THE POSITIVITY OFFSET THEORY OF ANHEDONIA ACROSS THE PSYCHOSIS CONTINUUM

by

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(Under the Direction of Greg Strauss)

ABSTRACT

Anhedonia, traditionally defined as a diminished hedonic capacity, has been considered a core component of schizophrenia (SZ) since the earliest conceptualizations of the disorder. Unfortunately, attempts to remediate anhedonia via pharmacological and psychosocial treatments have been ineffective. Limited treatment progress may result in part from poor conceptual clarity and mechanistic understanding of the symptom. Across 3 programmatic manuscripts, this compilation dissertation attempts to address these issues by using conceptual and mathematical models from Cacioppo's Evaluative Space Model to resolve two apparent anhedonia paradoxes, deemed the "liking-wanting" and "schizophrenia-spectrum" paradoxes, as abnormalities in the positivity offset. Manuscript 1 included 44 SZ and 48 healthy controls (CN) who completed 6 days of ecological momentary assessment, which indicated a reduction in the positivity offset in daily life in SZ that was associated with anhedonia and objective digital phenotyping markers of behavioral reduction. Manuscript 2 included two laboratory-based studies conducted in adults with SZ (n = 98) and 84 CN (experiment 1) and youth at clinical high-risk for psychosis (n = 45) and CN (n = 51) (experiment 2) who rated evocative stimuli. Replicating prior results, Manuscript 2 indicated that SZ displayed a reduction in the positivity offset that was associated

with anhedonia; however, this deficit was absent in CHR youth. Manuscript 3 included 100 CHR youth and 57 CN who completed 6 days of EMA, which indicated that CHR had reductions in the positivity offset in daily life that were associated with anhedonia. Across the 3 manuscripts, there was inconsistent evidence regarding whether mood symptoms were associated with positivity offset reductions. Collectively, findings support the notion that reductions in the positivity offset occur across the schizophrenia-spectrum and that these deficits are associated with anhedonia. Reductions in the positivity offset provide a novel explanation for the liking-wanting anhedonia paradox in SZ, and although deficits in the positivity offset were found across phases of psychotic illness, consistent support for the "schizophrenia-spectrum anhedonia paradox" was not observed. Implications for conceptualizing and treating anhedonia as a reduction in the positivity offset are discussed.

INDEX WORDS: psychosis, negative symptoms, emotional experience, hedonic capacity, ecological momentary assessment, prodrome

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DEDICATION

I dedicate this dissertation to my family, friends, mentors, and colleagues who have supported me throughout my graduate training. Above all, I want to thank my dad and sister who have been there for me every step of the way – I could not have done this without you both and am forever grateful for your love and support.

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Schizophrenia (SZ) is a leading medical cause of functional disability worldwide (GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017) and associated with extremely high public health costs (Chong et al., 2016). Negative symptoms are the strongest predictor of functional disability, with anhedonia (i.e., diminished capacity to experience pleasure) and avolition (i.e., diminished interest and engagement in goal-directed behavior) being most critical (Foussias & Remington, 2008; Milev et al., 2005). Unfortunately, pharmacological and psychosocial treatments of negative symptoms have been ineffective at remediating anhedonia and avolition (Fusar-Poli et al., 2015). Limited treatment progress is due in part to a poor mechanistic understanding of anhedonia and avolition in this population.

Minimal progress in treating anhedonia and avolition may result from poor conceptual clarity regarding the nature of these symptoms and the interaction between hedonic and motivational processes in SZ. Evidence for anhedonia and avolition has primarily been derived from retrospective self-reports of emotional experience, motivation, and behavior that individuals with SZ make during clinical interviews. During these interviews, the majority of individuals with SZ are rated as having at least mild severity of these symptoms. Although such data has long been considered irrefutable evidence for diminished hedonic capacity and motivational deficits, the empirical literature provides a more mixed picture. For example, several laboratory-based and ecological momentary assessment (EMA) studies have shown that individuals with SZ demonstrate levels of in-the-moment positive emotion and arousal that are

comparable to psychiatrically healthy controls in response to pleasant stimuli and potentially pleasurable everyday activities (Cohen & Minor, 2010; Gard et al., 2014; Oorschot et al., 2011; Strauss, 2013). Neurophysiological studies using EEG/ERPs and fMRI also generally supported intact neural response to pleasant stimuli or monetary rewards in SZ (Radua et al., 2015; Horan et al., 2010). Although hedonic capacity may be preserved in SZ, it has consistently been demonstrated that normative consummatory pleasure does not translate into motivated approach behaviors. Indeed, consistent with clinical ratings indicating a reduced frequency of recreational and goal-directed activities, ecological momentary assessment (EMA) studies conducted in everyday life indicate that people with SZ engage in fewer pleasurable, goal-oriented, and active behaviors compared to CN (Cho et al., 2017; Granholm et al., 2020; Raugh et al., 2020; Strassnig et a., 2021; Strauss et al, 2022). Evidence for this disjunction between intact hedonic capacity and reduced approach behavior has led the field to address an important question that some have termed the "anhedonia paradox" or the "liking-wanting paradox": why do apparently normal hedonic experiences not produce the typical frequency of approach behaviors in SZ? (Strauss & Cohen, 2008).

Multiple theoretical accounts have been proposed to answer this question and provide insight into the liking-wanting paradox and mechanisms underlying negative symptoms in SZ. For example, Gold et al. (2008) proposed that negative symptoms reflect an impairment in generating, updating, and maintaining mental representations of value needed to guide decision-making processes used to initiate goal-directed activity. Kring, Gard, and colleagues (Gard et al., 2007; Kring & Elis, 2013) have proposed that abnormalities in the temporal dynamics of pleasure restrict the initiation of goal-directed behavior, such that fully intact experiences of positive emotion fail to persist and degrade quickly, subsequently causing greater difficulty with

encoding and retrieving positive experiences, as well as the ability to anticipate future pleasure that leads to reduced initiation of activity. Barch and Dowd (2010) proposed that several additional reward processes that are driven by dysfunctional cortico-striatal circuitry impede the initiation of motivated behavior, such as reinforcement learning, effort-cost computation, value representation, and reward anticipation (Barch & Dowd, 2010; Kring & Barch, 2014; Strauss, 2014; Strauss et al., 2013). While these models have been paramount for improving the field's understanding of negative symptoms, they have not yet led to significant advances in treatment, indicating an incomplete conceptual and mechanistic understanding.

Strauss et al. (2017) proposed that progress in resolving the liking-wanting paradox may be stalled due to a fundamental flaw in the most basic and widely accepted assumptions underlying recent theories on the mechanisms of negative symptoms. Namely, that the notion of hedonic normality, upon which all of the recent theoretical models rest, may be incorrect. Strauss et al. (2017) proposed that the true nature of emotion-motivation interactions in SZ are not fully captured in standard analyses of valence and arousal in response to evocative stimuli or events in daily life, but instead may be detected when more sophisticated conceptual frameworks and mathematical approaches from the field of affective science are adopted in relation to Cacioppo's seminal Evaluative Space Model (ESM) of emotional experience (Cacioppo, 1999; Cacioppo & Berntson, 1994).

The ESM posits that motivation is governed by positive and negative evaluation systems that exist independently within a bivariate space (Cacioppo, 1999; Cacioppo & Berntson, 1994). Each motivational system is characterized by an activation function that translates emotional responses into motivated behavior. In this framework, activation function refers to the relationship between the affective input into the system and the output from that system. Positive

and negative affective systems have evolved to be differentially calibrated in order to adaptively respond in specific environmental contexts. Because unpleasant stimuli have stronger implications for survival than equally intense pleasant stimuli, the affective system is calibrated to respond with incrementally greater levels of negative than positive emotion as the level of emotional input increases (Cacioppo, 1999; Cacioppo & Berntson, 1994; Ito & Cacioppo, 2005; Larsen et al., 2001; Norris et al., 2010), a tendency known as the "negativity bias." Conversely, the positive system is characterized by the "positivity offset," which reflects the tendency of the positive system to respond more strongly than the negative system in the absence of emotional input or when levels of input are weak (Cacioppo, 1999; Cacioppo & Berntson, 1994; Ito & Cacioppo, 2005; Larsen et al., 2001; Norris et al., 2010). As a result of these tendencies, healthy individuals typically experience a greater balance of positive than negative emotion in most situations, which tend to be neutral and characterized by little or no affective input. The positivity offset is adaptive because it promotes exploratory behavior and the approach of novel stimuli in neutral environments, as well as the production of motivated behaviors that are contextually adaptive. Support for the ESM comes from an extensive literature, such as psychophysiological, cognitive, personality, and decision-making studies. Importantly, the positivity offset and negativity bias have repeatedly been found in laboratory-based studies using a variety of stimulus types (e.g., audio, visual) (Ito & Cacioppo, 2005; Larsen et al., 2009; Norris et al., 2011), with neuroimaging studies in healthy controls indicating that the positivity offset is associated with greater activation of the medial prefrontal cortex, nucleus accumbens, and caudate nucleus (Colibazzi et al., 2010; Wager et al., 2003), and the negativity bias with hyperactivation of the amygdala and anterior cingulate cortex (Norris & Cacioppo, 2010; Wager et al., 2003).

Using laboratory-based emotional experience data, which like dozens of prior SZ studies indicated that individuals with SZ did not differ from controls in mean self-reported positive emotion or arousal to pleasant stimuli, Strauss et al. (2017) demonstrated that it was still possible for SZ patients to have a hedonic deficit from the perspective of Cacioppo's ESM. Specifically, SZ had an abnormality in the positivity offset, as evidenced by lower intercept values for positive emotion than controls, suggesting that patients experience diminished positive emotion output at lower levels of stimulus input. SZ also displayed a reduction in the positive - negative intercept difference score, indicating an imbalance in the level of positive to negative emotion at lower levels of affective input, which was associated with greater severity of negative symptoms. At first glance, the diminished positivity offset appears to conflict with the nonsignificant group differences found in self-reported positive emotion to pleasant stimuli that has consistently been observed in the literature and was replicated in this study. However, this discrepancy can be explained by the significantly greater slope values observed for positive emotion in SZ compared to controls. Together, these findings indicate that SZ have lower positive emotion than controls when affective stimulus input is low, but as input increases, patients are able to ramp up their level of positive emotion normally, or even to a greater extent than controls. The more simplistic analysis of how positive patients feel in relation to pleasant stimuli may therefore mask the important observation that a hedonic deficit exists only at lower levels of affective input, but this deficit is overcome when affective input increases. These findings are inconsistent with the notion of diminished hedonic capacity in SZ, which would be indicated by a reduction in positive emotion for stimuli with high affective input (i.e., those that tax maximal affective response). Hedonic abnormalities appear to emerge in SZ only at low levels of affective input. The absence or inversion of the positivity offset may explain why SZ patients often fail to initiate goal-

directed activities in real-life contexts where the majority of time points are neutral and have low levels of affective input (Gard et al., 2007; Gard et al., 2014); however, this has yet to be tested empirically because prior studies have relied exclusively on laboratory-based paradigms. Thus, the most critical tenet of the positivity offset theory of anhedonia has yet to be tested, as more ecologically valid methods are needed to demonstrate a direct link between reductions in the positivity offset and diminished frequency of recreational and goal-directed behavior in SZ. If such a link is supported, this would be consistent with the notion that the positivity offset explains the liking-wanting paradox (i.e., it is not a paradox at all, but rather that reductions in the positivity offset instead of hedonic capacity explain diminished motivated behavior in SZ).

In addition to the liking-wanting paradox, evidence has emerged for a second "anhedonia paradox" over the past decade. Strauss and Cohen (2018) termed this the "schizophrenia-spectrum anhedonia paradox." They reviewed evidence indicating that while the most severe end of the psychosis continuum appears to have intact hedonic capacity (i.e., schizophrenia), the less severe ends of the continuum display genuine reductions in hedonic responsivity. Specifically, individuals with schizotypal personality disorder display reduced emotional responding at the subjective and neurophysiological level to pleasant stimuli compared to controls (Cohen et al., 2011; Cohen et al., 2012; Martin et al., 2020; Najolia et al., 2011). Similarly, laboratory-based studies indicate that relative to controls, individuals at clinical high-risk (CHR) for psychosis (i.e., those with putatively prodromal syndromes who have functional decline and attenuated psychotic symptoms that place them at-risk for schizophrenia) endorse lower levels of positive emotion and display reduced neurophysiological responses to pleasant stimuli (Gruber et al., 2018; Strauss et al., 2018). Since schizophrenia is a more severe form of illness that schizotypal personality disorder or CHR in nearly every imaginable way, this pattern of findings regarding

hedonic response appears to be paradoxical. Why would there be greater deficits in the less severe than more severe ends of the psychosis continuum?

