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Affective Traits in Schizophrenia and Schizotypy

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This article reviews empirical studies of affective traits in individuals with schizophrenia spectrum disorders, population-based investigations of vulnerability to psychosis, and genetic and psychometric high-risk samples. The review focuses on studies that use self-report trait questionnaires to assess Negative Affectivity (NA) and Positive Affectivity (PA), which are conceptualized in contemporary models of personality as broad, temperamentally-based dispositions to experience corresponding emotional states. Individuals with schizophrenia report a pattern of stably elevated NA and low PA throughout the illness course. Among affected individuals, these traits are associated with variability in several clinically important features, including functional outcome, quality of life, and stress reactivity. Furthermore, evidence that elevated NA and low PA (particularly the facet of anhedonia) predict the development of psychosis and are detectable in high-risk samples suggests that these traits play a role in vulnerability to schizophrenia, though they are implicated in other forms of psychopathology as well. Results are discussed in terms of their implications for treatment, etiological models, and future research to advance the study of affective traits in schizophrenia and schizotypy.

Key words: schizophrenia/schizotypy/emotion/affective traits/review

Introduction

Emotional experience can be studied at 2 distinct levels of analysis. One level focuses on short-term, transient positive or negative emotional states (see Kring et al.).[1] The second focuses on affective traits, which represent stable individual differences in the tendency to experience corresponding emotional states. The current review of affective traits in schizophrenia focuses on this second level, which has its historical roots in personality psychology.

Disturbances in personality and emotional characteristics have figured prominently in clinical descriptions of people with schizophrenia and those believed to be at heightened vulnerability for this disorder throughout history. These descriptions encompass a remarkably diverse range of perspectives. For example, the character of affected and vulnerable individuals has been variously described as a diminished capacity to experience pleasure (anhedonia), a pervasive decrease in the experience of any type of emotion, or a heightened experience and sensitivity to negative emotional states.[2,3] In addition, some have proposed a basic continuity between pre- and post-onset personality, others suggest that personality influences the expression of particular symptoms and illness course, and yet others suggest that the onset of schizophrenia results in a dramatic alteration or even destruction of personality.[3–5] The purpose of this article is to review empirical studies of personality and emotion in schizophrenia in the context of contemporary models of affective traits.

Over the past 2 decades, emotion and personality researchers have converged on a consensual taxonomy of basic affective traits.[6,7] Although the affective trait framework has been usefully employed to investigate many forms of psychopathology,[6,7] its relevance to schizophrenia-related disorders has received relatively little attention. In this review, we first provide a brief overview of research on nonclinical samples indicating that 2 traits referred to in this article as Positive Affectivity (PA) and Negative Affectivity (NA) reflect basic emotional dispositions or temperaments (terminology used to describe affective traits is discussed in a later section). This provides the organizing framework for the second part of the article, in which we review studies of PA and NA in individuals with schizophrenia spectrum disorders, population-based studies of risk factors for psychosis, and high-risk samples. We conclude by discussing the implications of this body of evidence for treatment, etiological models, and further research into affective traits in schizophrenia.

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Affective Traits in Personality and Psychopathology Research

Throughout much of the 20th century, adult personality researchers generated a bewildering array of self-report measures and structural models of personality. Some models emphasized just a few broad traits. For example, Eysenck's pioneering model originally proposed a 2-factor model consisting of the broad traits of neuroticism (vs emotional stability) and extraversion (vs introversion).[8] Subsequent analyses led him to include a third broad dimension labeled psychoticism which, despite its name, is better viewed as a measure of disinhibition, an aspect of psychopathy.[9] At the other extreme, some personologists proposed over a dozen key traits.[10] These were largely phenotypic models that sought to develop comprehensive descriptive taxonomies, generally ignoring the etiology of the identified traits. For example, influential "Big Five" models initially developed out of attempts to understand the structure of natural language trait descriptors.[11,12] Extensive structural analyses consistently revealed 5 variably labeled factors: Neuroticism (vs Emotional Stability), Extraversion (or Surgency), Conscientiousness (or Dependability), Agreeableness (vs Antagonism), and Openness to Experience (or Imagination, Intellect, or Culture). Structural models emphasized the identification of traits that are largely independent of each other, persistent over time, and generalizable across situations.

In a separate research tradition, developmentalists established various models of temperament. Temperaments are defined as being at least partly attributable to innate biological factors, substantially stable over time, and having emotional processes as core features.[13] Early work by Thomas and Chess[14] on childhood development included 9 temperaments (eg, approach/withdrawal from new stimuli, predominant quality of mood, intensity of mood expression). A range of additional temperamental traits of children were proposed by others, such as behavioral inhibition, specific "primary" emotions, or sociability/affiliation.[15] Despite differences in labels, more recent research suggests that temperament structure can be represented by a small number of basic categories subsuming more focal "lower order" manifestations. For example, the 3 broad dimensions of NA, PA, and effortful control comprise Rothbart's model.[16] These temperamental dimensions are presumed to reflect innate, neurobiological tendencies that form the foundation for later personality development.

After decades of divisiveness and slow progress, adult personality and developmental researchers began to converge upon an integrative, consensual structure of personality and temperament in the 1980's and 1990's. A key element in achieving consensus was the recognition that the major personality traits represent manifestations of basic psychobiological dimensions of temperament. This recognition was influenced by 3 key developments.[7] First, an explosion of research demonstrated that most personality traits are substantially heritable.[17,18] Second, the major dimensions of personality were strongly and systematically associated with individual differences in affective experience, which is a defining feature of temperament.[19] Third, structural research finally began converging on a consensual phenotypic taxonomy of personality traits. This was greatly facilitated by the recognition that personality traits are ordered hierarchically, so that there is no fundamental incompatibility between models emphasizing a few higher order factors and those that include a much larger number of narrower traits that are seen as facets of these broader factors.[11,20,21] With the emergence of temperament-based personality models, traits provide plausible causal explanations of behavior rather than mere descriptions of it.

The "Big Two"

The recognition that personality traits represent basic psychobiological dimensions of temperament has led to the development of integrative, temperament-based hierarchical models of personality. According to Clark and Watson's model,[6,7] adult personality traits emerge through differentiation from 3 innate biobehavioral dimensions, 2 of which are affective systems—PA and NA—and the third of which ([dis]inhibition) is an affect and behavior regulatory system. This article focuses on the so-called Big Two affective dimensions of NA and PA. NA reflects individual differences in the extent to which a person views the world as threatening, problematic, and distressing. High scorers on the trait experience elevated levels of negative emotions and report a broad array of psychological and physical problems, whereas low scorers are calm, emotionally stable, and satisfied with themselves and their lives. PA involves an individual's willingness to engage the environment. High scorers on trait PA approach life actively, with energy, enthusiasm, cheerfulness, and confidence; as part of this general approach tendency, they seek out and enjoy the company of others. In contrast, those low on this trait tend to be reserved and socially aloof, reporting anhedonia and lower levels of energy and confidence. Finally, (dis)inhibition reflects individual differences in the tendency to behave in an undercontrolled vs overcontrolled manner.

