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Unpacking the Anhedonia Paradox Across the Psychosis Continuum: The Role of the Positivity Offset

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Abstract

Conceptual models of negative symptoms evolved to explain how seemingly intact hedonic capacity fails to translate to motivated behavior in SZ; however, Cacioppo's Evaluative Space Model indicates that hedonic deficits are apparent in the form of a reduced positivity offset (i.e., experiencing lower levels of positive relative to negative emotion when affective input is absent). Prior evidence indicates that the positivity offset is reduced across the psychosis continuum and associated with negative symptoms, suggesting it may contribute to the disjunction between hedonic and volitional responding in SZ, as well as differences in hedonic capacity along the psychosis continuum. The current study examined the positivity offset during a laboratory-based emotional experience task in two samples: (1) individuals with SZ (n = 98) and healthy controls (CN: n = 84); (2) individuals at clinical high-risk (CHR) for psychosis (n = 45) and CN (n = 51). Results indicated that SZ is best characterized by intact hedonic capacity, as well as a reduced positivity offset that is associated with more severe anhedonia and avolition. CHR demonstrated an intact positivity offset that was not associated with anhedonia or avolition. Findings add to current conceptual models of negative symptoms by demonstrating distinct emotional abnormalities that may underlie anhedonia at different phases of psychotic illness.

Keywords: Ultra-high-risk; attenuated psychosis syndrome; schizophrenia; emotion; negative symptoms

Introduction

Negative symptoms are a highly prevalent and debilitating feature of schizophrenia (SZ) that are associated with a host of poor outcomes, including lower quality of life (Ritsner et al., 2011), cognitive impairment (Foussias & Remington, 2008; Green & Harvey, 2014), and poor social, role, and recreational functioning (Foussias & Remington, 2008). Deficits in motivation and pleasure (i.e., anhedonia and avolition) are the core drivers of this dysfunction and therefore pertinent intervention targets (Strauss et al., 2021); however, the field has made limited progress toward developing effective treatments for negative symptoms because their mechanistic processes are not yet fully understood.

Contributing to these gaps in understanding is the so-called "liking-wanting" anhedonia paradox (Pizzagalli, 2010; Strauss & Gold, 2012), which describes how seemingly intact hedonic capacity fails to translate into motivated behavior among individuals with SZ. Previously, decoupled hedonic and volitional responding in SZ has been attributed to impairments in generating, updating, and maintaining mental representation of reward value (Gold et al., 2008). However, Strauss et al. (2017) proposed that the liking-wanting paradox may actually be a misnomer, and suggested that hedonic abnormalities can be detected in SZ that contribute to impairments in generating motivated approach behaviors when anhedonia is viewed in relation to more sophisticated conceptual and computational models.

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Specifically, the frameworks posited in Cacioppo's seminal Evaluative Space Model (ESM) (Cacioppo, 1999; Cacioppo & Berntson, 1994; Cacioppo et al., 2011; Norris et al., 2010) were applied to examine whether anhedonia could be detected in SZ, even in the presence of intact hedonic capacity (Strauss et al., 2017). The ESM proposes that self-reported positive and negative emotions are influenced by separate motivational systems. Both motivational systems are driven by activation functions (i.e., the extent to which affective input into the system produces motivational output from that system) that allow positive and negative emotional responses to give rise to motivated approach or withdrawal behaviors. At low levels of input, the affective system is calibrated to activate the positivity function to yield greater levels of positive than negative emotion, resulting in approach motivation. This tendency of having greater levels of positivity than negativity at low levels of arousal is referred to as the "positivity offset." In contrast, at high levels of evaluative activation, the affective system is calibrated to activate the negativity function to yield greater levels of negative than positive emotion, resulting in withdrawal motivation. This describes the "negativity bias," or the tendency to respond with greater levels of negative than positive emotion at high levels of arousal. Both activation functions are adaptive in different environments, such that the positivity offset promotes exploratory behavior in neutral contexts and the negativity bias promotes withdrawal behavior in highly negative or risky contexts.

Strauss et al. (2017) applied the ESM framework and methodology to evaluate self-reported positive emotion, negative emotion, and arousal in relation to pleasant, unpleasant, and neutral scenes in a sample of outpatients with SZ. Compared to healthy controls (CN), individuals with SZ demonstrated a reduced positivity offset that was predictive of greater trait anhedonia. In a follow-up study, Bartolomeo et al. (in press) examined whether the positivity offset deficit could be demonstrated in daily life using ecological momentary assessment (EMA) and importantly, whether it was associated with reductions in motivated behavior measured via EMA and passive digital phenotyping. Replicating this prior laboratory-based study (Strauss et al., 2017), results indicated that the positivity offset was diminished in the real-world and associated with more severe anhedonia and avolition measured via clinical interviews, EMA surveys, and passive digital phenotyping (i.e., accelerometry). Thus, findings obtained using both laboratory and EMA/digital phenotyping methods suggest that the positivity offset is reduced in SZ and associated with greater severity of anhedonia and reductions in approach behaviors; such findings suggest that positivity offset impairments may help explain the liking-wanting anhedonia paradox (i.e., individuals with SZ fail to initiate approach behavior due to reductions in the positivity offset, even in the context of intact hedonic capacity).