Strauss and Cohen (2018) proposed multiple factors to explain the apparent schizophrenia-spectrum anhedonia paradox. For example, increased frequency of environmental stress and prevalence for mood and anxiety disorders among CHR and schizotypal participants may contribute to greater hedonic deficits at the mild end. Antipsychotics, which are widely prescribed at the severe end of the psychosis spectrum, may normalize hedonic response among individuals with schizophrenia, but not among those at CHR or with schizotypal personality disorder who rarely receive antipsychotics. Cognitive impairment, which is approximately 1.5 SD greater among SZ than CHR or schizotypy (Ettinger et al., 2015; Heinrichs & Kakzanis, 1998; Kwapil & Barrantes-Vidal, 2012; Lam et al., 20108; Seidman et al., 2010; Siddi et al., 2016), may lead those at the severe end of the psychosis continuum to have less insight into their emotional experiences. Better cognition among those at CHR and with schizotypy may lead to more accurate self-reports of hedonic experience. Another possibility, not proposed by Strauss and Cohen (2018), that is an equally plausible explanation for the schizophrenia-spectrum anhedonia paradox is that both mild and severe ends of the psychosis continuum display reductions in the positivity offset that account for clinically rated anhedonia and reductions in the frequency of real-world goal-directed and recreational behaviors. If true, this would suggest that the schizophrenia-spectrum anhedonia paradox is not a paradox at all, and that the nature of emotional experience abnormalities contributing to anhedonia is one in the same throughout the psychosis continuum (i.e., reductions in the positivity offset).

In the current compilation dissertation, three experiments were conducted that are reported in three programmatic studies that use Cacioppo's theoretical and mathematical models

from the ESM to determine whether reductions in the positivity offset account for the liking-wanting and schizophrenia-spectrum anhedonia paradoxes.

Study 1 aimed to determine if the positivity offset accounts for the liking-wanting anhedonia paradox in the real-world by: 1) examining whether the positivity offset reduction in SZ also occurs in daily life measured via active digital phenotyping (i.e., mobile EMA surveys) and 2) determining whether the reduced positivity offset in the daily lives of individuals with SZ is associated with anhedonia and avolition measured via clinical ratings and active and passive (i.e., accelerometry, geolocation) digital phenotyping.

Study 2 included two laboratory-based experiments and aimed to examine the schizophrenia-spectrum anhedonia paradox by: 1) determining whether the positivity offset deficit occurred in a broad sample of outpatients with full-psychotic disorders and youth/young adults at CHR for psychosis who meet diagnostic criteria for an attenuated psychosis syndrome; and 2) determining if the laboratory-based positivity offset is associated with clinically rated anhedonia among adults with SZ-spectrum disorders (i.e., comparing individuals with schizophrenia and schizoaffective disorder to healthy controls) and individuals at CHR for psychosis.

Study 3 further explored the schizophrenia-spectrum anhedonia paradox using one-week of EMA and passive digital phenotyping to determine whether: the positivity offset is reduced in the daily lives of individuals at CHR for psychosis measured via EMA surveys; and 2) whether reductions in the positivity offset exhibited in daily life are associated with anhedonia and avolition measured via clinical ratings, EMA, and passive digital phenotyping.

Furthermore, across all three studies, secondary analyses were conducted to determine whether reductions in the positivity offset: (1) occur in those with and without comorbid mood

disorder diagnoses to examine whether the positivity offset abnormality explains anhedonia only among depressed individuals or also among those experiencing anhedonia and other negative symptoms who are not depressed; (2) are associated with cognition across phases of psychotic illness; (3) appear in those prescribed antipsychotics, as well as those who are not, to determine the role of medication; (4) is associated with cross-sectional conversion risk among individuals at CHR for psychosis.

Collectively, results of these three studies have potential to shed light onto the liking-wanting and schizophrenia-spectrum anhedonia paradoxes and evaluate whether they are paradoxes at all. The positivity offset reduction would at least in part explain these paradoxes if results of the 3 studies indicate that: (1) the diminished positivity offset occurs on laboratory-based paradigms in both attenuated and fully psychotic phases of illness and predicts clinically rated anhedonia; (2) reductions in the positivity offset occur in daily life among individuals with SZ and those at CHR for psychosis and predict reductions in the frequency of positive emotion/recreational activities and goal-directed behavior measured via EMA and passive digital phenotyping. Testing these overarching hypotheses has potential to identify novel mechanistic targets for negative symptoms that are currently being overlooked by studies adopting recent theoretical accounts of negative symptoms that are firmly rooted in the assumption of hedonic normality. If hypotheses are supported, results could suggest that cognitive behavioral interventions could be augmented to focus on increasing the positivity offset in real-world contexts where emotional input is absent or weak (e.g., altering the ratio of positive to negative emotion in neutral contexts using behavioral activation techniques). If positivity offset abnormalities are observed in both laboratory and real-world settings, cognitive and behavioral

interventions could be delivered in real-time over the cell phone via mobile health apps designed specifically for this purpose.

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CHAPTER 2

THE POSITIVITY OFFSET THEORY OF ANHEDONIA IN SCHIZOPHRENIA: EVIDENCE FOR A DEFICIT IN DAILY LIFE USING DIGITAL PHENOTYPING[1]

[1]Bartolomeo, L.A., Raugh, I.M., and Strauss, G.P. 2023. *Psychological Medicine.* 1-9. Reprinted here with permission of the publisher

Abstract

Negative symptoms of schizophrenia have recently been proposed to result from a decoupling of (intact) hedonic experience and (diminished) approach behavior. The current study challenged this view by exploring the hypothesis that negative symptoms are driven by a specific type of emotional experience abnormality, a reduction in the positivity offset (i.e., the tendency to experience greater levels of positive relative to negative emotion in low-arousal contexts), which limits the production of approach behaviors in neutral environments. Participants included outpatients with SZ (n = 44) and healthy controls (CN: n = 48) who completed one week of active (ecological momentary assessment surveys of emotional experience and symptoms) and passive (geolocation, accelerometry) digital phenotyping. Mathematical modeling approaches from Cacioppo's Evaluative Space Model were used to quantify the positivity offset in daily life. Negative symptoms were assessed via standard clinical ratings, as well as active (EMA surveys) and passive (geolocation, accelerometry) digital phenotyping measures. Results indicated that the positivity offset was reduced in SZ and associated with more severe anhedonia and avolition measured via clinical interviews and active and passive digital phenotyping. These findings suggest that current conceptual models of negative symptoms, which assume hedonic normality, may need to be revised to account for reductions in the positivity offset and its connection to diminished motivated behavior. Findings identify key real-world contexts where negative symptoms could be targeted using psychosocial treatments.

Introduction

Deficits in motivation and pleasure have been considered core features of schizophrenia (SZ) since its initial conceptualization (Bleuler, 1911; Diefendorf & Kraepelin, 1907; Kraepelin, 1921). In psychiatrically healthy individuals, these processes are reciprocally connected, with

motivational deficits leading to less frequent pleasurable experiences and hedonic deficits leading to reduced motivation for seeking out these experiences (Bradley & Lang, 2007). In contrast, emotional experience and behavior are decoupled in SZ, such that intact hedonic capacity fails to translate into volitional responding (Heerey & Gold, 2007). To explain this discrepancy, several conceptual models posit that negative symptoms result from dysfunctional cortico-striatal circuitry (Barch & Dowd, 2010; Kring & Barch, 2014; Strauss et al., 2013). Implicit among these models is the assumption that hedonic capacity is intact and deficits in multiple aspects of reward processing (e.g., reinforcement learning, effort-cost computation, value representation) that impact decision-making prevent intact hedonic responses from motivating approach behaviors. Although these models have been vital for our understanding of negative symptoms, they have not led to significant treatment breakthroughs, suggesting that our current mechanistic understanding is incomplete. One limitation of current models may be that the assumption of hedonic normality in SZ is premature, leading to a failure to adequately consider the role of emotional experience abnormalities in negative symptoms.

Caccioppo's seminal Evaluative Space Model (Cacioppo, 1999; Cacioppo & Berntson, 1994) presents a novel approach to understanding how emotional responses fail to generate motivated behavior in SZ. The ESM proposes that separate positive and negative evaluation systems evolved to guide motivated behavior. Both systems are characterized by an activation function representing the relationship between affective input into the system (i.e., arousal) and the resulting output (i.e., emotional response). Positive and negative activation functions are differentially calibrated to translate emotional responses from the affective system into adaptive motivated behaviors in specific contexts. The positive system is calibrated to respond with greater amounts of positive relative to negative emotion at lower levels of affective input, a

function known as the "positivity offset." As affective input into the system increases, the negative system is calibrated to respond with greater levels of negative than positive emotion, a function termed the "negativity bias." (Cacioppo, 1999; Cacioppo & Berntson, 1994; Larsen et al., 2001; Larsen et al., 2009; Norris et al., 2010). Healthy individuals typically experience a greater balance of positive than negative emotion in most situations, which tend to be neutral and characterized by minimal affective input. The positivity offset is therefore adaptive because it promotes exploratory behavior and approach of novel stimuli in neutral environments that allows for the acquisition of new rewards and resources. The negativity bias is adaptive because it leads to withdrawal behavior at high levels of arousal that are characteristic of highly negative or dangerous environments.

Using mathematical approaches validated in the ESM, Strauss et al. (2017) used a laboratory-based paradigm to compare the positivity offset and negativity bias in adults with SZ and controls (CN). Participants made unipolar reports of positivity, negativity, and arousal in response to pleasant, unpleasant, and neutral images from the International Affective Picture System (IAPS) (Lang et al.,1997). Following methods from Ito and Cacioppo (2005), two separate regression equations were used to quantify the parameters used to calculate the positivity offset and negativity bias. The predictor in these equations represents the affective input into the evaluative system (i.e., self-reported arousal). The dependent variable is the resulting output from the affective system (i.e., self-reported levels of positivity or negativity). From these equations, the intercepts and slopes for the positivity and negativity functions were used to calculate the positivity offset and negativity bias, where a greater intercept for positivity relative to negativity reflects the prototypical positivity offset and a greater slope for negativity reflects the negativity bias. Results indicated that although individuals with SZ displayed

hedonic normality, as reported in dozens of other studies (i.e., comparable self-reported positive emotion and arousal to pleasant stimuli between SZ and CN) (Cohen & Minor, 2010; Llerena et al., 2012), emotional experience abnormalities were present in SZ and associated with clinically rated anhedonia. Specifically, individuals with SZ displayed a reduction in the positivity offset compared to CN, as indicated by lower intercept values for positive emotion and reductions in the positive/negative intercept ratio score. Lower ratio scores also predicted higher ratings of anhedonia. Importantly, this positivity offset deficit existed in the presence of intact *hedonic capacity*, as indicated by greater slope for positive emotion in SZ than CN (i.e., at highest levels of arousal when stimuli are most motivationally significant, SZ produce comparably greater positive emotional responses than CN). Riehle et al. (2022) replicated these findings in an online paradigm that was administered to a community sample of individuals varying in trait psychotic-like experiences and anhedonia. Collectively, findings from these two laboratory-based studies provide a novel explanation for how individuals with SZ-spectrum symptoms can have deficits in initiating motivated behavior, despite intact hedonic capacity. However, the link between the diminished positivity offset and reductions in motivated behavior has only been inferred and not yet demonstrated empirically. To fully test the hypothesis that reductions in approach motivated behaviors are associated with reductions in the positivity offset (despite intact hedonic capacity), it will be necessary to examine emotional experience and motivated behavior in ecologically valid contexts during daily life.

The current study used active and passive digital phenotyping to determine whether the positivity offset is reduced in daily life and associated with greater severity of self-reported and objectively quantified measures of anhedonia and avolition. Active digital phenotyping refers to measurements collected via mobile devices in the real-world that are purposefully triggered by

the participant (e.g., surveys) (Onnela & Rauch, 2016). In contrast, passive digital phenotyping involves unobtrusively collecting data via sensors within a mobile device (e.g., geolocation, accelerometry) (Onnela & Rauch, 2016). Preliminary psychometric studies support the reliability and validity of active and passive digital phenotyping measures of negative symptoms in SZ, as well as their feasibility and tolerability (Depp et al., 2019; Fulford et al., 2021; Granholm et al., 2019; Harvey et al., 2021; Miller et al., 2022; Narkhede et al., 2021; Raugh et al., 2020; Raugh et al., 2021; Strauss et al., 2022). When used in tandem, active and passive digital phenotyping methods offer promise for exploring questions regarding the nature of emotion-motivation interactions in SZ since the same computational approaches validated for the ESM can be used in conjunction with objectively measured and self-reported behaviors.

