-central PINV topography and the primary negative syndrome in schizophrenia. *Schizophr Res.* 1993;8:251–256.
58. Zemishlany Z, Alexander GE, Prohovnik I, Goldman RG. Cortical blood flow and negative symptoms of schizophrenia. *Neuropsychobiology.* 1996;33:127–131.
59. Sanfilipo M, Lafargue T, Rusinek H, et al. Volumetric measure of the frontal and temporal lobe regions in schizophrenia: relationship to negative symptoms. *Arch Gen Psychiatry.* 2000;57:471–480.
60. Wolkin A, Choi SJ, Szilagyi S, Sanfilipo M, Rotrosen JP, Lim KO. Inferior frontal white matter anisotropy and negative symptoms of schizophrenia: a diffusion tensor imaging study. *Am J Psychiatry.* 2003;160:572–574.
61. Dollfus S, Petit M. Negative symptoms in schizophrenia: their evolution during an acute phase. *Schizophr Res.* 1995;17:187–194.
62. Eckert SL, Diamond PM, Miller AL, Velligan DI. A comparison of instrument sensitivity to negative symptom change. *Psychiatry Res.* 1996;63:67–75.
63. Silver H, Aharon N, Kaplan A. Add-on fluvoxamine improves primary negative symptoms: evidence for specificity from response analysis of individual symptoms. *Schizophr Bull.* 2003;29:541–546.
64. Eckblad ML, Chapman LJ, Chapman JP, Mishlove M. The Revised Social Anhedonia Scale. Unpublished test, University of Wisconsin, Madison; 1982.
65. Chapman LJ, Chapman JP. The Revised Physical Anhedonia Scale, Unpublished test. University of Wisconsin, Madison; 1978.
66. Herbener ES, Harrow M. The course of anhedonia during 10 years of schizophrenic illness. *J Abnorm Psychol.* 2002;111:237–248.
67. Horan WP, Green MF, Kring AM, Nuechterlein KH. Does anhedonia in schizophrenia reflect faulty memory for subjectively experienced emotions? *J Abnorm Psychol.* In press.
68. Berlin I, Givry-Steiner L, Lecrubier Y, Puech AJ. Measures of anhedonia and hedonic responses to sucrose in depressive and schizophrenic patients in comparison with healthy subjects. *Eur Psychiatry.* 1998;13:303–309.
69. Fawcett J, Clark DC, Scheftner WA, Gibbons RD. Assessing anhedonia in psychiatric patients: the pleasure scale. *Arch Gen Psychiatry.* 1983;40:79–84.
70. Loas G, Boyer P, Legrand A. Anhedonia in the deficit syndrome of schizophrenia. *Psychopathology.* 1999;32:207–219.
71. Snaith RP, Hamilton M, Morley S, Humayan A. A scale for the assessment of the hedonic tone: the Snaith-Hamilton pleasure scale. *Br J Psychiatry.* 1995;167:99–103.
72. Silver H, Shlomo N. Anhedonia and schizophrenia: how much is in the eye of the beholder? *Compr Psychiatry.* 2002;43:65–68.
73. Berenbaum H, Oltmanns TF. Emotional experience and expression in schizophrenia and depression. *J Abnorm Psychol.* 1992;101:37–44.
74. Blanchard JJ, Bellack AS, Mueser KT. Affective and social-behavioral correlates of physical and social anhedonia in schizophrenia. *J Abnorm Psychol.* 1994;103:719–729.
75. Clementz BA, Grove WM, Katsanis J, Iacono WG. Psychometric detection of schizotypy: perceptual aberration and physical anhedonia in relatives of schizophrenics. *J Abnorm Psychol.* 1991;100:607–612.
76. Franke P, Maier W, Hardt J, Hain C, Cornblatt BA. Attentional abilities and measures of schizotypy: their variation and covariation in schizophrenic patients, their siblings, and normal control subjects. *Psychiatry Res.* 1994;54:259–272.
77. Grove WM, Lebow BS, Clementz BA, Cerri A. Familial prevalence and coaggregation of schizotypy indicators: a multi-trait family study. *J Abnorm Psychol.* 1991;100:115–121.
78. Horan WP, Dawson ME, Schell AM, Ventura J, Subotnik KL, Nuechterlein KH. Longitudinal stability and psychophysiological correlates of physical anhedonia in recent-onset schizophrenia. Paper presented at: Annual Meeting of the Society for Research in Psychopathology; 2004; St. Louis, Mo.
79. Katsanis J, Iacono WG, Beiser M. Anhedonia and perceptual aberration in first-episode psychotic patients and their relatives. *J Abnorm Psychol.* 1990;99:202–206.