Although there remains some controversy about what traits lie beyond PA and NA, the Big Two are strongly represented across virtually all major models of personality and temperament.[7,16] Table 1 provides examples of several influential models that, despite differences in the trait labels, demonstrate a high degree of convergence. Several lines of research indicate that the 3- and 5-Factor Model traits listed in the table provide converging evidence that PA and NA represent core temperamental dispositions. First, the various scales that measure broad PA-related constructs, as well as narrower aspects of

Table 1. Positive and Negative Affectivity Represented in Major Models of Personality

Model	Theorists	Negative Affectivity	Positive Affectivity	Other Traits	Questionnaires
Three-factor models	Clark and Watson	Positive temperament	Negative temperament	Disinhibition	GTS (L. A Clark, D. Watson, unpublished manuscript)
	Tellegen	Positive emotionality	Negative emotionality	Constraint	MPQ (A. Tellegen, unpublished manuscript)
	Eysenck	Neuroticism	Extraversion	Psychoticism	EPQ and EPQ—Revised[209]
Five-factor models	Costa and McCrae	Neuroticism	Extraversion	Openness to experience, agreeableness, conscientiousness	NEO-personality inventory—revised and NEO-FFI[210]
	Digman[11]	Neuroticism	Extraversion	Intellect, friendly compliance, will to achieve	
	Goldberg	Emotional stability (vs neuroticism)	Extraversion	Intellect/imagination, agreeableness, conscientiousness	Unipolar adjective markers[12]
	John	Neuroticism	Extraversion	Openness agreeableness, conscientiousness	Big Five Inventory[211]
Biosocial model	Cloninger	Harm avoidance	Novelty seeking (some facets), reward dependence (some facets)	Persistence, self-directedness, cooperativeness, transcendence	TPQ, TCI[29]

NA, Negative Affectivity; PA, Positive Affectivity; GTS, General Temperament Survey; MPQ, Multidimensional Personality Questionnaire; EPQ, Eysenck Personality Questionnaire; TPQ, Tridimensional Personality Questionnaire; TCI, Temperament and Character Inventory; NEO-FFI, NEO Five-Factor Inventory.

PA such as anhedonia, are highly intercorrelated, as are those that measure NA. In this connection, it is important to note that despite their opposite-sounding names, NA and PA are largely independent of each other.[7,22,23] Second, both traits have a substantial genetic component, with a median heritability estimate of approximately 0.50.[7] Along these lines, rapidly expanding research links these traits to corresponding approach/avoidance neurobiobehavioral systems.[24–26] Third, both traits have strong and systematic links to emotional experience. For example, neuroticism is broadly correlated with state and trait measures of negative emotionality, whereas extraversion is strongly associated with indices of positive emotions, indicating that emotion is a core feature of these traits.[19,23,27] Fourth, these traits show impressive long-term stability among individuals, even in early childhood, and their stability increases with age to at least age 50.[28] Finally, beyond their structural validity, these traits show important links to a range of real-world behaviors. For example, higher NA is associated with heightened stress reactivity and various physical health complaints, whereas PA is strongly correlated with various aspects of social engagement and activity, diurnal variation in mood, and sleep patterns.[7] As mentioned earlier, in this article, we will use the terms NA and PA to refer to the various corresponding trait labels from the 3- and 5-Factor Models listed in Table 1.

Table 1 also includes the model of Cloninger et al.,[29] which is widely used in clinical research, particularly outside the United States. This theoretical model originally proposed 3 "temperament" dimensions labeled Harm Avoidance, Novelty Seeking, and Reward Dependence. A fourth dimension of Persistence subsequently was broken out of reward dependency and included as a separate dimension. Four "character" dimensions not discussed in this article were added later. The original 3 dimensions have conceptual links to NA and PA: Harm Avoidance is the tendency to inhibit responses to signals of aversive stimuli that lead to avoidance of punishment and nonreward, Novelty Seeking is the tendency to respond actively to novel stimuli leading to pursuit of rewards and escape from punishment, and Reward Dependence is the tendency for a positive response to conditioned signals of reward that maintain behavior. Although these traits have theoretical relevance to NA and PA, the empirical links among them are not entirely clear. Harm Avoidance shows good convergence with other NA measures as well as state negative emotion, yet also correlates negatively with measures of PA (rather than being relatively independent), whereas Novelty Seeking and Reward Dependence are inconsistently related to measures of PA and state emotion.[30–32] Despite some uncertainty about how these scales map onto PA and NA, they have demonstrated relevance to a variety of clinical conditions.[6]

Relevance to Psychopathology Research

The emergence of temperament-based models of personality led to a rebirth of interest in relationships between personality and psychopathology, which continues to flourish.[6,33,34] For example, NA is broadly elevated across a diverse array of psychological disorders, including mood, anxiety, somatoform, eating, and personality disorders, leading some to propose that it is a general predictor of overall psychological functioning rather than a predictor of specific syndromes.[13] Although NA is a broad and general predictor of psychopathology, it is more strongly linked to syndromes that involve a substantial distress component than to other types of dysfunction. For example, among the mood and anxiety disorders, NA is most strongly related to disorders characterized by chronic, pervasive distress (major depressive and generalized anxiety disorders), moderately with syndromes characterized by more specific and limited forms of distress (eg, panic disorder and social phobia), and weakly related to those characterized primarily by behavioral avoidance (eg, specific phobias).[35,36] NA appears to share a genetic diathesis with so-called "distress disorders" (as compared with "fear disorders"), helping to explain the comorbidity among them.[37]

In contrast, PA shows relatively specific links to psychological disorders and symptom domains, particularly depressive disorders and anhedonia, respectively. For example, low PA appears to be a relatively specific feature of major depressive disorder and dysthymia that distinguishes it from most anxiety disorders.[35,36] However, low PA is clearly not unique to depressive disorders and anhedonia. For example, it is also associated with anxiety disorders in the social/interpersonal realm as reflected by correlations between PA and both social phobia and agoraphobia.[35,36,38] In addition to research relating the Big Two to Axis I disorders, there is growing evidence that abnormal and normal personality variation is best described within a single integrative hierarchy. PA and NA are consistently represented near the top of this hierarchical structure with Axis II disorders reflected in the extremes of these affective (and other) traits.[21,33] However, as mentioned earlier, despite great progress applying this affective trait framework to various forms of psychopathology, its relevance to schizophrenia spectrum disorders has received relatively limited attention.

How Can Studying Affective Traits Contribute to Schizophrenia Research?