A second anhedonia paradox has also emerged over recent years, which Strauss and Cohen (2018) termed the "schizophrenia-spectrum anhedonia paradox." This paradox refers to an emerging literature indicating that although hedonic capacity is intact in the most severe disorder within the psychosis continuum (i.e., schizophrenia), it is impaired in those with less severe clinical presentations at the milder end of the continuum, such as schizotypal personality disorder and among individuals at clinical high-risk for psychosis (CHR) (Cohen et al., 2012; Cohen & Minor, 2010; Gruber et al., 2018; Strauss et al., 2018). For example, those with psychometrically defined schizotypy self-report lower levels of positive emotion in response to pleasant stimuli compared to healthy controls (Cohen et al., 2011; Cohen et al., 2012; Najolia et al., 2011) and demonstrate reduced neurophysiological responses to pleasant stimuli (Martin et al., 2020). Similarly, individuals at CHR evidence deficits in subjective and neurophysiological responses to pleasant stimuli relative to controls (Gruber et al., 2018; Strauss et al., 2018). Why disorders at the milder end of the psychosis continuum display a true hedonic deficit and those at the most severe end do not is paradoxical. Strauss and Cohen (2018) proposed several explanations for this apparent schizophrenia-spectrum anhedonia paradox, including: 1) mood and anxiety disorders being more prevalent in CHR and schizotypy than SZ; 2) antipsychotics having a normalizing effect in SZ, with CHR and schizotypy being much less likely to be prescribed antipsychotics; 3) greater cognitive impairment in SZ than CHR and schizotypy may be paradoxically protective in SZ, causing less awareness of hedonic deficits and therefore more normal emotional self-reports; 4) more frequent effects of environmental stress on schizotypy and CHR, which causes

subsequent "stress-induced anhedonia" effects. However, an unexplored possibility is that the schizophrenia-spectrum anhedonia paradox is not a paradox at all, and anhedonia is present among the more severe and milder ends of the psychosis continuum when conceptualized as a reduction in the positivity offset. Consistent with this possibility, a recent study by Riehle et al (2022) that examined a community sample which included participants with sub-threshold psychotic-like experiences reported an association between anhedonia and reductions in the positivity offset. Further research into transdiagnostic emotional experience abnormalities will be important for untangling the "schizophrenia-spectrum anhedonia paradox" and identifying mechanisms underlying negative symptoms that can be used to inform personalized treatments. Such efforts may also be important for preventing the progression of negative symptoms among individuals at CHR for psychosis, for whom negative symptoms are not only highly prevalent, but also associated with blunted emotional experience and heightened conversion risk (Demjaha et al., 2010; Paetzold et al., 2021; Piskulic et al., 2012; Valmaggia et al., 2013). Additionally, it is unclear whether or how mood symptoms influence hedonic capacity and the positivity offset across the psychosis continuum, although there is evidence that the positivity offset is also reduced among adults with major depressive disorder (Gollan et al., 2016). Examining these factors may provide insight into personalized targets for those with and without mood symptoms.

To evaluate the schizophrenia-spectrum anhedonia paradox, the current study used mathematical approaches from the ESM to replicate prior laboratory and naturalistic evidence for the reduced positivity offset in SZ and its association with negative symptoms. We also extended prior studies by examining participants at CHR for psychosis and the role of current mood symptoms across SZ and CHR samples. The following hypotheses were made: 1) Consistent with prior laboratory-based and EMA studies (Riehle et al., 2002; Strauss et al., 2017; Bartolomeo et al., in press), participants with SZ and CHR would exhibit a diminished positivity offset that is associated with clinically rated anhedonia and avolition; 2) The negativity bias would be intact in SZ and CHR based on past findings (Bartolomeo et al., in press; Strauss et al., 2017); 3) SZ would demonstrate an intact or elevated hedonic capacity measured by the slope for the positivity function, whereas CHR would show a reduced hedonic capacity compared to CN.

Method

Study 1

Participants

Ninety-eight individuals with DSM-IV (*Diagnostic and statistical manual of mental disorders : DSM-IV*, 1994) or DSM-5 (*Diagnostic and statistical manual of mental disorders : DSM-5™*, 2013) diagnoses of schizophrenia (n = 62) or schizoaffective disorder (n = 36) (SZ) and 84 psychiatrically healthy controls (CN) participated in the study. Groups did not significantly differ on age, sex, ethnicity, or parental education. Individuals with SZ had lower personal education and cognitive functioning than CN. SZ had moderately severe negative symptoms on average (see Table 2).

Individuals with SZ were recruited from local community outpatient mental health centers and advertisements. Clinical diagnoses were determined via either the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (First et al., 2002) or the Structured Clinical Interview for DSM-5 Disorders (SCID-5) (First, Williams, Benjamin, & Spitzer, 2015). CN were recruited from the local community using posted flyers and electronic advertisements. CN were free of current psychiatric diagnoses as established via the SCID-I or SCID-5, no current SZ-spectrum personality disorders as established via the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II) (First, 1997) or the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) (First et al., 2015), family history of psychosis, and psychotropic medications. All participants denied lifetime neurological disease and did not meet criteria for a substance abuse disorder within the last 6 months.