80. Laurent A, Biloa-Tang M, Bougerol T, et al. Executive/attentional performance and measures of schizotypy in patients with schizophrenia and in their nonpsychotic first-degree relatives. *Schizophr Res.* 2000;46:269–283.
81. Schlenker R, Cohen R, Hopmann G. Affective modulation of the startle reflex in schizophrenic patients. *Eur Arch Psychiatry Clin Neurosci.* 1995;245:309–318.
82. Schürhoff F, Szöke A, Bellivier F, et al. Anhedonia in schizophrenia: a distinct familial subtype? *Schizophr Res.* 2003;61:59–66.

83. Horan WP, Blanchard JJ. Neurocognitive, social, and emotional dysfunction in deficit syndrome schizophrenia. *Schizophr Res.* 2003;65:125–137.
84. Kirkpatrick B, Buchanan RW. Anhedonia and the deficit syndrome of schizophrenia. *Psychiatry Res.* 1990;31:25–30.
85. Loas G, Noisette C, Legrand A, Boyer P. Anhedonia, depression and the deficit syndrome of schizophrenia. *Acta Psychiatr Scand.* 1996;94:477–479.
86. Schuck JR, Leventhal D, Rothstein H, Irizarry V. Physical anhedonia and schizophrenia. *J Abnorm Psychol.* 1984;93:342–344.
87. Katsanis J, Iacono WG, Beiser M, Lacey L. Clinical correlates of anhedonia and perceptual aberration in first-episode patients with schizophrenia and affective disorder. *J Abnorm Psychol.* 1992;101:184–191.
88. Loas G, Salinas E, Guelfi JD, Samuel-Lajeunesse B. Physical anhedonia in major depressive disorder. *J Affective Disord.* 1992;25:139–146.
89. Blanchard JJ, Horan WP, Brown SA. Diagnostic differences in the temporal stability of social anhedonia: a longitudinal study of schizophrenia and major depressive disorder. *J Abnorm Psychol.* 2001;110:363–371.
90. Herbener ES, Harrow M, Hill SK. Change in the relationship between anhedonia and functional deficits over a 20-year period in individuals with schizophrenia. *Schizophr Res.* 2005;75:97–105.
91. Berenbaum H, Fujita F. Schizophrenia and personality: exploring the boundaries and connections between vulnerability and outcome. *J Abnorm Psychol.* 1994;103:148–158.
92. Horan WP, Subotnik KL, Reise S, Ventura J, Nuechterlein KH. Personality characteristics in recent-onset schizophrenia: longitudinal stability and clinical correlates. *Psychol Med.* 2005;35:995–1005.
93. Watson D, Clark LA. Negative affectivity: the disposition to experience aversive emotional states. *Psychol Bull.* 1984;96:465–490.
94. Meehl PE. Primary and secondary hypohedonia. *J Abnorm Psychol.* 2001;110:188–193.
95. Myin-Germeys I, Delepaul PA, deVries MW. Schizophrenia patients are more emotionally active than is assumed based on their behavior. *Schizophr Bull.* 2000;26:847–853.
96. Myin-Germeys I, van Os J, Schwartz JE, Stone AA, Delepaul PA. Emotional reactivity to daily life stress in psychosis. *Arch Gen Psychiatry.* 2001;58:1137–1144.
97. Franke P, Maier W, Hardt J, Hain C. Cognitive functioning and anhedonia in subjects at risk for schizophrenia. *Schizophr Res.* 1993;10:77–84.
98. Arnfred SM, Chen ACN. Exploration of somatosensory P50 gating in schizophrenia spectrum patients: reduced P50 amplitude correlates to social anhedonia. *Psychiatry Res.* 2004;125:147–159.
99. Gruzelier J, Davis S. Social and physical anhedonia in relation to cerebral laterality and electrodermal habituation in unmedicated psychotic patients. *Psychiatry Res.* 1995;56:163–172.
100. Van den Bosch RJ, Rozendaal N, Mol JM. Slow potential correlates of frontal function, psychosis, and negative symptoms. *Psychiatry Res.* 1988;23:201–208.
101. Horan WP, Green MF, Sergi M, et al. Motivation and social cognition in schizophrenia: differential associations with distinct aspects of community functioning. Paper presented at: International Congress of Schizophrenia Research; 2005; Savannah, Ga.
102. Erlenmeyer-Kimling L, Cornblatt BA, Rock D, Roberts S. The New York high-risk project: anhedonia, attentional deviance, and psychopathology. *Schizophr Bull.* 1993;19:141–153.