Investigations of affective traits potentially can provide key information about the clinical presentation, etiology, and treatment of schizophrenia in several ways. First, schizophrenia is a diagnostic syndrome that encompasses a remarkably diverse set of signs and symptoms. As some have speculated,[3] individual differences in affective traits among schizophrenia patients may help to explain the expression of particular clinical symptoms and associated features such as functional outcome. Second, studying affective traits may help to explain a number of common comorbid conditions that occur in schizophrenia, such as mood disorders, anxiety disorders, and substance use disorders.[39–41]

Third, the study of affective traits in patients and in various high-risk populations can provide insights into etiological processes. Four basic models of possible relations between personality and psychopathology have been described[42,43]: (1) the *vulnerability model* proposes that maladaptive personality traits increase the likelihood that a person will eventually develop a disorder; (2) the *pathoplasty model* posits that once a disorder has developed, trait factors will interact with psychopathology to influence severity, course, or response to treatment; (3) the *complication/scar model* reverses the direction of causality, arguing that psychopathology influences personality either transiently or permanently; and (4) the *spectrum model* argues that normal and abnormal processes fall on the same underlying continua, such that individual differences in temperament essentially represent subclinical manifestations of psychopathology. Although these models are not mutually exclusive, they have different implications for research and intervention efforts. Fourth, information about affective traits may eventually inform treatment development and treatment planning.[44] For example, identification of disturbed affective traits in schizophrenia can draw upon the rapidly growing basic literature on their neurobiological and genetic correlates to identify intervention targets. Alternatively, clinicians can use information about affective traits to tailor treatments to the particular characteristics of people with schizophrenia, which is a key emphasis of recovery-focused treatment approaches.[45,46]

Affective Traits in Schizophrenia

The last major review of personality traits in schizophrenia was published in 1994 by Berenbaum and Fujita,[47] which covered 7 studies that compared schizophrenia patients and healthy controls on self-report questionnaires. Results indicated that schizophrenia patients had elevated NA (neuroticism) and low PA (extraversion), as well as elevations on a nonemotional trait they termed "peculiarity." The authors concluded that disturbances in these affective traits, although not specific to schizophrenia, may help explain clinical symptoms, course, and associated features. In the following sections, we review studies of schizophrenia patients that used questionnaires to assess traits related to PA and NA since the review by Berenbaum and Fujita. We subdivided the results of comparisons between schizophrenia and matched nonclinical control groups into 3 sections: (1) PA and NA assessed by questionnaires based on Three- and Five-Factor personality models, (2) The Social and Physical Anhedonia Scales,[48] which have been used extensively in schizophrenia and schizotypy

Table 2. Patients vs Control Comparisons on Three- and Five-Factor Personality Model Questionnaires

Study	Patients	Controls	Questionnaire	NA (Effect Size)	PA (Effect Size)
Blanchard et al.[57] (USA)	37 chronic outpatients (73% male)	15	MPQ	Patients > controls (1.34)	Patients < controls (−0.78)
Guerra et al.[63] (USA)	24 chronic outpatients (100% male)	46	NEO-FFI	Patients > controls (0.99)	Patients = controls (−0.46)
Blanchard et al.[54] (USA)	55 chronic inpatients (84% male)	41	GTS	Patients > controls (1.62)	Patients < controls (−0.73)
Akdag et al.[212] (USA)	18 chronic inpatient/ outpatients (100% male)	16	NEO-FFI	Patients > controls (1.16)	Patients = controls (−0.65)
Horan and Blanchard[65] (USA)	36 chronic outpatients (100% male)	15	GTS	Patients > controls (0.79)	Patients < controls (−0.71)
Lysaker et al.[58] (USA)	59 chronic outpatients (97% male)	17	NEO-FFI	Patients > controls (1.49)	Patients < controls (−0.70)
Cohen et al.[213] (USA)	73 chronic forensic inpatients (75% male)	22	MPQ	Patients > controls (0.76)	Patients < controls (−0.68)
Camisa et al.[109] (USA)	63 chronic outpatients (97% male)	55	NEO-FFI	Patients > controls (1.65)	Patients < controls (−0.98)
Guerra et al.[64] (USA)	30 chronic outpatients (80% male)	45	NEO-FFI	Patients > controls (1.03)	Patients < controls (−0.51)
Onitsuka et al.[214] (USA)	24 chronic outpatients (100% male)	26	NEO-FFI	Patients > controls (1.79)	Patients < controls (−0.88)
Herran et al.[62] (Spain)	60 chronic outpatients (53% male)	43	EPQ	Patients > controls (0.96)	Patients < controls (−0.50)
Beauchamp et al.[215] (Canada)	79 recent-onset psychosis (status not reported) (73% male)	66	NEO-FFI	Patients > controls (0.76)	Patients < controls (−1.27)
Couture et al.[74] (Canada)	96 first-episode psychosis (66% male)	66	NEO-FFI	Patients > controls (0.39)	Patients < controls (−0.94)

Effect size estimates were calculated using sample-size–weighted pooled within-group standard deviations whenever possible; otherwise, they were based on reported *F*- or *t*-statistics.

research, and (3) Harm Avoidance, Novelty Seeking, and Reward Dependency by questionnaires based on Cloninger's Biosocial model. For illustrative purposes, standardized effect sizes for patient vs control group comparisons were computed when the necessary descriptive or statistical information was available. Effect size estimates were calculated using sample-size–weighted pooled within-group standard deviations whenever possible; otherwise, they were based on reported *F*- or *t*-statistics. Many studies included combined samples of patients with schizophrenia and schizoaffective disorder; studies that compared affective traits among patients with these diagnoses found no significant differences between patient groups.[49–51] For each type of measure, we also review data relevant to diagnostic specificity, longitudinal stability, and correlations with clinical symptoms and associated features among individuals with schizophrenia.

Studies Based on 3- and 5-Factor Models

As shown in Table 2, and consistent with the review of Berenbaum and Fujita, individuals with schizophrenia consistently demonstrate a pattern of high NA and low PA in studies on these questionnaires (also see[52]). Most studies were conducted in the United States and include predominantly male subjects. Effect sizes are generally large for NA and medium to large for PA. This pattern is quite consistent across samples reflecting different symptomatic states (inpatients vs outpatients), as well as recent-onset and chronic stages of illness.

Only a few studies evaluated diagnostic specificity and longitudinal stability. Although schizophrenia patients were found to report lower PA than bipolar patients,[53] they do not differ cross-sectionally from depressed patients.[53,54] However, the pattern of high NA and low PA appears to remain stable across changes in symptom status and over time in schizophrenia,[54,55] whereas it substantially normalizes in depressed patients with symptom remission.[54] Relatively good temporal stability for scores on these measures has been demonstrated for up to 12 months in chronic patients[54,56,57] and 15 months in recent-onset patients.[55]

Despite speculation that these traits are linked to particular clinical symptoms, results do not consistently support this notion. Although some studies report significant relationships between NA and positive psychotic

Table 3. Patients vs Control Comparisons on the Social and Physical Anhdeonia Scales