The following hypotheses were made: 1) Based on prior laboratory-based findings (Strauss et al., 2017), participants with SZ will demonstrate a reduced positivity offset compared to CN on measures of active digital phenotyping (EMA surveys) collected in daily life; 2) Hedonic capacity measured via active digital phenotyping will be intact or elevated in SZ based on prior evidence for an increased slope for positivity relative to CN (Strauss et al., 2017); 3) Consistent with findings from Strauss et al., (2017), the negativity bias, measured via active digital phenotyping, will be intact in SZ; 4) Reductions in the active digital phenotyping-derived positivity offset difference score, but not the negativity bias, will be significantly associated with anhedonia and avolition measured via clinical rating scales, active digital phenotyping measures of negative symptoms in daily life, active digital phenotyping measures of the frequency of positive emotional experiences, and passive digital phenotyping measures of behavior obtained via geolocation and accelerometry.

Method

Participants

Forty-six individuals with DSM-5 (American Psychiatric Association, 2013) diagnoses of schizophrenia or schizoaffective disorder (SZ) and 50 psychiatrically healthy controls (CN) participated in the study. Two SZ and 2 CN participants were excluded for not reaching a priori digital phenotyping compliance standards (i.e., responding to < 20% of momentary surveys), resulting in a final sample of 44 SZ (16 with schizophrenia and 28 with schizoaffective disorder) and 48 CN. Groups did not significantly differ on age, sex, ethnicity, or parental education. SZ had lower personal education than CN. Moderately severe symptoms and a typical magnitude of cognitive impairment were observed in SZ (see Table 1).

Individuals with SZ were recruited from local community outpatient mental health centers and advertisements. Clinical diagnoses were determined via the SCID-5 (First, 2015). CN were recruited from the local community using advertisements. CN were free of current major psychiatric diagnoses (i.e., mood and anxiety disorders) as established via the SCID-5, current SZ-spectrum personality disorders as established via the SCID-PD (First, Williams, Benjamin, & Spitzer, 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 (excluding nicotine use disorders). All participants received monetary compensation for their participation and provided written informed consent for a protocol approved by the University of Georgia Institutional Review Board. Participants were compensated $20 per hour for laboratory sessions, $1 per mobile survey completed, and an $80 bonus for returning the phone at the end of the study.

Procedure

The study consisted of three phases: 1) initial laboratory visit; 2) six consecutive days of digital phenotyping; and 3) final laboratory visit.

Phase 1: Initial laboratory visit

Clinical interviews were conducted to assess diagnoses and symptoms. Diagnostic and symptom interviews for SZ consisted of the SCID-5 (First, 2015) and Brief Negative Symptom Scale (BNSS; Kirkpatrick et al., 2011). CN interviews included the SCID-5 (First, 2015), and SCID-PD (First et al., 2015). All interviews were conducted by either Dr. Strauss or lab personnel trained to reliability standards (inter-rater reliability of alpha > .80) who established consensus for diagnoses.

Participants also received training on digital phenotyping procedures and were provided with a Blu Vivo 5R smartphone running Android operating system 7.0 programmed with the mEMA app from ilumivu to collect digital phenotyping data. Trained lab personnel instructed participants in the use of the phone and the mobile app, including a guided demonstration of survey notifications and completion of a practice survey, which provided an overview and explanation of the types of questions that would be asked. Participants were also training on how to use and charge the Empatica wristband.

Phase 2: Digital Phenotyping

Active Digital Phenotyping. Over the 6-day digital phenotyping phase, participants were prompted with eight momentary surveys per day that were quasi-randomly scheduled within 90-minute epochs between 9 AM and 9 PM. Surveys were scheduled between 18 minutes to 3 hours apart from each other. Attempts to respond to the survey after a 15-min window were not

permitted, but participants were allotted unlimited time to complete the questions. Surveys assessed the following:

Momentary Emotional Experience. Every survey probed current levels of positive and negative emotion using the modified Differential Emotions Scale (mDES; Fredrickson, Tugade, Waugh, & Larkin, 2003). Each prompt assessed five negative (anger, fear, sadness, shame, anxiety) and five positive emotions (amused, content, happy, love, pride) rated on a 0-100 sliding scale anchored between "Not at all" and "Extremely." Participants also identified whether their current emotional context was positive, negative, neutral, or mixed. Context responses were used to determine the frequency of positive experiences endorsed over the digital phenotyping period.

Momentary Emotional Arousal. Every survey probed current emotional arousal by asking "How keyed-up or excited are you right now?". Participants rated arousal on a 0-100 sliding scale anchored between "Not at all" and "Extremely."

Negative Symptoms. Momentary surveys probed for negative symptoms of anhedonia, avolition, and asociality. Anhedonia was measured by averaging across momentary responses for consummatory (i.e., "How much are you enjoying the activity?" and "How much are you enjoying this social interaction?") and anticipatory pleasure (i.e., "How much do you think you will enjoy that activity the next time you do it?" and "How much do you think you will enjoy interacting with them next time?"). Avolition was measured by assessing participants' level of interest in a current activity (i.e., "How interested are you in the activity?"). If participants reported they were not engaged in an activity (i.e., doing "Nothing."), avolition was measured via desire to engage in an activity (i.e., "How much do you want to be doing an activity right now?"). Lastly, asociality was measured by assessing participants' interest in

a social interaction (i.e., “How interested are you in this social interaction?”). If participants denied interacting with anyone, asociality was measured via responses about their desire to interact with others (i.e., “How much do you want to be interacting with someone right now?”). All items were rated on a 0 (not at all) to 100 (extremely) scale. These items have shown convergent validity via associations with clinical ratings of the same domains on the BNSS; confirmatory factor analysis also indicates that the items constitute 3 separate factors: anhedonia, avolition, asociality (Raugh et al., 2020).

Infrequent Responding. To monitor infrequent responding, a question from the Chapman Anhedonia Scale (Eckblad et al., 1982) was embedded within each momentary survey. Participants responded “True” or “False” to the items, which portrayed common, every day experiences (e.g., “Sometimes when walking down the sidewalk, I have seen children playing.”; “I cannot remember a time when I talked with someone who wore glasses.”). The rate of infrequent responding was low (< 7%) in both groups.

Passive Digital Phenotyping. Geolocation was passively measured throughout the digital phenotyping phase via the smartphone. Geolocation involves collecting GPS coordinates at predetermined intervals or every time the participant moves a certain radius in a space. Phone sensors were programmed to collect geolocation every 10 minutes or when participants moved more than 10 meters. To index change in geolocation, distance from home and percentage of time at home were extracted. Distance from home (m) was calculated by the Haversine formula (Sinnott, 1984). Secondary geolocation variables were calculated for exploratory purposes (e.g., number of flights, transition time) that were previously validated in relation to negative symptoms (Granholm et al., 2019; Raugh et al., 2020).

Using the mEMA application, sensors within the study phones were programmed to measure accelerometry with each change in XYZ coordinate motion (every change in accelerometry being logged as a single instance), with separate values output for X, Y, and Z movement axes. Participants wore an Empatica wristband that collected accelerometry as a gravitational force (g units) at 32 Hz between -16 g and 16 g. Accelerometry data was transferred to the phone via Bluetooth connection or stored on the band for up to 14 hours if a connection was not available. Passive digital phenotyping data stored on the Ilumivu or Empatica servers was encrypted and de-identified until downloaded by research staff. Accelerometry has shown convergent validity with clinically rated negative symptoms (Strauss et al., 2022). See Table 2 for information on geolocation and acceleration variables.

Phase 3: Final laboratory visit

The final laboratory visit occurred one week after the initial laboratory visit, at the end of the digital phenotyping protocol. Participants returned the phone to the lab, completed the MATRICS Consensus Cognitive Battery (MCCB; Nuechterlein et al., 2008), and other procedures not reported in the current study.

Data Analysis

All analyses were conducted using SPSS v.27 except for multi-level supplemental analyses performed in R. Methods for calculating the positivity offset and negativity bias are based on Ito and Cacioppo (2005). The positivity offset and negativity bias are characterized by regression parameters where the positivity offset is represented as the intercept for positivity (the output at zero input) and the negativity bias represented as the slope for negativity (greater rate of change in output per unit of input). Positivity offset and negativity bias were assessed across participants within each group. Two regression analyses were conducted for each subject using

the equation $E = Ax + b$, where E is either unipolar positivity or negativity ratings and A is the mean arousal rating. To model the positive motivational system, the intercept value derived from the equation represents the strength of the positivity offset, where positivity offset scores (i.e., the positive - negative intercept difference score) were calculated from multiple regression conducted on each participant and used to obtain the intercept score for positive and negative emotion for each day. To model the negative motivational system, the slope derived from the equation represents the strength of the negativity bias, which reflects the magnitude of increase in negative emotion output per unit of increase in affective input. The negativity bias difference score was calculated as the difference between the negativity and positivity slopes (i.e., negativity slope – positivity slope). The average intercept and slope difference scores were then calculated across all days and summarized at the level of the week to obtain the most reliable estimate. These difference scores then served as the dependent variables for primary analyses used to examine group differences and correlations.

Preliminary analyses of standard comparisons of valence and arousal, including effects of emotional context, were conducted as described in Strauss et al. (2017) and displayed in Supplemental Materials. For primary analyses, within-group paired sample t-tests were conducted comparing positive and negative intercepts and slopes to determine if each group demonstrated the prototypical positivity offset and negativity bias. Separate one-way ANOVAs were conducted on the 1-week average positivity offset intercept difference score, the negativity bias difference score, and the raw positivity and negativity intercept and slopes to assess group differences in positivity and negativity parameters. Raw intercept comparisons reflect group differences in how the positivity and negativity systems are calibrated to respond when affective input is absent, and the slope comparisons group differences in hedonic capacity. Pearson

correlations were used to examine the relationship between positivity and negativity parameters with accelerometry and geolocation as measures of avolition in the SZ group (see Table 2 for digital phenotyping variables included in correlational analyses), as well as avolition and anhedonia measured via the BNSS and digital phenotyping. Additionally, the association between the number of positive experiences participants endorsed over the digital phenotyping period and the positivity offset difference score was examined using bivariate correlations.

Additional exploratory analyses examining the effects of sex, diagnosis (i.e., schizoaffective disorder versus schizophrenia), and associations with medication status and cognition are reported in Supplemental Materials.

Results

Both SZ ($t = 2.13$, $p = .04$) and CN ($t = 9.61$, $p < .001$) demonstrated the positivity offset, with significantly higher intercepts for positivity than negativity. Slopes for the positivity function did not significantly differ between groups. As hypothesized, the positivity offset intercept difference score was significantly reduced in SZ compared to CN ($F(1, 92) = 10.86$, $p = .001$, $\eta_p^2 = .11$). Neither group demonstrated the negativity bias, evidenced by nonsignificant differences between the slope for positivity and negativity in SZ ($t = -1.69$, $p = .10$) and CN ($t = .52$, $p = .61$). Groups did not significantly differ on the negativity bias slope difference score (see Table 3).

In SZ, greater reductions in positivity offset were associated with reduced vigor of movement (i.e., ACLB.mean) ($r = .53$, $p = .02$) and greater variability in movement (i.e., ACLB.sd) ($r = -.52$, $p = .02$) measured by the wristband (see Table 2 for digital phenotyping variable definitions). Greater variability in movement is more common among sedentary compared to active individuals, with the latter demonstrating steady fluctuations in movement

and speed throughout the day. Past research suggests that individuals with SZ spend more time in sedentary than active contexts compared to CN (Strassnig et al., 2021), suggesting that this finding reflects the relationship between deficient activity and approach motivation in daily life. Lower positivity offset scores were associated with more severe avolition and anhedonia measured via the BNSS (avolition: r = -.34, p =.03; anhedonia: r = -.43, p < .01) and active digital phenotyping (avolition: r = -.57, p < .001; anhedonia: r = -.58, p < .001) in SZ. Lastly, grater reductions in the positivity offset were associated with a lower frequency of positive events in SZ (r = 0.34, p = 0.03). Correlations between the negativity bias score and clinically rated and active and passive digital phenotyping measures of avolition and anhedonia were all nonsignificant (p's all > .05).

In SZ, lower raw positivity intercepts were significantly associated with more severe anhedonia (r = -.36, p = .02) and avolition (r = -.32, p = .03) measured via the BNSS and active digital phenotyping (anhedonia: r = -.63, p < .001; avolition: r = -.57, p < .001). Greater reductions in the positivity slope in SZ were significantly correlated with more severe clinically-rated anhedonia (r = -.36, p = .02). In SZ, higher intercepts for the negativity function were associated with more severe anhedonia (r = .34, p = .02) and avolition (r = .41, p = .01) measured via active digital phenotyping, as well as greater variability in movement measured by the wristband (i.e., ACLB.sd) (r = .54, p = .01).