Procedure

All participants provided written informed consent and received monetary compensation for their participation. Study procedures were approved by the State University of New York at Binghamton and University of Georgia Institutional Review Boards. Participants completed a series of measures to assess diagnostic inclusion and exclusion criteria, including the SCID-I to assess current and lifetime criteria for psychiatric disorders within the DSM-IV. CN were also administered the Cluster A section of the SCID-II to assess current and lifetime criteria for DSM-IV SZ-spectrum personality disorders. All participants completed the Wechsler Test of Adult Reading (WTAR) (Wechsler, 2001) to measure premorbid IQ. For sympom assessments, SZ participants were administered the Brief Negative Symptom Scale (BNSS) (Kirkpatrick et al., 2010) and the Positive and Negative Syndrome Scale (PANSS) (Kay, Fiszbein, & Opler, 1987). Interviews were conducted by lab personnel or doctoral students trained to reliability standards (inter-rater reliability of alpha ≥ 0.80) who consulted with the PI (GPS) to establish consensus diagnoses and symptom ratings. After completing all clinical interviews, participants proceeded to the emotional picture viewing tasks.

The emotional experience task was based on the behavioral paradigm used to index the positivity offset by Strauss et al. (2017). During the task, participants passively viewed a series of pleasant, unpleasant, and neutral images from the International Affective Picture System (IAPS) (Lang et al., 1997). The task contained 24 images (8 pleasant, 8 unpleasant, 8 neutral. Pleasant, unpleasant, and neutral stimuli differed in normative IAPS valence (unpleasant < neutral < pleasant) and arousal (neutral < pleasant, unpleasant), while pleasant and unpleasant stimuli did not significantly differ in normative arousal. Stimuli depicted social and nonsocial content. Unpleasant stimuli depicted threat, injury, disgust, and phobic scenes. Pleasant stimuli depicted landscapes, food, romantic scenes, animals, and adventure. Neutral stimuli depicted common objects, expressionless people, and nature. IAPS stimuli included in the task are listed in the Supplemental Material. Following each image, participants responded to the following: 1) How positive does the picture make you feel?; 2) How negative does the picture make you feel?; and 3) How calm/excited does the picture make you feel (i.e., subjective arousal)? Ratings were made using the self-assessment manikin anchored between “not at all” to “extremely”. The order of ratings was kept constant on every trial to reduce cognitive demand.

Data Analysis

The positivity offset and negativity bias were calculated according to Ito and Cacioppo (2005). Two regression equations were conducted on each subject’s individual-level valence and arousal ratings from the emotional experience task. Specifically, the equation $E = Ax + b$ was used to model the positivity and negativity functions, where where rE is either the positive or negative subjective emotional response rating to either pleasant or unpleasant stimuli viewed during the emotion task, and rA is the mean arousal rating for either neutral and unpleasant stimuli or neutral and pleasant sitmuli. For the positivity function, the resulting intercept (b in the equation) represents the positivity offset (i.e., the level of positive emotion when affective input is absent). For the negativity function, the resulting slope (x in the equation) represents the negativity bias (i.e., the rate at which negative emotion changes with increasing affective input). To characterize the positivity offset and negativity bias at the individual level, two difference scores were calculated: 1) positive intercept − negative intercept to model the positivity offset and 2) negative slope – positive slope to model the negativity bias. See Table 1 for definitions and formulas for the ESM parameters. Separate one-way ANOVAs were conducted to compare positivity offset and negativity bias difference scores, as well as the raw intercepts and slopes for the positivity and negativity functions, between individuals with SZ and CN.

Bivariate (Spearman) correlations were conducted to determine if the positivity offset and negativity bias difference scores were associated with clinically rated anhedonia and avolition within the SZ group. To examine associations with mood symptoms, bivariate correlations were also conducted with the Depression item from the PANSS. All analyses using the positivity offset and negativity bias difference scores were repeated with the raw positivity and negativity intercept and slope parameters. Bonferroni’s

correction was used to adjust correlation coefficients for multiple comparisons as described in Curtin and Schulz (1998). Correlation plots for significant correlations are displayed in the Supplemental Material. Exploratory analyses, including standard comparisons of valence and arousal and examining effects of sex, medication, and cognition are described in Supplemental Materials. Supplemental analyses were also conducted with the SZ group broken out into individuals with SZ and schizoaffective disorder compared to CN.

Table 1. Evaluative Space Model Definitions and Formulas

Variable	Definition	Equation
Positivity Intercept	Positive affective output when affective input is absent	b in the equation E = Ax + b where E = mean unipolar positivity rating to neutral and pleasant stimuli and A = mean arousal rating to neutral and pleasant stimuli
Positivity Slope	Change in positive affective output per 1 unit change in arousal	x in the equation E = Ax + b where E = mean unipolar positivity rating to neutral and pleasant stimuli and A = mean arousal rating to neutral and pleasant stimuli
Negativity Intercept	Negative affective output when affective input is absent	b in the equation E = Ax + b where E = mean unipolar negativity rating to neutral and unpleasant stimuli and A = mean arousal rating to neutral and unpleasant stimuli
Negativity Slope	Change in negative affective output per 1 unit change in arousal	x in the equation E = Ax + b where E = mean unipolar negativity rating to neutral and unpleasant stimuli and A = mean arousal rating to neutral and unpleasant stimuli
Positivity Offset	Greater positive affective output than negative affective output when affective input is absent leading to approach motivation	Positivity Intercept – Negativity Intercept
Negativity Bias	Greater gain in negative affective output than positive affective output with increasing levels of affective input leading to withdrawal motivation	Negativity Slope – Positivity Slope

Note. Adapted from Cacioppo, Berntson, Norris, & Gollan (2012).