Study	Patients	Controls	Questionnaire	Social Anhedonia (Effect Size)	Physical Anhedonia (Effect Size)
Chapman et al.[48] (USA)	121 chronic inpatients (100% male)	241	SAS, PAS	Patients > controls (0.72)	Patients > controls (0.48)
Katsanis et al.[81] (USA)	118 first-episode psychosis (status not specified) (69% male)	155	SAS, PAS	Patients > controls (0.95)	Patients > controls (1.03)
Grove et al.[121] (USA)	17 chronic inpatient and outpatient (76% male)	18	PAS		Patients > controls (1.03)
Berenbaum and Oltmanns[80] (USA)	43 chronic outpatients (51% male)	20	SAS, PAS	Patients > controls	Patients > controls
Franke et al.[91] (Germany)	19 chronic and first-episode inpatients (gender not reported)	35	PAS		Patients > controls (1.98)
Blanchard et al.[57] (USA)	37 chronic outpatients (73% male)	15	R-SAS, R-PAS	Patients > controls (1.05)	Patients > controls (0.85)
Craver and Pogue-Geile[125] (USA)	39 chronic outpatients (64% male)	38	R-SAS	Patients > controls (1.09)	
Laurent et al.[93] (France)	23 chronic outpatients (91% male)	34	R-SAS, R-PAS	Patients > controls (1.73)	Patients > controls (1.18)
Blanchard et al.[54] (USA)	55 chronic inpatients (84% male)	41	R-SAS	Patients > controls (0.89)	
Schuroff et al.[79] (France)	80 chronic inpatients (53% male)	94	R-PAS		Patients > controls (0.53)
Cohen et al.[213] (USA)	73 chronic forensic inpatients (75% male)	22	R-SAS, PAS	Patients > controls (0.62)	Patients > controls (0.76)
Camisa et al.[109] (USA)	63 chronic outpatients (97% male)	55	R-SAS	Patients > controls (1.76)	
Horan et al.[88] (USA)	30 chronic outpatients (83% male)	31	R-SAS, R-PAS	Patients > controls (1.10)	Patients > controls (1.29)
Herbener et al.[97] (USA)	33 chronic outpatients (58% male)	28	R-SAS, R-PAS	Patients > controls (0.92)	Patients > controls (0.76)
Horan et al. (USA)	72 recent-onset inpatients (80% male)	54	R-PAS		Patients > controls (0.67)

PAS, Physical Anhedonia Scale; SAS, Social Anhedonia Scale; R-PAS, Revised Physical Anhedonia Scale; R-SAS, Revised Social Anhedonia Scale.

symptoms,[51,58–60] NA or PA and affective symptoms,[55,56,58,60] or PA and negative symptoms,[56,61,62] these and other studies[62–65] just as often failed to confirm these specific relations. These traits also do not show consistent relationships with general intellectual functioning or more specific aspects of neurocognition among affected individuals,[51,64–66] though there is some evidence that affective traits are related to indices that reflect cerebral asymmetry, particularly right hemisphere dysfunction (eg,[67,68]). In light of the prominent role of laterality in basic affective science models,[69,70] this issue warrants additional research attention. Although interpretation is complicated by the use of different symptom and neurocognitive measures, NA and PA do not consistently show significant relationships with either specific symptoms or neurocognitive functioning in terms of the number of replicated significant results across studies. However, the sample sizes and other characteristics of these studies differed considerably and, as is the case for all relations described in this review, it remains possible that meaningful patterns could be detected in a quantitative meta-analysis based on pooled results.

There are, however, several replicated associations between affective traits and clinical features among individuals with schizophrenia. Higher NA correlates with worse functioning in several domains, including occupational

functioning[71,72] and quality of life.[62,71,73,74] Higher NA (particularly in combination with higher levels of disinhibition) is also associated with higher levels of substance use, including smoking[75] and alcohol and drug use.[40,76,77] It also correlates with higher levels of self-reported stress and maladaptive coping.[55,58,65,66] In contrast, higher PA is associated with several more adaptive outcomes, including larger social networks and higher quality of life.[57,71] Interestingly, one study found that higher PA correlates with worse functioning in the occupational domain.[72] This was interpreted to suggest that when patients with high PA experience occupational stress, they may tend to cope by seeking social support rather than active problem-solving strategies. Thus, NA and PA show replicable relationships with a variety of associated features among individuals with schizophrenia.

Studies Based on Anhedonia Questionnaires

Consistent with the findings of low PA in schizophrenia, studies using the Social and Physical Anhedonia Scales demonstrate a consistent pattern of elevated trait anhedonia. As shown in Table 3, individuals with schizophrenia uniformly report higher levels of both physical and social anhedonia than do nonpsychiatric controls, a pattern that is consistent across symptom states (inpatients vs outpatients) and during both early and chronic stages of illness. The magnitude of these between-group differences is consistently large.

Regarding diagnostic specificity, individuals with schizophrenia reported higher anhedonia than bipolar patients.[78,79] Although cross-sectional studies indicate that anhedonia levels do not discriminate between depressed and schizophrenia patients,[80–82] anhedonia covaries substantially with clinical state in depressed patients but reflects an enduring trait in schizophrenia.[54] This is the same pattern observed for PA, supporting the notion that anhedonia and PA fall on opposite ends of a shared dimension.[33] Anhedonia has shown relatively high stability in recent-onset patients for periods up to 15 months[83] and in chronic patients for periods of up to 20 years.[54,57,84,85]

Among patients, anhedonia is not significantly related to positive symptoms (eg,[54,78,86]), and is typically not related to depression (eg,[54,78,86]). Associations with negative symptoms are somewhat inconsistent,[78,87,88] possibly reflecting differences in patient status and assessment instruments across studies, or limitations of negative symptom measures.[89] Cross-sectional assessments of negative symptoms might be particularly vulnerable to inaccuracy in measuring this symptom construct. Clinical ratings that rely on the assessment of primary and enduring negative symptoms have found associations with physical and social anhedonia.[61,90] In addition, anhedonia is not significantly correlated with general intellectual ability or most specific neurocognitive deficits ([47,48,91,92], but see[93]), though a recent study reported that higher anhedonia correlated with worse performance on a social cognitive measure of emotion perception.[87] In addition, a few studies report that higher anhedonia correlates with less frontal activation while performing cognitive tasks and with certain psychophysiological abnormalities.[68,94,95] A growing number of studies have examined relations between anhedonia and emotional experience during laboratory paradigms involving evocative stimuli. Findings thus far are mixed, with some reporting significant associations[78,87,96] and others not.[88,92,97] However, higher anhedonia does consistently correlate with worse community functioning both premorbidly and currently,[48,82,87,98] an association that is remarkably stable across the course of illness.[85]

Studies Based on Cloninger's Biosocial Model

As displayed in Table 4, Cloninger's scales have been used in a variety of countries outside of the United States with mixed results. Harm Avoidance is consistently elevated in chronically ill inpatients and outpatients with schizophrenia, with generally large between-group differences. For Novelty Seeking, 6 out of 7 studies report no significant group differences with generally small effect sizes. Four out of 7 studies report lower Reward Dependence in individuals with schizophrenia (particularly outpatients), with variable effect sizes across studies. Thus, these studies provide strong and consistent report for elevated Harm Avoidance across a range of cultural contexts. As discussed further below, the less consistent findings for Novelty Seeking and Reward Dependence may be attributable to the specific content of these scales, which may not tap strongly into the positive emotional core of the PA construct.