Discussion

The current study aimed to determine if the positivity offset was reduced in the daily lives of people with SZ and associated with negative symptoms measured via clinical interviews and digital phenotyping. Consistent with several past laboratory-based and experience sampling studies, digital phenotyping results indicated that hedonic capacity is intact in SZ across positive,

negative, and neutral contexts (Cohen & Minor, 2010; Gard & Kring, 2009; Gard et al., 2007; Gold et al., 2008; Kring & Moran, 2008). Conversely, emotional experience abnormalities emerged when the ESM was applied. Specifically, the positivity offset measured via active digital phenotyping was significantly reduced in SZ compared to CN, suggesting that patients experience reduced levels of positive relative to negative emotion at low levels of arousal. The nonsignificant group differences in the slope for the positivity function indicates that patients' positive emotions increase as arousal increases, suggesting that hedonic capacity is intact in SZ and hedonic deficits are only present when affective input is low. This extends laboratory-based evidence for the positivity offset reduction in SZ (Strauss et al., 2017) by demonstrating that it also occurs in the context of daily life, outside of a controlled laboratory setting with controlled emotional stimuli. Also extending past findings (Strauss et al., 2017), greater reductions in the positivity offset were associated with increased avolition and anhedonia measured via the BNSS and active and passive digital phenotyping. The reduced positivity offset was also associated with the frequency of positive experiences measured via the active digital phenotyping emotion context item (i.e., the behavioral component of anhedonia). Together, these findings extend past laboratory-based studies (Strauss et al., 2017; Riehle et al., 2022), providing a direct link between the positivity offset reduction and real-world behavior. The association between the positivity offset reduction and digital phenotyping measures of negative symptoms indicate that it may be a relevant target for improving deficits in approach behavior in SZ. Further, results indicated that the negativity bias is intact in SZ during everyday activities and unrelated to any measures of avolition or anhedonia. These findings were also consistent with prior laboratory evidence (Strauss et al., 2017), suggesting that within the ESM, negative symptoms were associated with the positivity offset rather than the negativity bias.

The current findings should be considered in the context of certain limitations. First, the positivity offset and negativity bias were calculated based on subjective reports of arousal and emotion. Future laboratory-based and digital phenotyping studies should incorporate physiological measures of arousal and emotional responding (e.g., heart rate variability, skin conductance, pupil dilation) to further understand abnormalities within the context of the ESM. Second, the accelerometry and geolocation variables included in the study belong to the "third generation" of negative symptom assessments that are still being validated. Additional work is needed to extend findings from preliminary validation studies (Narkhede et al., 2021; Raugh et al., 2020; Strauss et al., 2022) and identify which are the strongest, most reliable measures of negativity symptoms. Third, the current sample included adult outpatients with chronic, stable SZ. Thus, it is unclear if the results would extend to earlier stages of illness and those with greater symptom severity. Finally, a clinical comparison group was not included. Past laboratory-based behavioral findings indicate that the positivity offset is reduced in individuals with depression (Gollan et al., 2016), who also commonly experience avolition and anhedonia. This may suggest that the positivity offset is not specific to SZ and could be a transdiagnostic mechanism underlying avolition and anhedonia.

Findings have important clinical implications. Behavioral interventions, such as behavioral activation and activity scheduling, may be effective at targeting positivity offset abnormalities and improving avolition or anhedonia. These intervention tactics are main components of Negative Symptom Focused Cognitive Behavior Therapy (Perivoliotis et al., 2010), and past studies have shown that they are feasible and effective at reducing negative symptoms in SZ (Choi et al., 2016; Grant et al., 2012; Lee et al., 2018; Mairs et al., 2011); however, to our knowledge, no study to date has examined the relationship between behavioral

activation, the positivity offset, and deficits in motivation and pleasure in SZ. Mobile health (mHealth) interventions may provide an alternative way to target real-world impairments in hedonic and motivational processes from the perspective of the ESM. For example, mHealth apps could be programmed to assist in activity scheduling, including sending reminders for activities, and to notify patients to become behaviorally activated in neutral contexts in a way that will provide opportunities to increase positive affect and decrease negative affect. Passive digital phenotyping could be directly incorporated into treatment, such as sending notifications to become behaviorally activated when objective behavioral markers (e.g., speed of movement, activity index) fall below a relevant threshold. Lastly, emotion regulation interventions delivered via in-person therapy and/or mHealth may also help patients to more effectively increase positive emotion and decrease negative emotion as a means of normalizing the positivity offset to facilitate motivated behavior. Practicing emotion regulation strategies such as savoring and reappraisal may be particularly beneficial for increasing positive emotion and decreasing negative emotion, respectively (Favrod et al., 2019).

In conclusion, the present findings support the hypothesis that the diminished positivity offset is associated with negative symptoms in SZ. These results refute prior assumptions of hedonic normality and affect-behavior decoupling. Instead, deficits in motivated behavior appear to be driven by an imbalance in positive relative to negative affect in low arousal contexts. The specificity of when affective abnormalities drive motivated behavior deficits, at low levels of arousal, should be used to personalize novel treatment approaches to everyday contexts where avolition and anhedonia are most relevant. Pending replication and extension, conceptual models of negative symptoms should incorporate affective abnormalities like the positivity offset

reduction, in addition to dysfunction in other aspects of reward processing, as an important process leading to negative symptoms.

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Table 2.1

Participant Demographic and Clinical Characteristics

	SZ (n=44)	CN (n=48)	Test statistic	p-value
Age	39.34 (12.02)	38.56 (10.50)	$F = 0.11$	.74
Parental Education	13.98 (2.83)	13.51 (2.86)	$F = 0.58$	0.40
Personal Education	13.02 (2.27)	15.73 (2.82)	$F = 25.46$	< 0.001
Female (%)	63.60	72.90	$\chi^2 = 0.92$	0.34
Race (%)			$\chi^2 = 7.19$	0.21
Black	29.50	22.90		
Asian	0.00	6.30		
Latinx	4.50	12.50		
White	59.10	47.90		
Multiracial	6.80	6.30		
Other	0.00	4.20		
MCCB	41.91 (15.74)	52.40 (10.19)	$F = 13.90$	< 0.001
BNSS Total	16.23 (13.66)	-		
BNSS Avolition	2.10 (1.68)	-		
BNSS Anhedonia	1.51 (1.51)	-		
BNSS Asociality	1.50 (1.31)	-		
BNSS Alogia	0.30 (.91)	-		
BNSS Blunted Affect	1.16 (1.52)	-		

Note. SZ=schizophrenia group; CN=control group. MCCB = MATRICS Consensus Cognitive Battery. BNSS = Brief Negative Symptom Scale. BNSS domain scores reflect average of item scores within each domain.

Table 2.2

Geolocation and Accelerometry Variable Definitions

GPS or ACL	Variable	Abbreviation	Definition
GPS	Home time	Home	Amount of time at home
GPS	Distance change	Δd MPD Range	Distance traveled in meters from previous sample Total meters traveled per day Range of distance per day
GPS	Distance from home	Δmh maxmh	Meters from home for each sample Maximum distance from home per day
GPS	Stationary location clusters	nc	Number of meaningful locations
GPS	Location variance	lv	Variance within a specified timeframe: lv = log((σ2 lat + 1)+(σ2 long + 1))
GPS	Entropy and normalized entropy	ent nent	Equity of time spent in different locations
GPS	Transition time	tt	Percentage of samples taken in transit
GPS	Flights	f.dur f.dist f.num	Average duration of discrete trips per day Average distance of discrete trips per day Average number of discrete trips per day
ACL	ACL mean	ACLB.mean	Accelerometry band mean
ACL	ACL variance	ACLB.SD	Accelerometry band average standard deviation

Note. GPS = geolocation; ACL = accelerometry.

Table 2.3

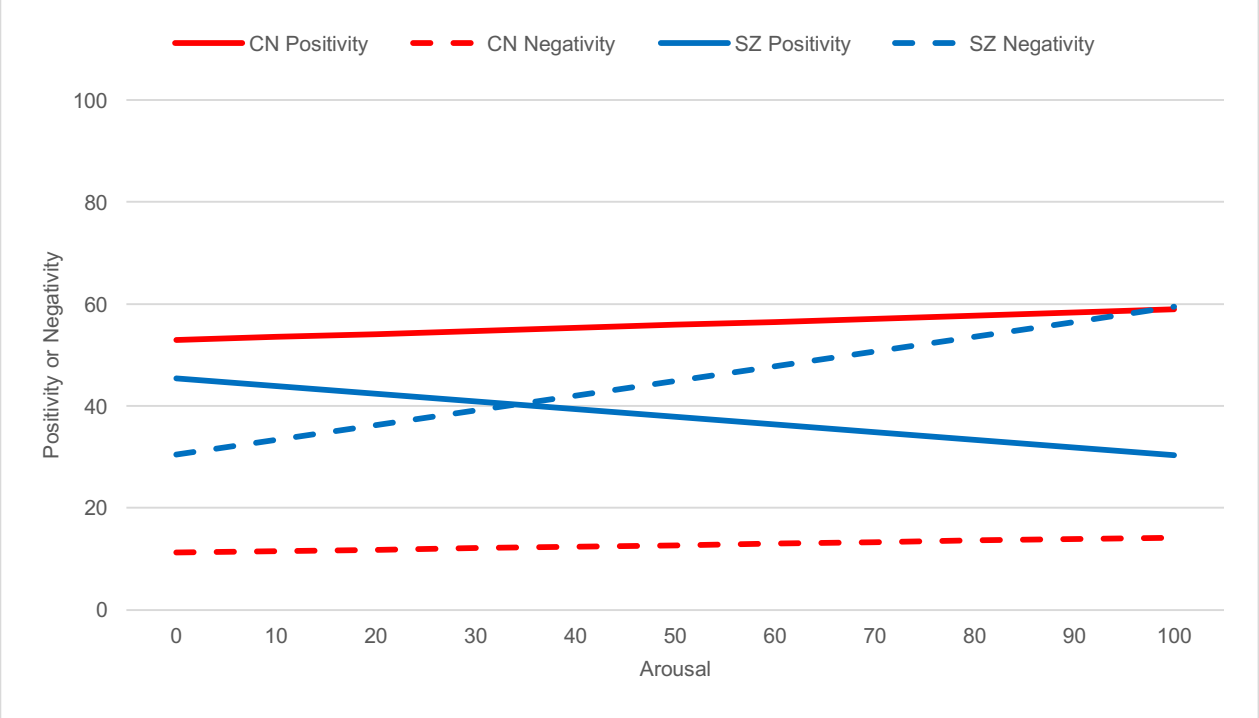
One-way ANOVAs Comparing Positivity and Negativity Parameters in Schizophrenia and Control Groups

	SZ	CN	Test Statistic
Positivity Intercept	45.38 (24.86)	52.91 (21.06)	$F(1, 92) = 2.47$, $p = .12$, $\eta_p^2 = .03$
Negativity Intercept	30.41 (30.25)	11.19 (12.60)	$F(1, 92) = 16.30$, $p < .001$, $\eta_p^2 = .15$
Positivity Slope	-.15 (1.06)	.06 (.26)	$F(1, 92) = 1.72$, $p = .19$, $\eta_p^2 = .02$
Negativity Slope	.29 (1.38)	.03 (.19)	$F(1, 92) = 1.73$, $p = .19$, $\eta_p^2 = .02$
Positivity Offset Difference Score	14.97 (46.66)	41.72 (30.07)	$F(1, 92) = 10.86$, $p = .001$, $\eta_p^2 = .11$
Negativity Bias Difference Score	.44 (1.73)	-.03 (.40)	$F(1, 92) = 3.37$, $p = .07$, $\eta_p^2 = .04$

Note. SZ = Schizophrenia 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 2.1

Positivity Offset and Negativity Bias Functions



Note. CN = Control group, SZ = Schizophrenia group.

CHAPTER 3

UNPACKING THE ANHEDONIA PARADOX ACROSS THE PSYCHOSIS CONTINUUM: THE ROLE OF THE POSITIVITY OFFSET[1]

[1]Bartolomeo, L.A., and Strauss, G.P. 2023. Accepted by *Journal of Emotion and Psychopathology,* 5/7/23.

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.

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. 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 1).

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. 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 E is either the positive or negative subjective emotional response rating to either pleasant or unpleasant stimuli viewed during the emotion task, and A is the mean arousal rating for either neutral and unpleasant stimuli or neutral and pleasant sitmuli. For the positivity function, the resulting intercept represents the positivity offset (i.e., the level of positive emotion when affective input is absent). For the negativity function, the resulting slope 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. 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. 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.

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 1).

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. Exploratory correlations between positivity and negativity parameters with cognition are described in the Supplemental Materials. 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) 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) 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), 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), 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), but no other variables were associated with depression severity.

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. 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).

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.

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Table 3.1

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. *$p < .05$, **$p < .01$, ***$p < .001$.