Study 2

Participants

Forty-five individuals at clinical high-risk for psychosis (CHR), including 17 individuals with a comorbid mood disorder diagnosis (i.e., depressive disorders, bipolar disorders) and 28 without, and 51 healthy controls (CN) participated in the study. CHR participants were recruited from two psychosis risk evaluation programs directed by the PI that consisted of diagnostic and monitoring evaluations for youth referred by community clinicians. Participants were also recruited via online and print advertisements, in-person presentations to community mental health centers, and calls or in-person meetings with members of the local school system. All CHR participants met criteria for a prodromal syndrome determined by the Structured Interview for Prodromal Syndromes (SIPS) (Miller et al., 2003), including brief intermittent psychotic symptoms (n = 1), attenuated positive symptoms (n = 42), and

genetic risk and deterioration (n = 2). None of the CHR participants met lifetime criteria for a DSM-5 psychotic disorder.

CN participants were recruited from the local community using print and online advertisements. Exclusion criteria for CN included current major psychiatric disorder diagnoses, SZ-spectrum personality disorders established by the SCID-5 and SCID-5-PD, family history of psychosis, and currently taking psychotropic medications. All participants were free from lifetime neurological disease. Groups did not significantly differ on age, ethnicity, sex, personal education, or parental education (see Table 2).

Table 2. Participant Demographics

	Study 1		
	SZ (n=98)	CN (n=84)	Test statistic
Age	39.60 (12.42)	39.77 (11.47)	.01
Parental Education	13.58 (2.70)	13.62 (2.44)	.01
Personal Education	12.94 (2.26)	15.74 (2.83)	44.39***
Female (%)	49.00	39.30	2.51
Race (%)	-	-	13.66
Black	21.40	16.67	-
Asian	1.00	5.95	-
LatinX	3.10	9.52	-
White	66.30	60.71	-
Multiracial	7.10	4.76	-
Other	1.01	2.38	-
Medication (n)			
Antipsychotic	50	-	-
Mood Stabilizer	21	-	-
Antidepressant	35	-	-
Anxiolytic	21	-	-
Stimulant	5	-	-
None	18	-	-
MCCB	37.58 (13.27)	50.29 (10.82)	45.12***
BNSS Total	17.92 (15.01)	-	-
Avolition	2.09 (1.78)	-	-
Anhedonia	1.66 (1.57)	-	-
Asociality	1.52 (1.45)	-	-
Alogia	.71 (1.33)	-	-
Blunted Affect	1.36 (1.67)	-	-
Study 2			
	CHR (n=45)	CN (n=51)	Test statistic
Age	20.38 (2.49)	20.22 (1.94)	.13

Parental Education	15.09 (2.50)	15.61 (2.29)	1.13
Personal Education	13.58 (1.71)	14.00 (1.54)	1.62
Female (%)	75.56	80.39	.33
Race (%)	-	-	2.67
Black	6.67	3.92	-
Asian	13.33	17.65	-
LatinX	11.11	5.89	-
White	66.67	72.55	-
Multiracial	2.22	0.00	-
Medication (n)			
Antipsychotic	2	-	-
Mood Stabilizer	2	-	-
Antidepressant	9	-	-
Anxiolytic	2	-	-
Stimulant	1	-	-
None	32	-	-
BNSS Total	12.91 (11.81)	-	-
Avolition	1.27 (1.34)	-	-
Anhedonia	1.61 (1.41)	-	-
Asociality	.88 (1.16)	-	-
Alogia	.49 (1.08)	-	-
Blunted Affect	.87 (1.40)	-	-

Note. SZ = schizophrenia group; CHR = clinical high-risk group; CN = control group. MCCB = MATRICS Consensus Cognitive Battery. PANSS = Positive and Negative Syndrome Scale. BNSS = Brief Negative Symptom Scale. Values reflect mean (standard deviation) unless otherwise indicated. Symptom ratings values reflect average score for each domain listed except total. *$p < .05$, **$p < .01$, ***$p < .001$.

Procedure

Participants provided written informed consent and received monetary compensation for their participation. Study procedures were approved by the State University of New York at Binghamton and University of Georgia Institutional Review Boards. Participants completed a structured clinical interview to rate the SCID-5, SCID-5-PD, SIPS, BNSS, and the Global Functioning Scale: Social (GFS:S) and Global Functioning Scale: Role (GFS:R) scales (Cornblatt et al., 2007). Interviews were conducted by the PI or examiners trained to reliability standards (>0.80) who established clinical consensus with the PI. In CHR participants, cross-sectional conversion risk was calculated based on the formula developed by Zhang et al. (2018) incorporating SIPS items measuring functional decline, positive, negative, and general symptoms. After the interview, participants completed the same emotional experience task used in Study 1.

Data Analysis

The data analytic plan for Study 2 was the same as Study 1, with the exception of exploratory analyses assessing the relationship between the positivity offset difference scores with medication status that were not conducted because too few participants in the CHR group were prescribed antipsychotics.

Bivariate (Spearman) correlations between the positivity and negativity parameters with cross-sectional conversion risk scores were also conducted (Zhang et al., 2018). To examine associations with mood symptoms, bivariate correlations were also conducted with the Dysphoric Mood item from the SIPS. Correlation plots are displayed in the Supplemental Material. Supplemental analyses were also conducted with the CHR group broken out into individuals with and without co-morbid mood disorders compared to CN.