The diagnostic specificity and longitudinal stability of these scales in relation to schizophrenia has not been examined. However, some studies listed in Table 4 and a few others have examined their correlates. Regarding clinical symptoms, no significant correlations were replicated across the relevant studies.[62,99–101] In addition, one study failed to find any relationships with neurocognitive functioning.[101] However, some replicable relationships with substance misuse/disinhibited behaviors and levels of functioning have been reported. Higher Novelty Seeking correlated with higher levels of alcohol and cannabis use,[102,103] as well as tobacco use[103] (but see[75]) and violent behavior.[104] Higher Harm Avoidance correlated with lower quality of life and community functioning,[49,50,62,105,106] whereas higher Reward Dependence correlated with better functioning in these areas.[62,105,106]

Summary of Affective Traits in Schizophrenia

Individuals with schizophrenia demonstrate a pattern of high NA across different questionnaires, symptom

Table 4. Patients vs Control Comparisons on the Cloninger's Biosocial Model Scales

Study	Patients	Controls	Measure	Harm Avoidance (Effect Size)	Novelty Seeking (Effect Size)	Reward Dependence (Effect size)
Guillem et al.[217] (Canada)	52 chronic outpatients (71% male)	25	TCI	Patients > controls (1.42)	Patients < controls (−0.94)	Patients = controls (−0.25)
Szoke et al.[218] (France)	45 chronic inpatients (58% male)	126	TPQ	Patients > controls (1.98)	Patients = controls (0.27)	Patients = controls (0.03)
Ritsner and Susser, 2004[100] (Israel)	90 chronic outpatients (86% male)	136	TPQ	Patients > controls (1.01)	Patients = controls (−0.10)	Patients < controls (−0.82)
Kurs et al.[105] (Israel)	47 chronic outpatients (81% male)	56	TPQ	Patients > controls (1.03)	Patients = controls (−0.29)	Patients < controls (−0.95)
Boeker et al.[101] (Germany)	22 chronic inpatients with first-rank symptoms (45% male)	22	TCI	Patients = controls (0.53)	Patients = controls (−0.07)	Patients = controls (0.15)
Herran et al.[62] (Spain)	59 chronic outpatients (53% male)	43	TPQ	Patients > controls (0.66)	Patients = controls (−0.22)	Patients < controls (−0.44)
Calvo et al.[131] (Argentina)	11 untreated chronic subjects (73% male)	12	TCI	Patients > controls (uncorrected for multiple comparisons)	Patients = controls	Patients < controls

statuses, stages of illness, and cultural contexts. They also show low PA throughout the illness course, although findings appear more dependent on the particular questionnaire used: results are strongest for specific measures of anhedonia, somewhat less strong for broader measures based on 3- and 5-Factor Models, and variable for conceptually related traits based on the Biosocial model. Among patients, although individual differences in affective traits do not show clear relationships to clinical symptoms or neurocognitive functioning, they are consistently related to functional outcome, quality of life, and stress reactivity. It should be noted that many studies had small samples and were strongly biased toward inclusion of male participants. It is also noteworthy that most comparisons using questionnaires based on 3- and 5-Factor Models and anhedonia were conducted in the United States, whereas those based on Cloninger's Biosocial model were all conducted in other countries, making potential differences across cultural contexts very difficult to evaluate.

Affective Traits in Schizophrenia-Spectrum Personality Disorders

Cluster A personality disorders, which include schizotypal, paranoid, and schizoid personality disorders, appear to share a common genetic diathesis with schizophrenia and are believed to reflect a spectrum of schizophrenia-related psychopathology.[107,108] In addition, individuals with schizotypal personality disorder, the most extensively studied of these disorders, evidence neuropsychological, psychophysiological, and neuroanatomical abnormalities similar to those observed in schizophrenia.[107] Thus, it can be informative to determine if traits found to be associated with schizophrenia are similarly related to schizophrenia-spectrum personality disorders. The study of these personality disorders also offers the possibility of examining traits in samples that eliminate or minimize confounds associated with medications, chronic institutionalization, and psychosis.

Only a few studies have examined affective traits in clinical samples of individuals diagnosed with these personality disorders. These studies consistently indicate that individuals with Cluster A personality disorders show the same pattern of high NA and low PA/anhedonia found in individuals with schizophrenia.[109–114] Similar results are found when moving beyond the use of normal trait measures (as employed in the 3- and 5-Factor Models), and employing measures that assess both normal range and pathological range personality.[115] Additional support for links between Cluster A personality disorder symptoms and affective traits comes from studies of heterogeneous clinical samples that include patients with a variety of personality disorders or associated symptoms, in which higher dimensional scores on Cluster A personality disorder symptoms correlate with higher NA and lower PA/anhedonia.[116–118] It should be noted, however, that the pattern of high NA and low PA is not unique to

patients with Cluster A disorders and is found across many personality disorders and depression.[112,113]

Affective Traits in Relevant Nonclinical Samples

From the studies reviewed thus far, it is unclear whether the pattern of high NA and low PA is solely a consequence of developing either schizophrenia or related spectrum personality disorders (ie, complication/scar model) or instead plays a more central role in vulnerability to these disorders. Research from several types of nonclinical samples helps shed light on this issue. These samples include (1) biological relatives of schizophrenia probands; (2) large-scale population-based prospective studies of risk factors for psychosis; and (3) non-clinical studies of psychometrically ascertained schizotypal individuals. Evidence from these populations increasingly supports the notion that these traits are indeed associated with vulnerability to schizophrenia.

Family Studies

Affective traits have been examined in several studies of schizophrenia patients' family members, who are at heightened genetic risk for the development of this disorder.[119] Most of the relevant studies used the Physical and Social Anhedonia scales. Of 8 family studies that assessed physical anhedonia, 5 reported significantly higher scores in relatives than controls,[81,91,120–122] 1 reported a borderline significant increase,[93] 1 reported elevated anhedonia only in relatives of probands who themselves had elevated anhedonia,[79] and one reported no differences between young children of schizophrenia probands and matched controls.[123] Of 4 studies that examined social anhedonia 2 reported higher scores in relatives than controls,[81,124] while 2 reported only nonsignificant trends toward higher scores in adult relatives.[93,125] Thus, elevated anhedonia, particularly in the physical realm, appears to be a replicable finding in the relatives of schizophrenia, though it does not distinguish the relatives of schizophrenia probands from those of probands with mood disorders.[79,81,123]

Fewer studies have examined questionnaires based on the other personality models, and the results of these studies are less consistent. Only 2 studies were identified that used questionnaires based on 3- or 5-Factor models that include independent control groups. Results are mixed, with one reporting higher NA and lower PA in male relatives[126] (also see[127]), while the other reported that relatives from multiply affected families did not differ from controls.[128] The relatives of schizophrenia probands do not differ from relatives of mood disorder probands on NA or PA.[128–130] Five studies were identified that used questionnaires based on the Biosocial model, with none reporting elevated Harm Avoidance or Novelty Seeking in relatives compared to controls and only one reporting lower RD in relatives.[105,122,131–133]

Overall, elevated anhedonia is the most consistently reported finding in family studies, though elevations on this trait are not found only in relatives of psychiatric probands with schizophrenia. Family studies using measures from other personality models show much less evidence of affective trait abnormalities. Inconsistencies across studies could reflect a number of methodological issues beyond differences in the constructs assessed by the measures themselves, such as differences in the types of relatives included (siblings, parents, children, second-degree relatives), sample sizes, inclusion criteria for relative and community comparison subjects (eg, screening for psychosis and other axis I disorders), and thresholds across studies used to correct (or not) for multiple comparisons. Sex effects could also influence findings, as some studies reported a more pathological pattern of affective traits in male relatives.[126,130] Despite inconsistencies in the pattern of relative vs control group differences, it is noteworthy that individual differences in affective traits among relatives have shown significant relationships with other variables. For example, higher NA shows associations with higher levels of Cluster A personality disorder symptoms or psychopathology,[132,134,135] and higher anhedonia shows associations with neurocognitive and social functioning.[130,136,137] Thus, individual differences in affective traits could help explain variance in several clinically relevant variables among genetically vulnerable family members.