Table 3.2

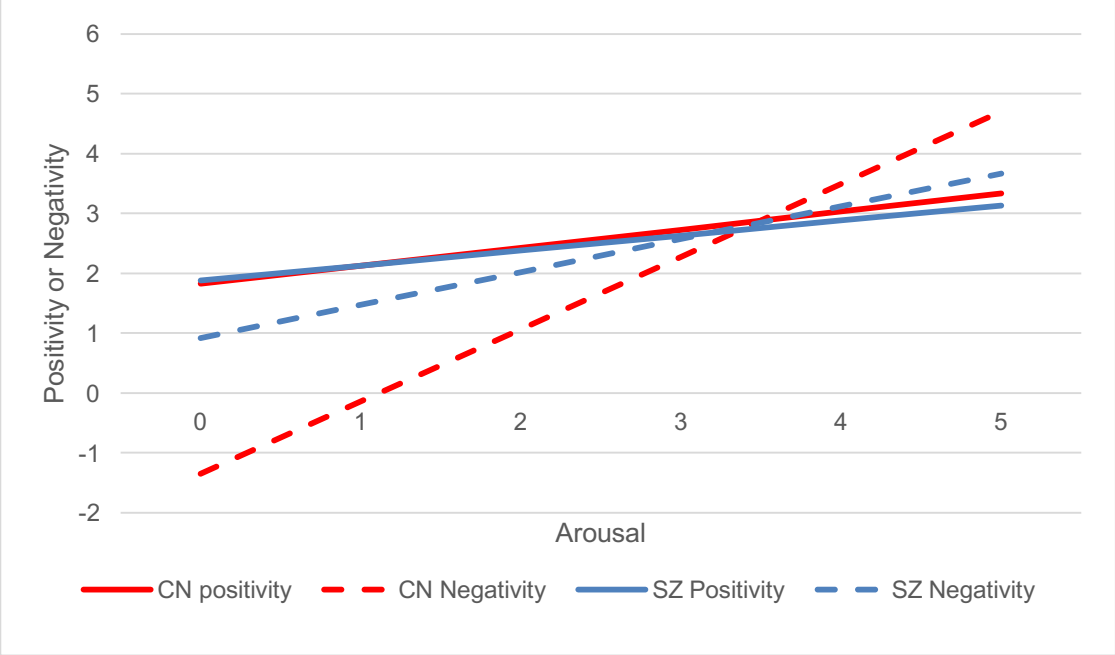
One-way ANOVA Results Comparing Positivity and Negativity Parameters in Clinical and Control Groups

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

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 3.1

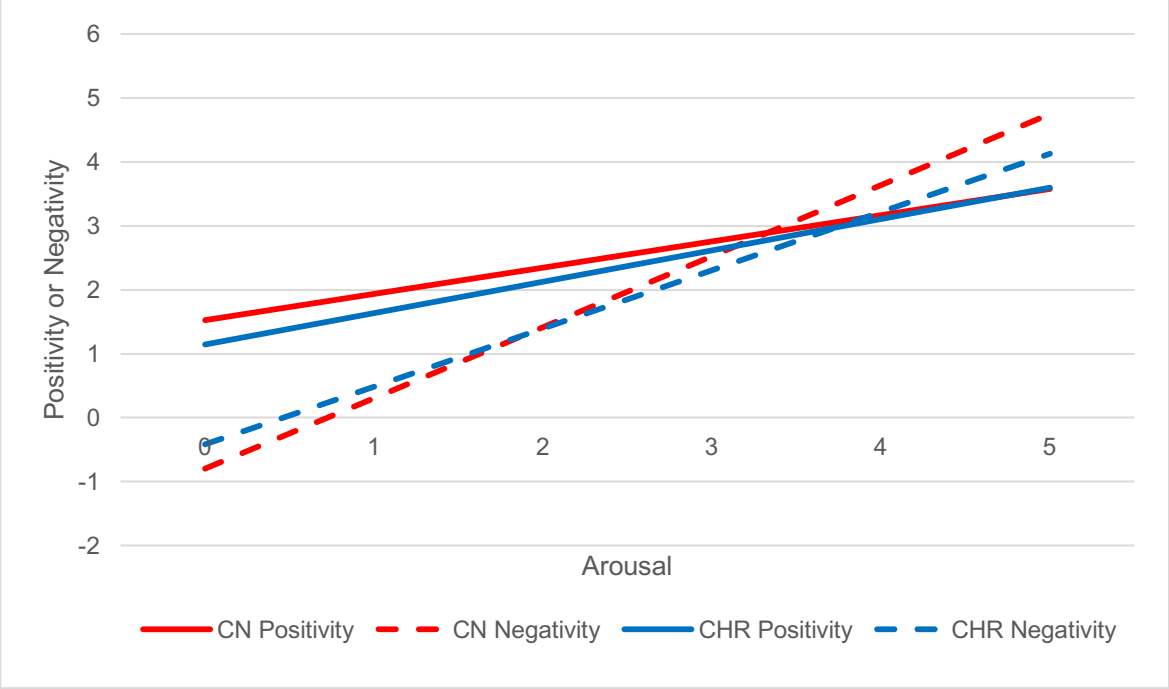
Positivity Offset and Negativity Bias Functions in Psychosis and Control Groups



Note. SZ = schizophrenia group; CN = control group.

Figure 3.2

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



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

CHAPTER 4

DIGITAL PHENOTYPING EVIDENCE FOR THE REDUCED POSITIVITY OFFSET AS A MECHANISM UNDERLYING ANHEDONIA AMONG INDIVIDUALS AT CLINICAL HIGH-RISK FOR PSYCHOSIS[1]

[1]Bartolomeo, L.A., James, S.H., Berglund, A.M., Raugh, I.M., Mittal, V.A., Walker, E.F., and Strauss, G.P. Submitted to *Clinical Psychological Science,* 3/22/23.

Abstract

The current study examined the novel hypothesis that anhedonia stems from a diminished positivity offset among individuals at clinical high-risk for psychosis (CHR) (i.e., a reduction in the normative tendency to experience greater positive than negative affect during low arousal neutral contexts that promote reward seeking behavior). Mathematical modeling approaches from Cacioppo's Evaluative Space Model were applied to six days of digital phenotyping data collected in daily life in100 individuals at CHR and 57 healthy controls. Results indicated that individuals at CHR demonstrated a diminished positivity offset that was associated with clinically-rated anhedonia. Findings clarify the nature of hedonic deficits among those at CHR and identify novel treatment targets.

Introduction

Negative symptoms, including deficits in motivation and pleasure, are highly prevalent among individuals at clinical high-risk (CHR) for psychosis and a key factor that leads individuals and their families to first seek contact with the medical system (Lencz et al., 2004; Velthorst et al., 2009; Yung & McGorry, 1996; Yung et al., 2003). Negative symptoms are also associated with greater conversion risk (Kwapil, 1998; Mason et al., 2004; Piskulic et al., 2012; Velthorst et al., 2009; Yung et al., 2005) and impaired social and role functioning among individuals at CHR, suggesting that they are a critical target for early identification and prevention of psychotic disorders (Carrión et al., 2016; Cornblatt et al., 2007; Devoe et al., 2021; Glenthøj et al., 2017; Schlosser et al., 2015). Unfortunately, current pharmacological and psychosocial interventions are relatively ineffective at reducing negative symptoms among individuals at CHR for psychosis (Devoe et al., 2017). Lack of treatment progress may reflect limited understanding of the mechanisms underlying negative symptoms in CHR.

Recent theoretical models of the mechanisms underlying negative symptoms in schizophrenia (SZ) implicate disruptions in various aspects of reward processing (e.g., reinforcement learning, effort-cost computation, value representation) that prevent intact hedonic responses from influencing decision-making processes needed to initiate goal-directed behaviors (Barch & Dowd, 2010; Gold et al., 2006; Strauss et al., 2014). However, accumulating evidence suggests that these models, which assume hedonic normality, should be updated because hedonic abnormalities are present in SZ and predictive of motivational deficits when emotional experience is viewed in relation to sophisticated theoretical and computational models from the field of affective science (Strauss et al., 2017). Specifically, Cacioppo's Evaluative Space Model (ESM) (Cacioppo, 1999; Cacioppo & Berntson, 1994; Ito & Cacioppo, 2005; Norris et al., 2010) has shown particular promise for understanding how hedonic abnormalities contribute to reductions in goal-directed activity in SZ.

The ESM proposes that two motivational systems, approach and withdrawal, are governed by activation functions that evolved to translate positive and negative emotional responses into motivated behavior (Ito & Cacioppo, 2005). Under absent or low levels of affective input (i.e., emotional arousal) into the system, the activation function is designed to respond with disproportionately greater levels of positive than negative emotion. This tendency, termed the "positivity offset," is adaptive because it promotes exploratory and approach behaviors that allow new rewards and resources to be acquired in relatively safe or neutral contexts. In contrast, the activation function responds with disproportionately greater levels of negative than positive emotion as affective input into the system increases. This tendency, termed the "negativity bias," is adaptive because it promotes withdrawal behaviors in highly negative, aversive contexts when threat to survival are highest. Thus, among healthy individuals,

the motivational system is predisposed to generate rapid emotional responses that most efficiently promote survival under high and low arousal contexts.

Across a series of studies, Strauss, Bartolomeo, and colleagues have applied mathematical approaches from Cacioppo's ESM to evaluate the hypothesis that anhedonia reflects a reduction in the positivity offset among individuals with SZ-spectrum disorders. In the first study, Strauss et al. (2017) conducted a laboratory-based study where outpatients with SZ and healthy controls (CN) provided self-reports of positive emotion, negative emotion, and arousal to complex emotional and neutral images. Compared to healthy controls, individuals with SZ demonstrated a diminished positivity offset that predicted greater clinically rated anhedonia. Importantly, these deficits existed in the context of intact hedonic capacity, as indicated by traditional self-reports of positive emotion to pleasant stimuli and the positivity slope derived from ESM equations. In the second study, Bartolomeo et al. (under review) replicated the findings of Strauss et al. (2017) in a large independent sample of outpatients with full psychotic disorders and extended them by demonstrating that the positivity offset reduction and its associations with anhedonia were greatest among those with mood disorders (i.e., schizoaffective disorder). The study also administered the same laboratory-based paradigm to those at CHR for psychosis, finding an intact positivity offset that was not significantly associated with anhedonia; however, among individuals at CHR for psychosis with a mood disorder diagnosis, greater reductions in the positivity offset were associated with more severe anhedonia. Further, individuals at CHR for psychosis without a mood disorder demonstrated a significant association between lower hedonic capacity measured by the slope for the positivity function and more severe anhedonia (i.e., anhedonia may reflect a true deficit in hedonic capacity in CHR, unlike SZ). In a third study, Bartolomeo et al. (in press) critically extended these laboratory-based

findings using ecological momentary assessment (EMA) and passive digital phenotyping. Outpatients with SZ and CN completed 1-week of EMA in the context of daily life. Participants provided self-reports of positive emotion, negative emotion, arousal, context (location, activity type, social interaction), and symptoms while accelerometry was passively recorded via a wristband and internal sensors of the phone. Similar to laboratory-based studies, the SZ group displayed a reduction in the positivity offset as measured via EMA surveys. Reductions in the positivity offset were also significantly associated with clinical ratings of anhedonia, EMA self-reported anhedonia and avolition, EMA behavioral markers of anhedonia (positive emotion context frequency), and objective markers of motivated behavior obtained via accelerometry (vigor and variability of movement). These EMA findings provide a critical link between the positivity offset reduction and real-world measures of motivated behavior. Finally, in a fourth study, Riehle et al. (2022), administered a laboratory-based self-report paradigm to a community sample with varying sub-clinical levels of anhedonia and psychotic-like experiences. Results also supported a link between anhedonia and reductions in the positivity offset among those with attenuated symptoms. Thus, across four studies to date, results provide support for a link between the positivity offset reduction and anhedonia in laboratory-based paradigms and more ecologically valid EMA measures acquired in daily life in full psychotic disorders; however, findings are inconsistent among individuals at less severe ends of the psychosis continuum (i.e., individuals at CHR for psychosis and with psychotic-like experiences).

Although these prior studies support the positivity offset theory of anhedonia (Strauss et al., 2017), there are several important questions yet to be fully addressed: (1) Does the positivity offset theory apply only to the most severe end of the psychosis continuum (i.e., schizophrenia, schizoaffective disorder), or do reductions exist in attenuated forms of the illness, such as CHR?