Results

Study 1

Consistent with past findings, CN demonstrated the prototypical positivity offset (t = 3.40, p = .001). In contrast, the positivity offset was not detected in SZ based on nonsignificant differences between the intercepts for the positivity and negativity functions (t = 1.67, p = .10). As expected, the positivity offset intercept difference score was significantly reduced in SZ compared to CN. None of the raw positivity parameters significantly differed between groups.

CN also demonstrated the prototypical negativity bias, evidenced by a significantly greater slope for the negativity than positivity function (t = -2.88, p = .01). SZ participants displayed nonsignificant differences between slopes for the positivity and negativity functions, suggesting a lack of negativity bias (t = -1.61, p = .11). Group differences in the negativity bias and the raw negativity parameters were nonsignificant. See Table 2 for results of group comparisons and Figure 1 for regression equations depicting the positivity and negativity functions.

In the SZ group, greater reductions in the positivity offset were associated with more severe avolition (r = -.31, p = .003) and anhedonia (r = -.23, p = .03)1 measured by the BNSS. When correlations were conducted with the raw positivity and slopes and intercepts, only associations between avolition and the positivity intercept (r = -.35, p = .001) and slope (r = .30, p = .004)1 were significant, such that more severe avolition was associated with a lower intercept and greater slope for the positivity function. Among individuals with SZ, higher negativity bias scores were associated with more severe avolition (r = .23, p = .03)1, while associations with anhedonia were nonsignificant (r = .14, p = .18). Neither the raw negativity intercept or slope were significantly correlated with BNSS avolition or anhedonia. Lastly, correlations between the severity of depressive symptoms and all of the positivity and negativity parameters were nonsignificant.

Study 2

Both CN and CHR groups demonstrated the prototypical positivity offset, with significantly higher intercepts for the positivity than negativity function (CN: t = 6.08, p < .001; CHR: t = 4.92, p < .001). Group differences in the positivity offset intercept difference score were nonsignificant, as were comparisons of the raw positivity parameters. Both groups also displayed the negativity bias, with significantly higher slopes for the negativity than positivity function (CN: t = -5.40, p < .001; CHR: t = -4.22, p < .001). Group differences in the negativity bias difference score and the raw negativity parameters were nonsignificant. See Table 2 for results of group comparisons and Figure 2 for regression equations depicting the positivity and negativity functions.

In participants at CHR for psychosis, correlations between intercept difference scores and clinically rated avolition (r = -.03, p = .84) and anhedonia (r = -.13, p = .39) were nonsignificant. When using the raw positivity scores, there was a significant association between anhedonia and the positivity slope (r = -.30, p = .045)[1], such that more severe anhedonia was associated with lower hedonic capacity. The associations between negativity bias difference scores and clinically rated avolition (r = -.17, p = .27) and anhedonia (r = -.17, p = .27) were nonsignificant among CHR participants, as were all correlations with the raw negativity parameters. Correlations between cross-sectional conversion risk with the positivity offset (r = .01, p = .94) and negativity bias (r = -.11, p = .47) difference scores were nonsignificant, as were correlations with the raw positivity and negativity parameters. Finally, lower

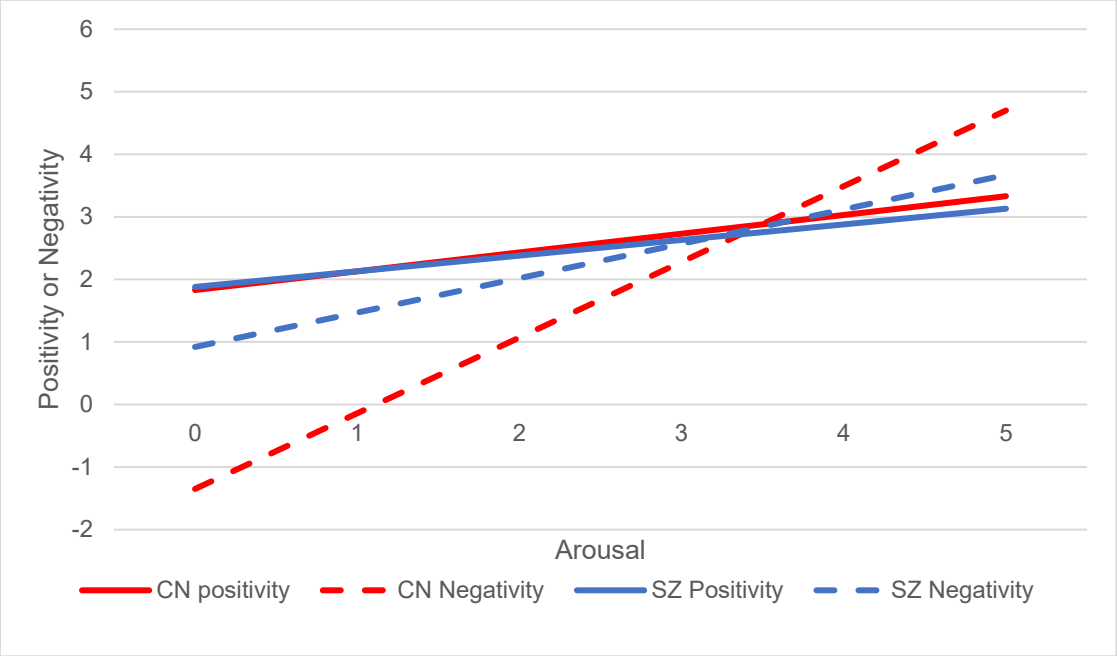
raw positivity intercepts were associated with more severe depressive symptoms among individuals at CHR (r = -.30, p = .04)[1], but no other variables were associated with depression severity.