Population-Based Studies

Large-scale, population-based studies from 3 countries support the notion that affective traits are risk factors for the development of schizophrenia or psychosis. First, Van Os and Jones[138] examined a national birth cohort of 5362 individuals born in Britain who completed measures of neuroticism and extraversion at age 16 and were assessed 9 subsequent times up to age 43. Neuroticism increased risk for schizophrenia and extraversion independently reduced it, even after adjustment for anxious and depressed mood/symptoms. Second, Krabbendam et al.[139] evaluated a population sample (mean age 41.5 years [SD = 11.8]) of 3929 individuals in the Netherlands with no lifetime history of psychosis on a measure of neuroticism at baseline, followed by assessments for psychosis 1 and 3 years later. Baseline neuroticism (and low self-esteem) significantly predicted first onset of psychosis at 3 years, even after controlling for symptoms of anxiety or depression. Finally, Goodwin et al.[140] evaluated a birth cohort of 1265 individuals in New Zealand assessed on neuroticism at age 14 and for psychiatric symptoms at ages 18 and 21. Neuroticism strongly predicted later psychotic symptoms, which persisted after controlling for co-morbid psychiatric disorders and various childhood adversity factors.

These studies suggest that neuroticism, and to a lesser extent extraversion, serves as risk and protective factors,

respectively, for the subsequent development of schizophrenia or psychosis. Importantly, these prospective studies convincingly show that disturbances in affective traits are not merely secondary consequences of psychosis. These traits are also risk/protective factors for the development of other forms of psychopathology, including depressive and anxiety disorders.[141,142] This has led some investigators to propose that these traits, particularly neuroticism, reflect a shared liability for both psychosis and depression.[143] However, given the range of disorders associated with NA, it is likely that NA represents a nonspecific vulnerability to a wide range of psychopathology.

In this context, it is noteworthy that a number of studies now indicate that the prevalence of psychotic symptoms in the general population is considerably higher than the prevalence of individuals who meet formal diagnostic criteria for psychotic disorders.[143] Higher levels of hallucination- or delusion proneness are associated with higher NA.[144–147] Because only a fraction of individuals who experience psychotic symptoms will ever meet criteria for a clinical disorder, it has been proposed that high NA increases the risk for decompensation, perhaps due to corresponding negative emotional appraisals that these individuals attach to these symptoms.[143,148]

Nonclinical Samples Using Psychometric Schizotypy Indicators

Finally, a number of studies have examined relationships between affective traits and psychometric measures of schizotypy or psychosis proneness in nonclinical student or community samples. Much of this research has been informed by the theorizing of P. E. Meehl who proposed that a genetically based neural defect—schizotaxia—manifests itself as a particular personality organization (schizotypy) that reflects vulnerability to develop schizophrenia.[149,150] The schizotypy construct is considerably broader than the diagnostic categories of schizophrenia or Cluster A personality disorders, and only a proportion of putative schizotypes are ultimately expected develop these clinical disorders. Research in this area has utilized either narrow personality measures that seek to assess specific traits from Meehl's model (eg, perceptual aberration, magical ideation, or anhedonia[151]) or have utilized broader diagnostically informed questionnaires that cover the breadth of current DSM criteria for schizotypal personality disorder.[107,152]

Two aspects of schizotypy have been extensively studied using the Chapmans' psychosis proneness scales: ''negative schizotypy'' traits, most commonly referring to pleasure deficits assessed by the Social and Physical Anhedonia Scales, and ''positive schizotypy'' traits, which include cognitive and sensory anomalies assessed by the Magical Ideation and Perceptual Aberration Scales.[151] Support for the validity of the negative schizotypy scales comes from studies indicating that individuals with markedly elevated social or physical anhedonia demonstrate neurocognitive, perceptual, and physiological abnormalities resembling those found in schizophrenia, including some evidence of right hemisphere dysfunction.[153–155] These individuals also report elevated levels of schizotypal, schizoid, and paranoid personality disorder symptoms[156–160] Finally, 2 prospective studies indicate that social anhedonia predicts the development of symptoms of schizophrenia spectrum disorders, whereas elevated positive schizotypy scores predict the development of symptoms of psychotic as well as other psychiatric disorders.[157,159]

A number of studies indicate that individual differences on these schizotypy measures are associated with affective traits. Studies of student and community samples consistently indicate that higher levels of positive and negative (anhedonia) schizotypy traits are both associated with higher NA.[158,161–166] Similarly, in nonclinical student and community samples, higher levels of interviewer-rated or self-reported symptoms of Cluster A personality disorders are associated with higher NA and lower PA (eg,[167–171]). Whereas high NA shows associations with both positive and negative schizotypy, low PA is more specifically associated with the negative features of schizotypy as measured by anhedonia scales.[162–166] Some researchers have suggested that NA may potentiate clinical outcomes among psychosis-prone individuals. For example, Horan et al.[158] found that among individuals with elevated social anhedonia, those with higher levels of NA also had higher levels of Cluster A personality disorder symptoms.

In summary, results from 3 types of nonclinical samples provide varying levels of support for the notion that high NA and low PA is characteristic not only of individuals diagnosed with schizophrenia and spectrum personality disorders but is also a risk factor for the development of psychosis and is detectable in those believed to possess heightened vulnerability to this disorder. As in schizophrenia, the evidence base is strongest for NA and, within PA, the narrow traits of social and physical anhedonia. Then again, however, the affective trait abnormalities found using these scales do not appear to be specifically associated with vulnerability to schizophrenia, as they are also associated with vulnerability to mood disorders. Relevant research based on Cloninger's Biosocial model has only been conducted in family studies, which provide minimal evidence of abnormalities on Harm Avoidance, Novelty Seeking, or Reward Dependency in the relatives of schizophrenia probands. Overall, these findings suggest that the high NA and anhedonia characteristic of individuals with schizophrenia cannot be solely explained by the complication/scar model. That is, these traits also appear to play a role in vulnerability to the development of this disorder.