Prior studies provide evidence for positivity offset reductions in those with anhedonia who report non-clinical psychotic like experiences (Riehle et al., 2022), but not the attenuated psychosis syndrome (i.e., CHR) (Bartolomeo et al., in press); however, sample sizes have been modest and replication and extension is needed; (2) Are the positivity offset deficits confined to laboratory-based paradigms, or do they also occur in the real-world and predict reductions in approach behavior? Prior EMA findings indicated that reductions in the positivity offset occurred in daily life and predicted anhedonia, as well as greater deficits in more objective measures of motivated behavior (Bartolomeo et al., under review); however, such findings have yet to be replicated or extended to those at CHR for psychosis; (3) Is the positivity offset primarily driven by mood symptoms across the schizophrenia-spectrum, or does it also occur in those without mood symptoms? Our prior results provide mixed support for the role of mood symptoms in the link between the positivity offset and anhedonia (Strauss et al., 2017; Bartolomeo et al., in press). Given the high comorbidity of mood disorders in those with prodromal syndromes (Addington et al., 2021), the CHR population is an ideal sample to examine whether the diminished positivity offset and its association with anhedonia are driven by depression.

To address these questions and extend the literature on anhedonia in the schizophrenia-spectrum, the current study collected one-week of EMA and passive digital phenotyping data in a large sample of CHR and CN participants. The following hypotheses were evaluated: 1) CHR participants would demonstrate a reduced positivity offset compared to healthy controls (CN) on EMA surveys; 2) Among those at CHR, reductions in the positivity offset would be associated with: greater anhedonia and avolition measured via clinical ratings and EMA, lower frequency of positive emotional experiences and recreational activities, diminished goal-directed activity measured via EMA, and reductions in passive markers of activity measured via accelerometry; 3)

The negativity bias would be intact in CHR and not associated with clinical or EMA measures of negative symptoms; 4) The positivity offset deficit would be present in CHR cases with and without comorbid mood disorders; 5) Reductions in the positivity offset difference score would be associated with greater cross-sectional risk for conversion to a psychotic disorder measured via the SHARP risk calculator (Zhang et al., 2018) and more severe depressive symptoms.

Method

Participants

One hundred thirty-six individuals at clinical high-risk for psychosis (CHR) and 57 healthy controls (CN) completed the study. Zero CN and 36 CHR participants were excluded for not meeting a priori digital phenotyping adherence standards (i.e., responding to < 20% of momentary surveys). This resulted in a final sample of 100 one hundred participants at CHR, including 66 sixty-six individuals with a mood disorder diagnosis (CHR-M) and 34 thirty-four without (CHR-NM). CHR participants were recruited from three independent research labs located in northeast Georgia, metro-Atlanta, and metro-Chicago, as well as through community referrals through the Georgia Psychiatric Risk Evaluation Program (G-PREP), Emory Mental Health & Development (MHAD) Program, and Adolescent Development and Preventive Treatment (ADAPT) Program. 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 attenuated positive symptom syndrome (APSS), a psychosis-risk syndrome determined by the Structured Interview for Psychosis-Risk Syndromes (SIPS) (Miller et al., 2003). Forty-six participants met criteria for APSS Persistence (i.e., experiencing attenuated positive symptoms meeting the CHR threshold defined by the SIPS that have occurred at least once per week over

the past month and have not emerged in the past year or worsened over time), 51 for APSS Progression (i.e., experiencing attenuated positive symptoms meeting the CHR threshold defined by the SIPS that have occurred at least once per week over the past month and that either onset or significantly worsened in the past year), 2 for APSS Partial Remission (i.e., having a history attenuated positive symptoms meeting the CHR threshold defined by the SIPS that have either fallen below the CHR threshold in the past 6 months or failed to meet the criteria of once per week over the past month), and 1 for APSS Full Remission (i.e., having a history attenuated positive symptoms meeting the CHR threshold defined by the SIPS that fell below the CHR threshold for over 6 months). None of the participants met criteria for Brief Intermittent Psychosis Syndrome or Genetic Risk and Functional Decline Syndrome or lifetime criteria for a DSM-5 psychotic disorder.

CN participants were recruited from the northeast Georgia community using printed and online advertisements. Exclusion criteria for CN included current major psychiatric disorder diagnoses, SZ-spectrum personality disorders established by the SCID-5 (First et al., 2015a). and SCID-5-PD (First et al., 2015b), family history of psychosis, and current psychotropic medications. All participants were free from lifetime neurological disease. CHR and CN groups did not significantly differ on age, ethnicity, sex, personal education, or parental education. CHR participants had significantly lower adherence to momentary mobile surveys than CN participants. CHR-M participants were significantly older than CHR-NM and CN participants, and CHR-NM participants completed significantly less education than CHR-M and CN participants. Neither age nor personal education were significantly correlated with the positivity and negativity parameters with the exception of the raw negativity intercept. CHR-M participants had significantly greater general symptoms measured by the SIPS compared to CHR-NM. All

other clinical symptoms were of comparable magnitude between CHR groups. A greater number of participants in the CHR-M group were taking medications compared to the CHR-NM group, but groups did not significantly differ on the distributions of specific medication classes, including antipsychotics, mood stabilizers, antidepressants, anxiolytics, or stimulants. See Table 1 for information regarding demographics, medication, and clinical ratings.

Procedure

The study consisted of an initial study visit to determine eligibility and complete clinical interviews and symptom ratings followed by 6 consecutive days of digital phenotyping.

Initial study visit

The initial laboratory visit took place either in-person or, due to COVID-19 safety regulations, via webcall. To establish diagnoses and symptom ratings, participants were administered the SCID-5, SCID-5-PD, SIPS, Negative Symptom Inventory-Psychosis Risk (NSIPR) (Strauss et al., 2020), and the Global Functioning Scale: Social (GFS:S) and Global Functioning Scale: Role (GFS:R) (Cornblatt et al., 2007). Lab personnel trained to reliability standards (>0.80) conducted the interviews and established clinical consensus with the PI. SIPS responses were also used to determine cross-sectional conversion risk among CHR participants based on the formula by Zhang et al. (2018) that incorporates items measuring functional decline, positive, negative, and general symptoms. Lab personnel also trained participants digital phenotyping procedures, including how to install the mEMA app from Ilumivu on their personal mobile device and how to respond to surveys within the app.

Digital Phenotyping

Active Digital Phenotyping. Participants received 8 survey notifications per day for 6 days. Surveys were quasi-randomly scheduled within 90-minute epochs between 9 AM and 9

PM. Participants had 15 minutes to respond to each survey and received unlimited time to complete the questions. Momentary surveys assessed the following:

Momentary Emotional Experience. In-the-moment levels of positive and negative emotion were assessed using items from the modified Differential Emotions Scale (mDES; (Fredrickson et al., 2003)). Participants were asked to rate five negative (anger, fear, sadness, shame, anxiety) and five positive emotions (amused, content, happy, love, pride) on a 1-100 sliding scale anchored between "Not at all" and "Extremely." Surveys also prompted participants to identify their current emotional context as positive, negative, neutral, or mixed, which were used to determine the frequency of positive experiences over the 6-day period.

Momentary Emotional Arousal. In-the-moment emotional arousal was assessed via the survey question "How keyed-up or excited are you right now?", which was rated on a 1-100 sliding scale anchored between "Not at all" and "Extremely."

Momentary Negative Symptoms. Each momentary survey assessed current negative symptoms, including anhedonia, avolition, and asociality. To measure anhedonia, responses for consummatory (i.e., "How much are you enjoying the activity?" and "How much are you enjoying this social interaction?") and anticipatory pleasure (i.e., "How much do you think you will enjoy that activity the next time you do it?" and "How much do you think you will enjoy interacting with them next time?") were averaged. To measure avolition, participants were asked to rate their level of interest in a current activity (i.e., "How interested are you in the activity?"). When participants denied currently being engaged in an activity (i.e., doing "Nothing."), avolition was instead measured by rating momentary desire to engage in an activity (i.e., "How much do you want to be doing an activity right now?"). Asociality was assessed via participants' current level of interest in a social interaction (i.e., "How interested are you in this social

interaction?"). If participants reported that they were not currently interacting with anyone, asociality was measured as current desire to interact with others (i.e., "How much do you want to be interacting with someone right now?"). All items measuring negative symptoms were rated on a 1 (not at all) to 100 (extremely) scale. All items have shown convergent validity via associations with corresponding BNSS subdomains and confirmatory factor analysis indicates that they reflect 3 independent factors (Raugh et al., 2020).

Infrequent Responding. Each momentary survey contained a question from the Chapman Anhedonia Scale (Eckblad et al.,1982) to monitor infrequent responding. Infrequency items required participants to respond "True" or "False" to common, every day scenarios (e.g., "Sometimes when walking down the sidewalk, I have seen children playing."; "I cannot remember a time when I talked with someone who wore glasses."). Surveys with infrequent responses were excluded from analysis. The rate of infrequent responding was low (<13%) in both groups.

Passive Digital Phenotyping. mEMA settings were applied to enable sensors within the participants' smartphones to measure accelerometry with each change in XYZ coordinate motion (i.e., every change in accelerometry being logged as a single instance with separate values output for X, Y, and Z movement axes). Accelerometry data was encrypted and de-identified while stored on Ilumivu servers until downloaded by the researchers. Accelerometry variables used in the current study have shown convergent validity with clinically rated negative symptoms (Strauss et al., 2022). The primary accelerometry variables included aggregate means (ACL.mean) and standard deviations (ACL.SD) calculated for each subject at the level of week.

Data Analysis

Consistent with prior work (Bartolomeo et al., in press), the positivity offset and negativity bias were calculated based on the approach described in Ito and Cacioppo (2005), where participants' subjective reports of valence and arousal were used to model the positivity and negativity functions at the individual-level. Momentary ratings of arousal, positive emotion, and negative emotion were entered as regression parameters in the equation $E = Ax + b$, where E represents the output of the affective system (i.e., level of positive or negative emotion) and A represents the affective input (i.e., level of arousal). The resulting intercepts from the positivity and negativity functions reflect the output when affective input is absent, and the slopes reflect the rate of change in positivity or negativity as affective input increases. To obtain the most reliable estimates, positivity and negativity parameters were calculated across all days and summarized at the level of the week. The positivity offset was modeled by subtracting the average intercept of the negativity function from the average intercept of the positivity function, and the negativity bias was modeled by subtracting the average positivity slope from the average negativity slope.

Preliminary analyses of standard comparisons of momentary valence and arousal, as well as additional exploratory analyses examining the effects of sex are also described in Supplemental Materials. To determine whether CHR and CN groups displayed the prototypical positivity offset and negativity bias, within-group paired sample t-tests were conducted comparing positive and negative intercepts and slopes. Separate one-way ANOVAs were conducted to compare positivity offset and negativity bias difference scores between groups, as well as the raw positivity and negativity intercepts and slopes. To evaluate the relationship between the positivity offset and negative symptoms, Pearson correlations were conducted

between the positivity offset difference score and anhedonia and avolition measured via the NSI-PR and active and passive digital phenotyping measures of anhedonia and avolition or activity (i.e., mean accelerometry and standard deviation). Pearson correlations were also used to examine the relationship between the positivity offset difference score and the number of positive experiences endorsed as a behavioral measure of anhedonia, time spent engaged in goal-directed activities, and number of recreational activities during the digital phenotyping period, as well as with cross-sectional risk for conversion calculated according to Zhang et al. (2018) and depressive symptoms measured by the SIPS Dysphoric Mood item. Correlations were also conducted using the negativity bias difference score and raw positivity and negativity parameters for exploratory purposes. All analyses were repeated with the CHR group divided into subgroups of individuals with (CHR-M) and without a mood disorder diagnosis (CHR-NM).

Results

Group Comparisons of Positivity and Negativity Parameters

Both CN and CHR exhibited the prototypical positivity offset with significantly greater intercepts for the positivity than negativity function (CN: $t = 12.53$, $p < .001$; CHR: $t = 2.13$, $p = .04$). The positivity offset difference score was significantly reduced in participants at CHR for psychosis compared to CN. Group differences in the raw positivity intercept and slope were nonsignificant.

When the CHR group was separated into participants with and without mood disorder diagnoses, CHR-NM demonstrated an intact positivity offset ($t = 2.98$, $p = .01$), whereas CHR-M participants exhibited comparable intercepts for the positivity and negativity functions ($t = .29$, $p = .77$). One-way ANOVA revealed a significant group difference in the positivity offset between CHR-NM, CHR-M, and CN. Post-hoc comparisons indicated that CHR-NM and CN did not

significantly differ on the positivity offset difference score, whereas CHR-M participants exhibited a significantly reduced positivity offset compared to both CHR-NM and CN. Regarding the raw positivity parameters, the positivity intercept was significantly different between groups. Similar to the positivity offset difference scores, post-hoc comparisons indicated that CHR-NM and CN did not significantly differ on the raw positivity intercept, whereas the raw positivity intercept was significantly reduced in CHR-M participants compared to both CHR-NM and CN. Group differences in the raw positivity slope were nonsignificant between CHR-NM, CHR-M, and CN.