Table 2. One-way ANOVA Results Comparing Positivity and Negativity Parameters in Clinical and Control Groups

	SZ	CN	Test Statistic
Positivity Intercept	1.88 (9.19)	1.83 (1.84)	$F(1, 182) = .002$, $p = .972$, $\eta_p^2 = 0$
Negativity Intercept	.92 (7.95)	-1.35 (8.66)	$F(1, 182) = 3.39$, $p = .07$, $\eta_p^2 = .02$
Positivity Slope	.25 (3.02)	.30 (.67)	$F(1, 182) = .03$, $p = .88$, $\eta_p^2 = 0$
Negativity Slope	.55 (2.64)	1.21 (2.87)	$F(1, 182) = 2.57$, $p = .11$, $\eta_p^2 = .01$
Positivity Offset Difference Score	.96 (5.70)	3.19 (8.60)	$F(1, 182) = 4.35$, $p = .04$, $\eta_p^2 = .02$
Negativity Bias Difference Score	.30 (1.86)	.91 (2.88)	$F(1, 182) = 2.89$, $p = .09$, $\eta_p^2 = .02$
	CHR	CN	Test Statistic
Positivity Intercept	1.15 (1.53)	1.53 (1.71)	$F(1, 96) = 1.26$, $p = .26$, $\eta_p^2 = .01$
Negativity Intercept	-.42 (2.01)	-.80 (12.60)	$F(1, 96) = .82$, $p = .37$, $\eta_p^2 = .01$
Positivity Slope	.49 (.43)	.41 (.57)	$F(1, 96) = .63$, $p = .43$, $\eta_p^2 = .01$
Negativity Slope	.91 (.58)	1.11 (.61)	$F(1, 96) = 2.79$, $p = .10$, $\eta_p^2 = .03$
Positivity Offset Difference Score	1.57 (2.14)	2.32 (2.73)	$F(1, 96) = 2.20$, $p = .14$, $\eta_p^2 = .02$
Negativity Bias Difference Score	.42 (.66)	.70 (.93)	$F(1, 96) = 2.95$, $p = .09$, $\eta_p^2 = .03$

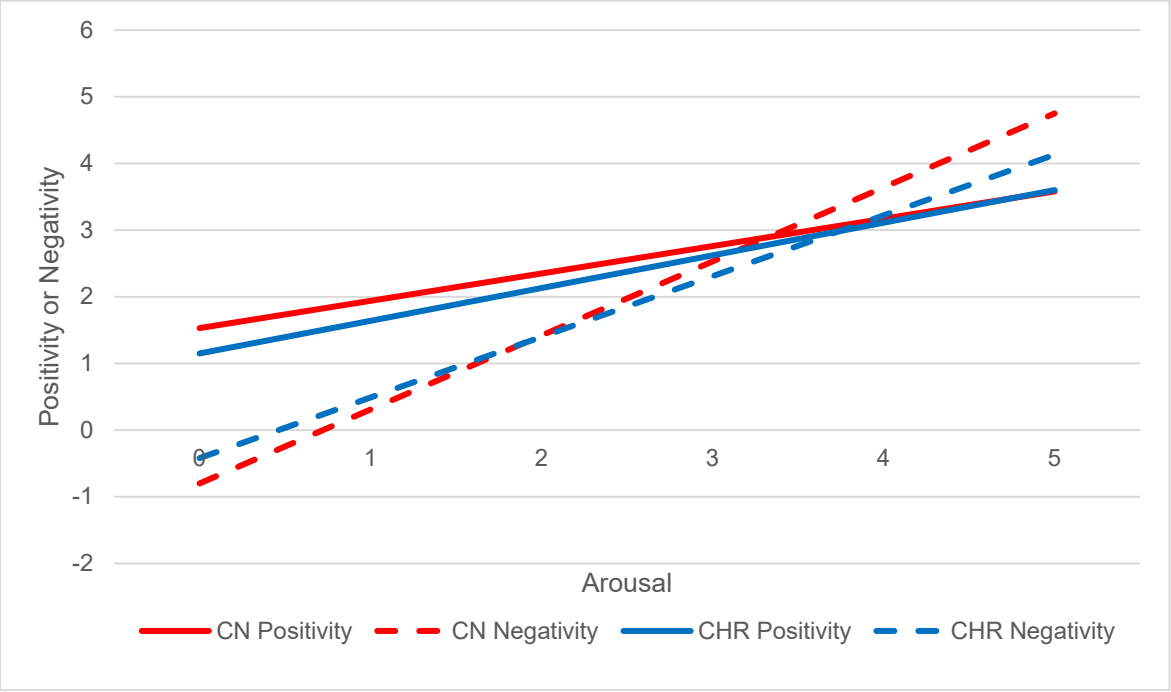
Note. SZ = schizophrenia group; CHR = clinical high-risk group; CN = control group. Positivity Offset Difference Score = Positivity Intercept – Negativity Intercept. Negativity Bias Difference Score = Negativity Slope – Positivity Slope. Values reflect Mean (SD) unless otherwise indicated.