Conclusions

Studies that have evaluated emotional experience in schizophrenia at the state-level generally indicate that affected individuals show a normal capacity to experience a full range of positive and negative emotions in response to evocative stimuli (see Kring et al.).[1] In contrast, the current review of emotional experience at the trait level indicates that schizophrenia is characterized by a relatively distinct pattern of affective trait disturbances, which also appears to contribute to vulnerability for the development of this disorder. The conclusions of this review are based on consistency of reported significant findings across studies rather than a quantitative meta-analysis, and must be interpreted in this context. From this review, we offer the following main conclusions:

1. At the group level, schizophrenia spectrum disorders are characterized by elevated NA across different measures and samples throughout the world. This appears to be relatively stable over time and clinical status.
2. At the group level, schizophrenia spectrum disorders also are characterized by stably low PA/elevated anhedonia, although results are more variable across particular trait measures.
3. Despite the above findings at the diagnostic level, there is sizable variability in these affective traits among individuals with schizophrenia spectrum disorders.
4. Among individuals with schizophrenia, NA and PA show minimal relations with specific clinical symptoms and neurocognitive functioning, but show meaningful relations to several key associated features, including level of functioning, quality of life, and stress reactivity.
5. The pattern of high NA and low PA/elevated anhedonia also appears to predict the development of psychosis and is detectable in nonclinical samples believed to possess heightened vulnerability to schizophrenia.

These findings raise a number of issues concerning the nature of disturbances in affective traits in schizophrenia and their implications for treatment, etiology, and continued research.

Scope and Treatment Implications of Affective Trait Disturbances in Schizophrenia

Since the review by Berenbaum and Fujita,[47] basic research on temperament-based models of affective traits and applied research on their relevance to schizophrenia and other forms of psychopathology have made considerable progress. As noted above, these authors predicted that the pattern of high NA and low PA, although not specific to schizophrenia, may help explain its clinical symptoms, course, and associated features. The current review is largely consistent with these predictions.

In stark contrast to some historical characterizations of individuals with schizophrenia as being essentially devoid of emotional experience, affected individuals clearly report frequent, intense experiences of negative emotions. Elevated NA has now been reported using a variety of questionnaires in over 25 empirical studies of patients in different clinical states, at early and late stages of illness, and in countries throughout the world. Thus, it is important for clinicians and others to appreciate that although many individuals with schizophrenia may express little emotion outwardly, the experience of negative emotions is a common characteristic of the emotional lives of people with this disorder.

Disturbances in PA appear more sensitive to differences in measures. Elevations in anhedonia are more strongly and consistently reported than are low scores on broad measures of PA derived from 3- and 5-Factor models. This may indicate that anhedonia represents a facet of PA that is particularly relevant for schizophrenia. It should be noted, however, that anhedonia is itself a multifaceted neurobiological construct. Recently, researchers have begun to investigate whether particular aspects of hedonic experience are impaired in schizophrenia. For example, studies of emotional states in schizophrenia indicate an intact capacity to experience pleasant emotions in response to evocative stimuli whereas studies of emotional traits indicate characteristically low levels of pleasure. To reconcile these findings, Kring et al. proposed that schizophrenia may be characterized by intact consummatory pleasure but impaired anticipatory pleasure. Using a new self-report trait measure to assess these distinctive aspects of hedonic experience,[172] preliminary findings support this hypothesis.[173] This line of research illustrates how considering emotion at both state and trait levels can provide important insights into emotional disturbances.

Among studies of PA-related questionnaires, findings were least consistent for the Reward Dependency and Novelty Seeking scales based on Cloninger's Biosocial model. As noted earlier, although these scales have conceptual links to PA, empirical studies have not uniformly demonstrated that they are strongly related to positive emotional core of PA. For example, Reward Dependence relates as or more strongly to the sociability aspects of extraversion than its PA component[30,174] and also is moderately strongly related to agreeableness,[175,176] whereas it typically shows only moderate to small correlations with alternative measures of higher and lower order PA scales (eg,[30,176], but see[174]). Novelty Seeking shows inconsistent relationships with alternative measures of PA, ranging from very low[30] to moderate,[174,175] to moderately strong,[176] and correlates more strongly with other traits, particularly disinhibition[30] or low conscientiousness.[175,176] Thus, Reward Dependence and Novelty Seeking may be less precise indicators of the PA construct. However, the constructs measured

by these traits do show associations with certain clinically relevant behaviors among individuals with schizophrenia (eg, substance abuse, violence), so they may tap other relevant, personality-related variance.

Future studies should attempt to isolate the most important aspects of PA for schizophrenia by conducting more fine-grained, facet-level analyses of broad traits and studies of narrower trait measures (eg, anticipatory vs consummatory pleasure). For example, extraversion and positive emotionality can be parsed broadly into social and affective components which, in turn, can be decomposed into social affiliation/gregariousness vs dominance/assertiveness and energy vs positive emotions.[17,23] The relative relevance of these subcomponents for schizophrenia remains unclear. In this context, it should be noted that for NA-related measures, this review focused only on higher order traits and did not include narrower traits analogous to anhedonia. This reflects the available research in schizophrenia, which has typically focused on the broad trait of NA, perhaps in part because NA facets tend to be more strongly correlated than PA facets.[22] Nevertheless, determining whether certain facets of NA (eg, fear, sadness, anger) are particularly important for schizophrenia would be useful in future research.

Although there is some evidence that high NA and low PA distinguish schizophrenia from bipolar disorder, this pattern does not cross-sectionally distinguish individuals with schizophrenia from those with major depression. There is accumulating evidence that NA and PA at least partly, and perhaps primarily, account for the high comorbidity among mood and anxiety disorders, with each individual syndrome hypothesized to involve both common and unique affective trait components.[6] Similarly, affective traits may also help to account for the high prevalence of mood and anxiety disorders (ie, distress-based disorders) in schizophrenia. As discussed further below, some shared vulnerability factors between schizophrenia and anxiety and mood disorders may help to account for the high comorbidity among these conditions, whereas other unique traits may contribute more specifically to the development of schizophrenia.

Although schizophrenia is characterized by a distinctive pattern of affective traits, there is also a large range of individual differences in NA and PA among affected individuals. These traits appear to be relatively independent of clinical symptoms and neurocognitive functioning but show relations with several clinically relevant features, particularly functional outcome, quality of life, and stress reactivity. Higher NA is uniformly associated with worse functioning, whereas higher PA typically appears to play a protective role. Poor functional outcome is a common, treatment-resistant feature of schizophrenia.[177] Although factors such as clinical symptoms and neurocognitive deficits have been identified as important determinants of poor functional outcome, they account for only a portion of the variance in outcome.[178] Affective traits may represent additional factors that contribute to how well affected individuals are able to function.