Neither group demonstrated the negativity bias, evidenced by nonsignificant differences in the positivity and negativity slopes (CN: $t = 1.84$, $p = .07$; CHR: $t = 1.91$, $p = .06$). Group differences in the negativity bias difference scores were nonsignificant. The raw negativity intercept was significantly greater in the CHR group compared to CN, whereas group differences in the raw negativity slope were nonsignificant.

When the CHR group was split into CHR-NM and CHR-M, CHR-NM did not display a negativity bias ($t = -.50$, $p = .62$). In contrast, the negativity bias was inverted among CHR-M participants, who demonstrated a significantly higher slope for the positivity than negativity function ($t = 2.77$, $p = .01$). Group differences in the negativity bias difference score between CHR-NM, CHR-M, and CN were marginally significant. Post-hoc comparisons indicated that both CHR-NM and CHR-M participants did not significantly differ from CN; however, the negativity bias was significantly reduced in CHR-M compared to CHR-NM. The raw negativity intercept also significantly differed between groups. CHR-NM and CN participants had comparable negativity intercepts, whereas CHR-M participants displayed significantly higher negativity intercepts than both CHR and CN groups. This suggests that both an elevated raw

negativity intercept and diminished positivity intercept contributed to the reduced positivity offset in the CHR-M subgroup. Group differences in the raw negativity slope were nonsignificant between CHR-NM, CHR-M, and CN.

See Table 2 for results of group comparisons of all positivity and negativity parameters and Figures 1 and 2 for plots of the positivity and negativity functions by group.

Correlations with Positivity and Negativity Parameters

Among participants at CHR for psychosis, lower positivity offset difference scores were associated with more severe anhedonia measured by the NSI-PR (r = -.33, p < .001), but not active digital phenotyping (r = -.16, p = .13). The positivity offset difference score was not significantly correlated with avolition measured via the NSI-PR (r = -.09, p = .41), active digital phenotyping (r = -.19, p = .07), or passive digital phenotyping (i.e., ACL.mean: r = .27, p = .18; ACL.sd: r = .09, p = .65). Additionally, correlations between the positivity offset and the number of positive contexts (r = .08, p = .45), time spent in goal-directed activities (r = -.05, p = .62), and number of recreational activities (r = .14, p = .18) endorsed during the digital phenotyping period were nonsignificant. In terms of raw positivity parameters, lower raw positivity intercepts were significantly associated with more severe clinically rated anhedonia (r = -.22, p = .04) and more severe avolition measured via active digital phenotyping (r = -.24, p = .02). Lower raw positivity slopes were associated with more severe avolition measured via passive digital phenotyping (i.e., ACL.mean, r = -.53, p = .01; ACL.sd, r = -.42, p = .03). None of the raw positivity parameters were significantly correlated with active digital phenotyping measures of anhedonia or clinically rated avolition (p's > .05).

In the CHR group, correlations between the negativity bias difference score and anhedonia and avolition measured via the NSI-PR and active digital phenotyping were

nonsignificant. Higher negativity bias difference scores were associated with more severe avolition measured via passive digital phenotyping (i.e., ACL.mean: $r = .45$, $p = .02$). Regarding the raw negativity parameters, higher negativity intercepts ($r = .40$, $p < .001$) and lower negativity slopes ($r = -.28$, $p = .01$) were significantly associated with more severe clinically rated anhedonia. Correlations between the raw negativity parameters and anhedonia measured via active digital phenotyping were nonsignificant, as were correlations with avolition measured via the NSI-PR and active digital phenotyping (p's > .05). Higher raw negativity slopes were associated with less severe avolition measured via passive digital phenotyping (i.e., ACL.mean: $r = .44$, $p = .03$). None of the positivity or negativity parameters were significantly correlated with the frequency of positive contexts endorsed during the digital phenotyping period as a behavioral measure of anhedonia in daily life (p's > .05).

Correlations between all positivity and negativity parameters and cross-sectional conversion risk were nonsignificant (p's > .05). Lower positivity offset difference scores were associated with more severe depressive symptoms measured by the SIPS (i.e., Dysphoric Mood item: $r = -.22$, $p = .03$). Higher negativity intercepts were also associated with more severe depressive symptoms ($r = .23$, $p = .03$).

Discussion

The present study aimed to determine if the positivity offset theory of anhedonia extends to individuals at CHR for psychosis using active and passive digital phenotyping. The role of mood pathology was also assessed by comparing individuals at CHR for psychosis with and without comorbid mood disorders and CN. Several important findings emerged.

Standard analyses of momentary emotional experience indicated intact hedonic responding in daily life among individuals at CHR for psychosis compared to CN (i.e., no group

differences in positive emotion reported during pleasant contexts); however, consistent with hypotheses, when mathematical approaches from the ESM were applied, participants at CHR for psychosis demonstrated a reduced positivity offset measured via active digital phenotyping compared to CN. Further, among individuals at CHR for psychosis, greater reductions in the positivity offset were associated with more severe clinically rated anhedonia, but not avolition. Inconsistent with hypotheses, the positivity offset was not significantly associated with other measures of anhedonia and avolition, including momentary mobile surveys, real-world activity measured via accelerometry, or the frequency of positive emotional experiences and recreational activities or time spent in goal-directed activity measured via active digital phenotyping. Additionally, lower raw intercepts for the positivity function were associated with more severe anhedonia, and reduced hedonic capacity measured by the raw slope for the positivity function was associated with reduced activity measured via accelerometry. Together, these results are generally consistent with laboratory findings from Riehle et al. (2022) and past digital phenotyping findings from Bartolomeo et al. (in press) by demonstrating that the diminished positivity offset and its association with clinically-rated and active digital phenotyping measures of anhedonia occurs across the psychosis continuum in the real world. Thus, in contrast to SZ who display intact hedonic capacity and a diminished positivity offset, negative symptoms among those at CHR are related to a reduced positivity offset and diminished hedonic capacity.

Consistent with hypotheses and prior findings in SZ (Bartolomeo et al., in press; Bartolomeo & Strauss, under review; Strauss et al., 2017) and CHR (Bartolomeo & Strauss, under review), the negativity bias was intact among individuals at CHR for psychosis. Inconsistent with hypotheses, smaller negativity bias difference scores were associated with more severe avolition measured via accelerometry, suggesting that those with motivational

deficits have difficulties ramping up negative emotion when it matters most (i.e., when arousal is high). This may reflect activation of withdrawal behavior resulting from elevated levels of negative emotional responding with increasing affective input, suggesting that the negativity function is adaptively calibrated in attenuated psychosis. These results differ from prior digital phenotyping findings in SZ showing no relationship with the negativity bias and real-world avolition (Bartolomeo et al., in press), as well as prior laboratory findings in SZ indicating that greater negativity bias difference scores were associated with more severe avolition (Bartolomeo & Strauss, under review). Collectively, findings point to distinct differences in the association between the negativity function and avolition across the psychosis continuum, which may reflect changes in the affective system that accompany illness onset and progression; however, the positivity offset was not associated with greater cross-sectional conversion risk.

Contrary to hypotheses, the positivity offset was only diminished in the CHR-M subgroup and intact among individuals at CHR without a mood disorder diagnosis. Further, the association between reductions in the positivity offset deficit and anhedonia was only significant in the CHR-M subgroup. This suggests that the positivity offset deficit found in the overall CHR group was driven by participants with co-occurring mood pathology, which is further supported by the significant correlation between depressive symptoms and lower positivity offset difference scores in the overall CHR group. This diverges from past digital phenotyping findings showing that both individuals with schizophrenia and schizoaffective disorder exhibit a reduced positivity offset compared to CN (Bartolomeo et al., in press), but is consistent with laboratory findings indicating that the positivity offset reduction is only present in affective psychosis (Bartolomeo & Strauss, under review). Mood symptoms may therefore be differentially related to the real-world positivity offset and associations with anhedonia across the psychosis

continuum. Additionally, the impact of mood symptoms on the positivity offset and its association with anhedonia may be more pronounced when measured using laboratory-based emotional experience paradigms that can fully tax the affective system, whereas digital phenotyping methods capture a more restricted range of affective inputs. Specifically, the laboratory paradigm includes stimuli with a range of normative valence and arousal ratings, allowing for full engagement of the affective system. In contrast, most of the situations in which participants complete mobile surveys tend to be in neutral, low arousal settings. Additionally, there are often barriers to completing surveys that do occur during highly pleasant or unpleasant and highly arousal contexts, which limits the extent to which we are tapping into the full range of affective responding. It is therefore important for future laboratory and digital phenotyping studies to include individuals with mood disorders without psychosis in order to determine what aspects of the ESM are unique to or shared between psychosis and mood pathology. Such findings are essential for identifying specific mechanistic pathways that can be targeted in intervention. For example, the current CHR-M sample demonstrated an inverted negativity bias and studies on adults with MDD indicate an elevated negativity bias (Gollan et al., 2015), neither of which has been observed in any of the previous SZ samples, even when split into affective versus nonaffective psychosis (Bartolomeo et al., in press; Bartolomeo & Strauss, under review; Strauss et al., 2017). Together, these findings suggest that the functions governing the affective system may be differentially calibrated across distinct symptom clusters and levels of clinical severity.

The current findings are subjected to certain limitations. First, the positivity and negativity parameters were calculated using subjective emotional responses and the physiological component of emotional responding was not addressed. For example, laboratory

studies indicate that skin conductance and heart rate variability, including at rest and in response to pleasant relative to neutral stimuli, are related to hedonic deficits among adolescents and adults with varying degrees of depressive symptoms and adults with schizophrenia (Benning & Ait Oumeziane, 2016; Borrione et al., 2018; Sanders & Abaied, 2015; Trémeau, 2022; Vazquez et al., 2016), though findings are inconsistent (Fitzgibbons & Simons, 1992; Trémeau, 2022; Ward et al., 1983; Watson, 1972). Examining such processes that can be passively measured using passive digital phenotyping may provide additional insight into biological processes contributing to the positivity offset deficit as a mechanism of anhedonia in daily life. Second, since EMA/digital phenotyping was used, long-term longitudinal clinical follow-ups designed to examine transition were not available, leaving gaps in understanding for how the positivity offset and its relationship to anhedonia changes over time among individuals at CHR for psychosis. Longitudinal research in this population is essential for understanding how affective abnormalities influence clinical trajectories in this population, including who is at greater risk for developing psychotic disorders versus maintaining mood pathology, which may warrant different approaches to early intervention and prevention.

In addition to informing current conceptual models of anhedonia in psychosis, the present findings have important clinical implications. Recently, novel treatments for anhedonia have expanded to include repetitive transcranial magnetic stimulation (rTMS) targeting specific cortical regions (e.g., dorsolateral prefrontal cortex) (Fukuda et al., 2021), which has been shown to have top-down effects on upregulating the serotonin system (Peng et al., 2018). Although the biological basis of the positivity offset is unknown, potential pathways may include the serotonin system (Ashare et al., 2013) and other neuromodulatory genes (Norris et al., 2011). In particular, past research has shown that polymorphisms in the HTR1A and HTR2A genes, which are related

to affective responding, are differentially related to the positivity offset based on sex (Ashare et al., 2013). In males, the 102T allele was associated with a greater positivity offset than the 102C allele, whereas in females, the 1019C allele was associated with a greater positivity offset than the 1019G allele (Ashare et al., 2013). Although the serotonergic system is implicated in the pathophysiology of SZ (Selvaraj et al., 2014), evidence regarding the association between these specific polymorphisms and SZ is conflicting (Melkersson & Hulting, 2009; Newman-Tancredi et al., 2012; Zhang et al., 2004); however, further research into the neurobiological mechanisms underlying the positivity offset across the psychosis continuum may inform novel targets for intervention and prevention that can be targeted using treatments like rTMS. Additionally, the present findings add further support for psychosocial interventions aiming to enhance positive emotional experience in schizophrenia, such as the Positive Emotions Program for Schizophrenia (Favrod et al., 2015; Favrod et al., 2019; Nguyen et al., 2016) and suggest such treatments should also prioritize increasing positive emotion in neutral contexts.

In conclusion, the present results suggest that the positivity offset theory of anhedonia does not only apply to the most severe end of the psychosis continuum but also attenuated forms of illness. Results extend prior evidence (Bartolomeo et al., in press) that the positivity offset reduction occurs in the context of daily life not only among adults with psychotic disorders, but also among individuals at CHR for psychosis. Unlike studies in adults with psychotic disorders that indicate the diminished positivity offset is associated with reductions in real-world approach behavior, the positivity offset in daily life was only associated with clinically rated anhedonia and not any of the digital phenotyping measures among individuals at CHR; however, deficits in hedonic capacity measured by the slope for the positivity function were associated with reduced motivated behavior measured via passive digital phenotyping. This discrepancy may suggest that

the nature of the abnormality in the affective system and its impact on real-world motivated behavior differs across phases of illness. In other words, while individuals at both phases demonstrate the positivity offset deficit, abnormalities in hedonic capacity (i.e., the affective system's ability to respond with greater levels of positivity to increasing affective input) may have a unique impact on motivated behavior in attenuated forms of illness. Importantly, the positivity offset reduction in attenuated psychosis occurred only among individuals with comorbid mood disorders, consistent with past studies suggesting that depression accounts for hedonic abnormalities observed in CHR (Strauss et al., 2018; Gruber et al., 2018).