Figure 1. Positivity Offset and Negativity Bias Functions in Psychosis and Control Groups



Note. SZ = schizophrenia group; CN = control group. The solid red and blue lines represent how the positivity function is calibrated to respond at different levels of arousal in CN and SZ, respectively. The dashed red and blue lines represent how the negativity system is calibrated to respond at different levels of arousal in CN and SZ, respectively. The intercepts of these functions represent the level of positive or negative emotion when affective input is absent. The difference of the intercepts for the positivity and negativity functions (positive – negative) represents the positivity offset. The slopes of these functions represent how positive or negative emotion change per one unit change in affective input. The difference of the slopes for the positivity and negativity functions (negative – positive) represents the negativity bias.

Figure 2. Positivity Offset and Negativity Bias Functions in Clinical High-Risk and Control Groups



Note. CHR = clinical high-risk group; CN = control group.

Discussion

The current study applied the ESM to evaluate the link between emotional experience and negative symptoms in individuals at CHR and those with full psychotic disorders to determine whether abnormalities in the positivity offset account for the liking-wanting and schizophrenia-spectrum anhedonia paradoxes. Consistent with hypotheses, group-level analyses indicated that individuals with SZ demonstrated a reduced positivity offset and an intact negativity bias compared to CN. However, there was a trend toward a significant group difference in the negativity bias, suggesting that individuals with SZ demonstrated a lesser gain in negative emotional responding with increasing levels of affective input (i.e., lower withdrawal motivation) relative to CN. Additionally, there was also a trend toward a significant group difference in the intercept for the negativity function, which may account for the reduced slope in negativity in SZ compared to CN. As such, emotional experience abnormalities in SZ primarily involve higher intercepts for negativity. Correlations also indicated that lower positivity offset difference scores were associated with greater severity of clinically rated anhedonia and avolition. This replicates our original laboratory-based study showing the same pattern in those with chronic SZ (Strauss et al., 2017), as well as findings linking low positivity offset scores to deficits in real-world motivated behavior using digital phenotyping (Bartolomeo et al., in press). Collectively, these findings suggest that the positivity offset theory may in part explain the liking-wanting anhedonia paradox. Although prior studies have shown a disjunction between hedonic capacity and volitional behavior (e.g., Heerey & Gold, 2007), which was logically interpreted as a decoupling of intact emotional experience and motivation, the current findings point to a more nuanced type of hedonic deficit that impedes motivated behavior. The nature of the hedonic abnormality is not simply a deficit in capacity, but rather a reduction in the positivity offset (i.e., a reduction in levels of positive relative to negative affect specifically in neutral contexts). This finding is supported not only by correlations with clinical ratings that encompass frequency of pleasurable activity, but also reductions in the frequency of positive experiences and volitional behavior via measured EMA surveys and accelerometry (Bartolomeo et al., in press). Thus, these findings help explain the liking-wanting paradox by highlighting a connection between the positivity offset and the frequency of pleasurable experiences (measured by clinical ratings), but not hedonic capacity (measured by the slope for the positivity function). Further, consistent with previous findings from EMA and laboratory studies of elevated negative affective responding in SZ (Cho et al., 2017; Cohen and Minor, 2010; Horan et al., 2008), reductions in the positivity offset may be driven by a greater intercept for the negativity function.

Importantly, this interpretation regarding the liking-wanting paradox is made in the context of intact hedonic capacity based on nonsignificant group differences in the raw slope metric for the positivity function. Evidence for a reduction in the positivity offset in conjunction with the intact positivity slope suggests that traditional notions of anhedonia in SZ as a reduction in the capacity for pleasure may be incorrect. Anhedonia can exist as a reduction in the positivity offset, even in the context of normal hedonic capacity. In fact, more severe avolition scores were associated with higher hedonic capacity as measured via slope for positive emotion (i.e., the opposite of what one would expect if hedonic capacity deficits drove motivation difficulties), suggesting that the positivity offset deficit can also exist amidst a decoupling between hedonic capacity and reductions in motivated behavior. Thus, our previous notion that anhedonia is characterized by a reduction in the positivity offset, not diminished hedonic capacity, does not appear to conflict with recent proposals that motivational deficits in SZ are driven by aspects of reward processing other than hedonic capacity, such as value representation (Gold et al., 2007).

Replicating prior evidence for a hedonic deficit among individuals at CHR for psychosis (Gruber et al., 2018; Strauss et al., 2018), standard analyses of valence and arousal ratings indicated that the CHR group endorsed reduced levels of positive emotion in response to pleasant images compared to CN. However, contrary to hypotheses, the positivity offset and hedonic capacity measured via the slope for the positivity function were both intact among individuals at CHR. Consistent with hypotheses, the negativity bias was intact in the CHR group. Although the positivity offset difference score was not

significantly correlated with anhedonia or avolition, lower hedonic capacity indexed by the slope for the positivity function was associated with more severe anhedonia among individuals at CHR. In contrast to prior findings from Riehle et al., (2022), results indicated that the positivity offset is not reduced or associated with anhedonia across the psychosis continuum. Instead, the nature of the hedonic abnormality in SZ is characterized by a diminished positivity offset that impedes approach motivation in neutral contexts. In contrast, individuals at CHR for psychosis have an intact positivity offset and ability to increase positive emotional responding as affective input increases. Although the present findings do point to a differential pattern of affective responding and associations with negative symptoms between individuals with SZ and at CHR, the ESM approach did not reveal distinct components of the positivity or negativity functions that could account for the schizophrenia-spectrum anhedonia paradox. Notably, the correlation between reduced hedonic capacity measured by the slope for the positivity function and more severe clinically-rated anhedonia was specific to CHR and was not detected in SZ; however, the nonsignificant difference in positivity slope between CHR and CN indicates that a deficit in hedonic capacity does not fully explain the schizophrenia-spectrum paradox.