103. Freedman LR, Rock D, Roberts SA, Cornblatt BA, Erlenmeyer-Kimling L. The New York high-risk project: attention, anhedonia and social outcome. *Schizophr Res.* 1998;30:1–9.
104. Horan WP, Blanchard JJ, Gangestad SW, Kwapil TR. The psychometric detection of schizotypy: do putative schizotypy indicators identify the same latent class? *J Abnorm Psychol.* 2004;113:339–357.
105. Craver JC, Pogue-Geile MF. Familial liability to schizophrenia: a sibling study of negative symptoms. *Schizophr Bull.* 1999;25:827–839.
106. Kendler KS, Thacker L, Walsh D. Self-report measures of schizotypy as indices of familial vulnerability to schizophrenia. *Schizophr Bull.* 1996;22:511–520.
107. Gooding DC, Miller MD, Kwapil TR. Smooth pursuit eye tracking and visual fixation in psychosis-prone individuals. *Psychiatry Res.* 2000;93:41–54.
108. Horan WP, Brown S, Blanchard JJ. Social anhedonia, schizotypy, and candidate potentiators of clinical outcome. Submitted.
109. Kwapil TR, Crump RA, Pickup DR. Assessment of psychosis proneness in African-American college students. *J Clin Psychol.* 2002;58:1601–1614.
110. Gooding DC, Kwapil TR, Tallent KA. Wisconsin card sorting test deficits in schizotypic individuals. *Schizophr Res.* 1999;40:201–209.
111. Kerns JG, Berenbaum H. Aberrant semantic and affective processing in people at risk for psychosis. *J Abnorm Psychol.* 2000;109:728–732.
112. Luh KE, Gooding DC. Perceptual biases in psychosis-prone individuals. *J Abnorm Psychol.* 1999;108:283–289.
113. Tallent KA, Gooding DC. Working memory and Wisconsin card sorting test performance in schizotypic individuals: a replication and extension. *Psychiatry Res.* 1999;89:161–170.
114. Gooding DC. Antisaccade task performance in questionnaire-identified schizotypes. *Psychiatry Res.* 1999;35:157–166.
115. Simons RF. Physical anhedonia and future psychopathology: an electrocortical continuity? *Psychophysiology.* 1982;19:433–440.
116. Simons RF, Russo KR. Event-related potentials and continuous performance in subjects with physical anhedonia or perceptual aberrations. *J Psychophysiology.* 1987;1:401–410.
117. Gooding DC, Tallent KA, Matts CW. Clinical status of at-risk individuals 5 years later: further validation of the psychometric high-risk strategy. *J Abnorm Psychol.* 1995;114:170–175.
118. Kwapil TR. Social anhedonia as a predictor of the development of schizophrenia-spectrum disorders. *J Abnorm Psychol.* 1998;107:558–565.
119. Dworkin R. Affective deficits and social deficits in schizophrenia: what's what? *Schizophr Bull.* 1992;18:59–64.
120. Bradley MM, Lang P. Measuring emotion: behavior, feeling, physiology. In: Lane R, Nadel L, eds. *Cognitive Neuroscience of Emotion.* New York: Oxford University Press; 2000:242–276.
121. Kring AM, Earnst KS. Stability of emotional responding in schizophrenia. *Behav Ther.* 1999;30:373–388.
122. Kring AM, Neale JM. Do schizophrenic patients show a disjunctive relationship among expressive, experiential,

- and psychophysiological components of emotion? *J Abnorm Psychol.* 1996;105:249–257.
123. Kring AM, Kerr SL, Smith DA, Neale JM. Flat affect in schizophrenia does not reflect diminished subjective experience of emotion. *J Abnorm Psychol.* 1993;102:507–517.
 124. Salem JE, Kring AM. Flat affect and social skills in schizophrenia: evidence for their independence. *Psychiatry Res.* 1999;87:159–167.
 125. Aghevli M, Blanchard JJ, Horan WP. The expression and experience of emotion during social interactions in schizophrenia. *Psychiatry Res.* 2003;119:261–270.
 126. Volz M, Hamm AO, Kirsch P, Rey E. Temporal course of emotional startle modulation in schizophrenia patients. *Int J Psychophysiol.* 2003;49:123–137.
 127. Curtis CE, Lebow B, Lake DS, Katsanis J, Iacono WG. Acoustic startle reflex in schizophrenia patients and their first-degree relatives: evidence of normal emotional modulation. *Psychophysiology.* 1999;36:469–475.