The links between individual differences in affective traits and functional outcome in schizophrenia suggest that clinical interventions tailored to particular patient characteristics may help enhance adaptive functioning and life satisfaction. For example, patients with higher NA may benefit from interventions aimed at emotion regulation, stress management, and modifying negative schematic beliefs about themselves and others.[179,180] Patients with low PA may benefit from interventions to increase engagement in pleasurable experiences, mastery experiences, social networks, and physical activity, as well as modifying maladaptive expectations about pleasurable experiences.[181,182] Of course, many patients may experience both high NA and low PA, and interventions for these emotional styles are not incompatible. In the longer term, the rapidly expanding research into the neurobiological underpinnings of NA and PA may help to inform pharmacological treatment development.[25,183]

Affective Traits, Vulnerability to Schizophrenia, and Research Implications

Available evidence suggests that affective traits may play a contributing role in the development of schizophrenia-related psychopathology. Affective traits are believed to reflect individual differences across a broad range of functioning, subsuming both normal and abnormal processes.[6] Clark and Watson[184] proposed that, "what we call personality in one context shares a common origin (not only a genetic but perhaps environmental or learning-based etiologies as well) with what we call psychopathology in another" (p. 418). While affective traits have been investigated extensively in the context of vulnerability to mood, anxiety, and externalizing disorders,[6] the current review suggests that they are also quite relevant for the development of schizophrenia.

It is somewhat difficult to compare the patterns of affective traits across schizophrenia patients, patients with Cluster A personality disorders, family members, population-based studies, and psychometric schizotypy studies due to substantial variability in the number of relevant studies and the specific trait questionnaires used. However, 2 broad patterns emerge across these types of samples. First, physical or social anhedonia is elevated or identified a risk factor in each of type of sample except population-based samples, in which it has not yet been studied. Thus, available evidence consistently indicates that this facet of PA plays a key role in vulnerability to schizophrenia. Second, for broader affective traits, high NA and low PA are present or implicated as risk factors in 4 of the 5 types of samples, providing substantial evidence that these traits may also contribute to vulnerability. The exception to this pattern is family

members. Five of the 7 relevant family studies used questionnaires based on the Biosocial model and generally failed to identify differences in Harm Avoidance, Reward Dependence, or Novelty Seeking between relatives and comparison samples, while the 2 studies based on 3- or 5-Factor Model questionnaires provided mixed results. Although questionnaires based on the Biosocial model have also been used in schizophrenia, they have not to our knowledge been used the other types of samples considered. Thus, it is not clear whether the general absence of disturbances in trait NA and PA in family members reflects the specific, Biosocial model-based questionnaires that have been used in most family studies to date. Additional family studies that use questionnaires based on other models of personality will help to address this issue.

Specifying exactly how affective traits and psychopathology are related is extremely complex,[13] and much additional research will be required to understand these relationships in schizophrenia. We comment on just a few possible directions for future research. NA and PA/anhedonia are heritable traits, and abnormalities in these traits are stable features of schizophrenia that are also sometimes detectable in biological relatives of schizophrenia probands. These characteristics suggest that affective traits (particularly anhedonia) may be useful endophenotypes in genetic studies of schizophrenia.[185,186] Endophenotypes are hypothesized to reflect intermediate characteristics along the developmental pathway between genes and psychopathology. As such, they are believed to have simpler genetic architectures than the diverse signs and symptoms that comprise conventional clinical syndromes. Although the neurobiological and genetic bases of emotional endophenotypes is an active topic of research in affective neuroscience,[187] emotional variables have received limited attention in schizophrenia research that employs the endophenotype strategy.[188,189] Findings that high NA and low PA are also associated with disorders such as depression and anxiety have led some to propose that these and related traits may reflect shared vulnerability across these disorders.[6,190,191] Genetic studies of schizophrenia may benefit from investigating emotional characteristics in conjunction with more commonly studied candidate endophenotypes, such as neurocognitive deficits.[188,192]

Although schizotypy is widely regarded as a heterogeneous construct, most research in this area consists of cross-sectional comparisons of individuals classified as either schizotypal or not schizotypal. It will be useful in future studies to examine affective trait correlates of particular schizotypy facets or factors. Factor-analytic studies of schizophrenia and schizotypy have yielded very consistent structures that suggest analogous positive, disorganized, and negative symptom domains.[193] As reviewed previously, a few studies indicate that NA is broadly associated with all symptom domains of schizotypy, whereas PA/anhedonia appears more specifically related to negative schizotypy features (and possibly primary negative symptoms of schizophrenia).[61,90] Further investigation of symptom domains or factors, particularly using longitudinal designs, may yield more informative findings about the developmental processes that contribute to symptom dimensions or subtypes of schizophrenia.

In future studies of both schizotypy and schizophrenia, it will be important to advance beyond simple examinations of NA and PA to more comprehensive evaluations of how they interact with other traits and environmental factors. Although this review focused on broad trait measures of emotional experience, the growth of affective science has led to the development of trait measures that assess a range of emotion-related processes.[194] For example, recent studies of schizophrenia have begun to use trait measures of emotion regulation, empathy, and behavioral approach/avoidance tendencies.[88,195–197] Studies of schizotypy have examined processes such as emotional clarity, emotional awareness, alexithymia, and attention to emotions.[161,162,167,198] Investigations of other emotion-related traits, and their interactions with NA and PA, can help clarify emotional disturbances associated with schizophrenia-related psychopathology. It is noteworthy that basic research in personality and affective science often demonstrates sex differences in some emotional traits, including NA, PA, expressivity, and awareness.[199–201] Investigations of affective traits may also help shed light on poorly understood sex differences found in age of onset, psychosocial adjustment, and outcome in schizophrenia.[202]

It will also be useful to examine interactions with traits that are not expressly affective. For example, schizotypal personality disorder is associated with traits of mistrust and detachment.[115] Some researchers have proposed that conceptually related traits labeled ''oddity'' or ''peculiarity'' (reflecting cognitive/perceptual anomalies and disorganization) are distinct from other Big Five personality traits and may be particularly associated with schizophrenia-related psychopathology.[115,203,204] The interaction of such traits with NA and PA could be informative for understanding risk in those individuals not yet evincing clinical diagnoses. Consistent with this possibility, a longitudinal study by Kwapil et al.[205] found that anhedonia interacted with magical ideation to result in the highest rates of psychosis.

To unravel causal relationships within these interactive models, it ultimately will be necessary to understand how individual differences in affective traits reported on questionnaires are manifest in the physiology, cognition, and behavior of individuals with schizotypy and schizophrenia. For example, affective traits may contribute to domains such as stress reactivity,[190,206] the generation of stressful environments,[55] or negative appraisals of life events,[207] which could potentiate clinical outcomes.

It may be particularly useful to investigate how these traits are expressed within interpersonal behaviors. For example, socially anhedonic individuals have been found to show diminished emotional expressivity and engagement during social exchanges, which may create unrewarding or uncomfortable interactions for others.[208] Such behaviors could contribute to social rejection and isolation, thereby depriving these individuals of social reward and opportunities to assess the validity of their beliefs and ideas and ultimately leading to clinical deterioration. Alternatively, it has been proposed that delusional beliefs may develop as a result of increased levels of attention to emotion being combined with other factors such as anomalous experiences, a jumping-to-conclusions bias, or aberrant assignment of salience to external objects and internal representations.[167] We believe that continued investigation of affective traits will provide key insights into to the complex causes of schizophrenia and its associated functional deficits.

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