Supplemental analyses also examined whether the positivity offset account of anhedonia was primarily driven by mood diagnosis/symptoms across phases of illness. In SZ, all correlations with mood symptoms were nonsignificant. However, in CHR, lower raw positivity intercept scores (but not lower positivity offset difference scores) were associated with greater mood symptoms. These findings suggest that depressive symptoms may minimally account for the positivity offset deficit in SZ. However, categorical analyses examining affective subgroups (i.e., individuals with schizophrenia versus schizoaffective disorder and individuals at CHR for psychosis with and without comorbid mood disorders) revealed mood-based differences in the positivity offset reduction and associations with negative symptoms (see supplemental materials). The reason for discrepancy between the dimensional and categorical approaches, and which is more valid, is unclear. On the one hand, the dimensional approach to examining correlations with current depressive symptoms would be expected to have high reliability, and the categorical approach may have greater issues with diagnostic reliability and validity; however, the positivity offset was not significantly associated with depressive symptoms in the SZ group. Additionally, the schizophrenia and schizoaffective subgroups differed in cognitive ability and had somewhat different demographic and medication profiles. It is unclear whether these confounding factors are driving the differences observed between categorically defined mood subgroups. Alternatively, the categorical results may reflect an underlying trait disposition toward positivity offset abnormalities in people with a liability for mood pathology regardless of whether they are experiencing a current mood episode. This is supported by the similar pattern of findings across phases of illness, as well as past evidence that the positivity offset is reduced among adults with major depressive disorder (Gollan et al., 2016). Thus, future studies are needed to determine whether depressive symptoms account for positivity offset reductions transdiagnostically and transphasically.

The present findings should be considered in the context of certain limitations. First, the study only measured emotional experience at the subjective level within a controlled laboratory setting. It is unknown whether abnormalities in the positivity offset or negativity bias would also extend to the physiological component of emotional responding or within the context of daily life. Incorporating neurophysiological and ambulatory psychophysiological measures of emotional responding into future studies applying the ESM in these populations may help identify underlying biological abnormalities and real-world behaviors that could inform targets for intervention. Second, the CHR mood-based diagnostic subgroups were small and follow-up replication studies are needed to determine the nature of affective abnormalities and associations with mood symptoms in this population. Lastly, the study was cross-sectional and did not assess how the positivity offset functions over time in the CHR group. Approximately 20% of individuals at CHR for psychosis will develop a psychotic disorder within two years (Salazar de Pablo et al., 2021), and it is unknown whether positivity offset deficits are greater for converters than non-converters. Further, the majority of individuals in our CHR sample will not go on

to develop SZ and are more likely to develop or continue to have a mood disorder. As such, it is likely that insufficient power explains why the positivity offset was intact in both CHR groups.

Findings also have important implications for treatment. Behavioral activation in low arousal contexts may be an effective approach to remediating hedonic and volitional deficits across the psychosis continuum. Further, pairing behavioral activation with emotion regulation strategies (e.g., reappraisal, savoring) may have downstream effects on anhedonia and avolition by increasing both the frequency and intensity of positive emotional experience. This is supported by a recent randomized control trial of the Positive Emotions Program for Schizophrenia (PEPS), a psychosocial treatment designed to enhance positive emotional experience, which found that PEPS was effective at reducing anhedonia and avolition in patients with primary negative symptoms (Favrod et al., 2019a; Favrod et al., 2019b). It is important for future studies to explore the relationship between the positivity offset, psychosocial stressors, emotion regulation, and other potential moderators to understand how the positivity offset reduction is developed and maintained in SZ, associations with core negative symptoms, and how these can be targeted in psychosocial therapy. Such efforts may be paramount for developing effective interventions targeting this abnormality and negative symptoms in psychotic disorders, as well as preventing the progression of these hedonic and volitional deficits in youth at CHR for psychosis.

Additional Information

Supplementary Material

Supplemental material is available at https://osf.io/uzphc/.

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Conflict of Interest

Strauss is one of the original developers of the Brief Negative Symptom Scale (BNSS) and receive royalties and consultation fees from Medavante-ProPhase LLC in connection with commercial use of the BNSS and other professional activities; these fees are donated to the Brain and Behavior Research Foundation. Strauss has received honoraria and travel support from Medavante-ProPhase LLC for training pharmaceutical company raters on the BNSS. In the past 2 years, he has consulted for and/or been on the speaker bureau for Minerva Neurosciences, Acadia, Lundbeck, Sunovion, Boehringer Ingelheim, and Otsuka pharmaceutical companies. All other authors have no relevant disclosures to report.

Ethical approval

Ethical approval provided by the University of Georgia IRB study number Study00004437.

Data Availability

Data is available through the National Institute of Mental Health National Data Archive or by contacting the senior author.

Author CRediT Statement

Bartolomeo: Conceptualization, Formal analysis, Investigation, Data curation, Writing – original draft, Writing – review and editing, Visualization. **Strauss:** Conceptualization, Software, Resources, Writing – review and editing, Supervision, Project administration, Funding acquisition.

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