 128. Paradiso S, Andreasen NC, Crespo-Facorro B, et al. Emotions in unmedicated patients with schizophrenia during evaluation with positron emission tomography. *Am J Psychiatry.* 2003;160:1775–1783.
 129. Kring AM, Germans MK. Subjective experience of emotion in schizophrenia. In: Jenkins JH, Barrett RJ, eds. *Culture, Subjectivity, and Schizophrenia.* New York: Russell Sage Foundation; 2003:329–348.
 130. Kring AM, Feldman-Barrett L, Gard D. On the broad applicability of the affective circumplex: representations of affective knowledge in schizophrenia. *Psychol Sci.* 2003;14:207–214.
 131. Earnst KS, Kring AM, Kadar MA, Salem JE, Shepard D, Loosen PT. Facial expression in schizophrenia. *Bio Psychiatry.* 1996;40:556–558.
 132. Kring AM, Kerr SL, Earnst KS. Schizophrenic patients show facial reactions to emotional facial expressions. *Psychophysiology.* 1999;36:186–192.
 133. Mattes RM, Schneider F, Heimann H, Birbaumer N. Reduced emotional response of schizophrenic patients in remission during social interaction. *Schizophr Res.* 1995;17:249–255.
 134. Lewine RRJ. Anhedonia and the amotivational state of schizophrenia. In: Andreasen NC, Tsuang MT, eds. *Negative versus Positive Schizophrenia.* Berlin: Springer-Verlag; 1991:79–85.
 135. Aleman A, Hijman R, de Haan EHF, Kahn RS. Memory impairment in schizophrenia: a meta-analysis. *Am J Psychiatry.* 1999;156:1358–1366.
 136. Heinrichs RW, Zakzanis KK. Neurocognitive deficit in schizophrenia: a quantitative review of the evidence. *Neuropsychol.* 1998;12:426–445.
 137. Depue RA, Iacono WG. Neurobehavioral aspects of affective disorders. *Ann Rev Psychol.* 1989;40:457–492.
 138. Gray JA. *The Psychology of Fear and Stress.* Cambridge, UK: Cambridge University Press; 1987.
 139. Klein D. Depression and anhedonia. In: Clark DC, Fawcett J, eds. *Anhedonia and affect deficit states.* New York: PMA Publishing; 1984.
 140. Berridge KC, Robinson TE. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Res Rev.* 1998;28:309–369.
 141. Berridge KC, Robinson TE. Parsing reward. *Trends Neurosci.* 2003;26:507–513.
 142. Knutson B, Fong GW, Adams CM, Varner JL, Hommer D. Dissociation of reward anticipation and outcome with event-related fMRI. *Neuroreport.* 2001;12:3683–3687.
 143. O'Doherty J, Kringelbach ML, Rolls ET, Hornak J, Andrews C. Abstract reward and punishment representations in the human orbitofrontal cortex. *Nat Neurosci.* 2001;4:95–102.
 144. Germans MK, Kring AM. Hedonic deficit in anhedonia: support for the role of approach motivation. *Pers Individ Dif.* 2000;28:659–672.
 145. Kring AM. Emotion in schizophrenia: old mystery, new understanding. *Psychol Sci.* 1999;8:160–163.
 146. Gard DE, Germans-Gard M, Kring AM, John OP. Anticipatory and consummatory components of the experience of pleasure: a scale development study. Submitted.
 147. Gard DE, Germans-Gard MK, Horan WP, Kring AM, John OP, Green MF. Anticipatory and consummatory pleasure in schizophrenia: a scale development. Paper presented at: Annual Meeting of the Society for Research in Psychopathology; . 2003; San Francisco, Calif.
 148. Delespaul AEG. *Assessing Schizophrenia in Daily Life.* Maastricht, The Netherlands: Universitaire Pers Maastricht; 1995.
 149. Berenbaum H. Varieties of joy-related pleasurable activities and feelings. *Cogn Emotion.* 2002;16:473–494.
 150. Crespo-Facorro B, Paradiso S, Andreasen NC, et al. Neural mechanisms of anhedonia in schizophrenia: a PET study of response to unpleasant and pleasant odors. *JAMA.* 2001;286:427–435.
 151. Moberg PJ, Arnold SE, Doty RL, et al. Impairment of odor hedonics in men with schizophrenia. *Am J Psychiatry.* 2003;160:1784–1789.
 152. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: The PANAS scales. *J Pers Soc Psychol.* 1988;54:1063–1070.
 153. Blanchard JJ, Horan WP, Collins LM. Examining the latent structure of negative symptoms: is there a distinct subtype of negative symptom schizophrenia? *Schizophr Res.* 2005;7:151–165.