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Table 4.1

Participant Demographic and Clinical Characteristics

	CHR (n = 100)	CN (n = 57)	Test statistic
Age	22.36 (4.08)	21.18 (2.77)	$F = 3.81$, $p = .05$, $\eta^2 = .02$ [0, .09]
Parental Education	15.38 (2.92)	15.79 (2.74)	$F = .77$, $p = .38$, $\eta^2 = .01$ [0, .05]
Personal Education	14.10 (2.44)	14.36 (1.46)	$F = .54$, $p = .47$, $\eta^2 = .003$ [0, .04]
Female (%)	79	77	$\chi^2 = .07$, $p = .79$
Race (%)			$\chi^2 = 4.75$, $p = .48$
Black	13	9	-
Asian	12	14	-
Latinx	11	5	-
White	53	67	-
Multiracial	10	5	-
Other	1	0	-
Medication (n)			
Antipsychotic	6	-	
Mood Stabilizer	6	-	
Antidepressant	22	-	
Anxiolytic	6	-	
Stimulant	3	-	
None	71	57	
NSI-PR Avolition	1.74 (.92)	-	-
NSI-PR Anhedonia	1.47 (.86)	-	-

NSI-PR Asociality	1.49 (.92)	-	-
NSI-PR Blunted Affect	1.02 (1.27)	-	-
NSI-PR Alogia	.58 (1.00)	-	-
SIPS Positive	2.13 (.69)	-	-
SIPS Negative	1.22 (.78)	-	-
SIPS General	1.92 (1.11)	-	-
SIPS Disorganized	1.03 (.64)	-	-
Survey Adherence Rate	.61 (.37)	.74 (.19)	5.28, p = .02, η^2 = .03 [0, .10]

	CHR-NM (n=34)	CHR-M (n=66)	CN (n=57)	Test Statistic
Age	21.18 (3.66)	22.97 (4.18)	21.18 (2.77)	F = 4.73, p = .01 η^2 = .06 [.004, .13] CHR, CN < CHR-M
Parental Education	15.18 (2.79)	15.48 (2.99)	15.79 (2.74)	F = .51, p = .60 η^2 = .007 [0, .04]
Personal Education	13.38 (2.24)	14.47 (2.46)	14.36 (1.46)	F = 3.28, p = .04 η^2 = .04 [0, .11] CHR < CHR-M, CN
Female (%)	71	77	77	χ^2 = 2.22, p = .33
Race (%)				χ^2 = 11.55, p = .32
Black	9	15.2	9	-
Asian	12	12	14	-
Latinx	6	14	5	-
White	56	52	67	-
Multiracial	15	8	5	-
Other	3	0	0	-
Medication (n)				

Antipsychotic	1	5	-	
Mood Stabilizer	1	5	-	
Antidepressant	4	18	-	
Anxiolytic	0	6	-	
Stimulant	0	3	-	
None	30	41	57	
NSI-PR Avolition	1.76 (.89)	1.74 (.95)	-	$F = .01, p = .92$ $\eta^2 = 0$ [0, .02]
NSI-PR Anhedonia	1.38 (.78)	1.52 (.90)	-	$F = .53, p = .47$ $\eta^2 = .01$ [0, .07]
NSI-PR Asociality	1.36 (.90)	1.56 (.93)	-	$F = 1.09, p = .30$ $\eta^2 = .01$ [0, .09]
NSI-PR Blunted Affect	1.00 (1.31)	1.03 (1.27)	-	$F = .01, p = .91$ $\eta^2 = 0$ [0, .03]
NSI-PR Alogia	.53 (1.13)	.61 (.92)	-	$F = .13, p = .72$ $\eta^2 = .001$ [0, .05]
SIPS Positive	2.19 (.74)	2.10 (.67)	-	$F = .37, p = .54$ $\eta^2 = .004$ [0, .07]
SIPS Negative	1.08 (.83)	1.30 (.75)	-	$F = 1.80\ p = .18$ $\eta^2 = .02$ [0, .10]
SIPS General	1.73 (1.13)	2.01 (1.10)	-	$F = 1.32, p = .25$ $\eta^2 = .01$ [0, .09]
SIPS Disorganized	1.04 (.78)	1.02 (.57)	-	$F = .02, p = .89$ $\eta^2 = 0$ [0, .03]
Survey Adherence Rate	.64 (.21)	.60 (.43)	.74 (.19)	$F = 2.84, p = .06$ $\eta^2 = .04$ [0, .10]

Note. CHR = overall clinical high-risk group; CN = control group; CHR-NM = clinical high-risk group without comorbid mood disorders; CHR-M = clinical high-risk group with comorbid mood disorders. NSI-PR = Negative Symptom Inventory – Psychosis Risk. SIPS = Structured

Interview for Prodromal Syndromes. NSI-PR and SIPS domain scores reflect average of item scores within each domain.

Table 4.2

One-way ANOVAs Comparing Positivity and Negativity Parameters Between Clinical High-Risk and Control Groups

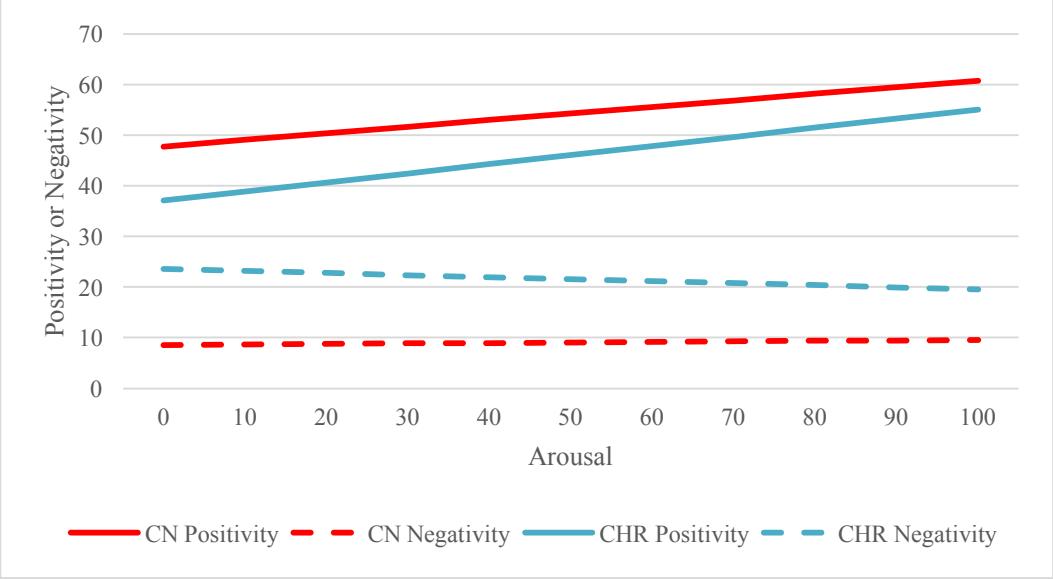
	CHR (n = 100)	CN (n = 57)	Test Statistic
Positivity Intercept	37.06 (43.92)	47.76 (16.04)	$F(1, 156) = 3.14$, $p = .08$ $\eta^2 = .02$ [0, .08]
Negativity Intercept	23.57 (29.60)	8.58 (12.14)	$F(1, 156) = 13.31$, $p < .001$ $\eta^2 = .08$ [.02, .17]
Positivity Slope	.18 (.86)	.13 (.35)	$F(1, 156) = .17$, $p = .68$ $\eta^2 = .001$ [0, .03]
Negativity Slope	-.04 (.47)	.01 (.21)	$F(1, 156) = .75$, $p = .39$ $\eta^2 = .01$ [0, .05]
Positivity Offset	13.49 (63.24)	39.18 (23.61)	$F(1, 156) = 8.69$, $p = .004$ $\eta^2 = .05$ [.01, .13]
Negativity Bias	-.22 (1.16)	-.12 (.48)	$F(1, 156) = .43$, $p = .51$ $\eta^2 = .003$ [0, .04]

	CHR-NM (n=34)	CHR-M (n=66)	CN (n=57)	Test Statistic
Positivity Intercept	49.91 (60.40)	30.45 (30.91)	47.76 (16.04)	$F(2. 156) = 4.95$, $p = .01$ $\eta^2 = .06$ [.004, .14] CHR-M < CN, CHR-NM
Negativity Intercept	35.11 (32.73)	27.16 (28.61)	8.58 (12.14)	$F(2, 156) = 10.79$, $p < .001$ $\eta^2 = .12$ [.04, .22] CN, CHR-NM < CHR-M
Positivity Slope	-.06 (1.06)	.30 (.72)	.13 (.35)	$F(2, 156) = 2.92$, $p = .06$ $\eta^2 = .04$ [0, .10]
Negativity Slope	.04 (.32)	-.09 (.53)	.01 (.21)	$F(2, 156) = 1.59$, $p = .21$ $\eta^2 = .02$ [0, .07]
Positivity Offset	35.71 (69.87)	2.05 (56.77)	39.18 (23.61)	$F(2, 156) = 9.46$, $p < .001$ $\eta^2 = .11$ [.03, .20] CHR-M < CN, CHR-NM
Negativity Bias	.10 (1.15)	-.39 (1.14)	-.12 (.48)	$F(2, 156) = 3.12$, $p = .047$ $\eta^2 = .04$ [0, .11] CHR-M < CHR-NM

Note. CHR = overall clinical high-risk group; CN = Control group; CHR-NM = Clinical high-risk participants without a comorbid mood disorder; CHR-M = Clinical high-risk participants with a comorbid mood disorder; Positivity Offset Difference Score = Positivity Intercept – Negativity Intercept. Negativity Bias Difference Score = Negativity Slope – Positivity Slope. Values reflect Mean (SD) unless otherwise indicated.

Figure 4.1

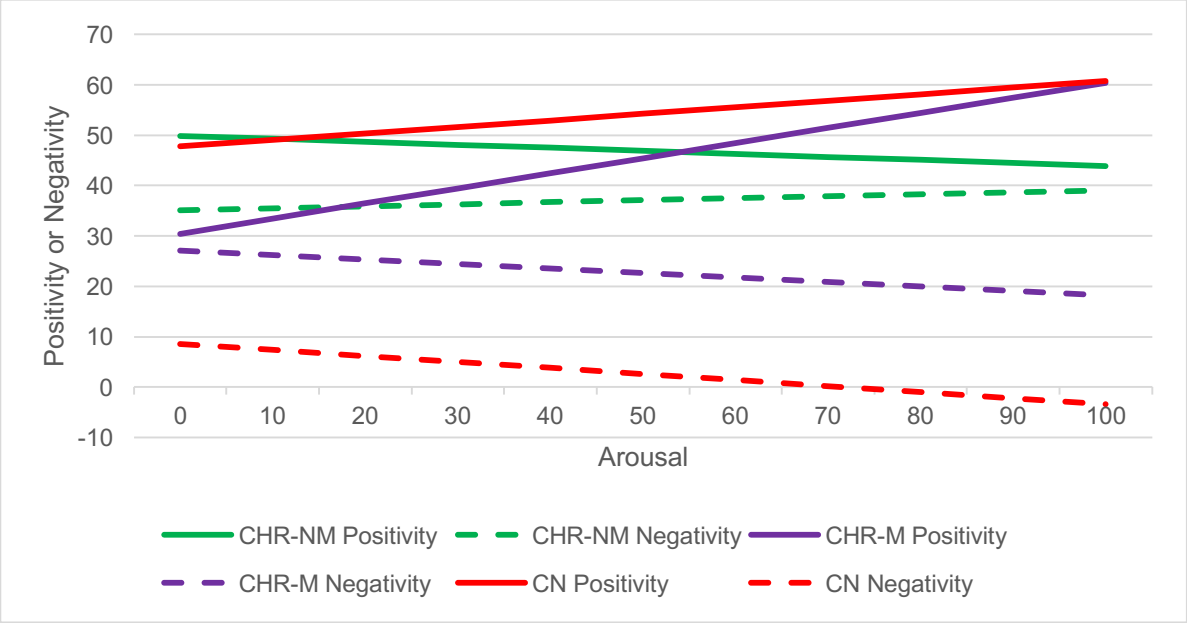
Positivity and Negativity Functions in Clinical High-Risk and Control Groups



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

Figure 4.2

Positivity and Negativity Functions in Mood-Based Clinical High-Risk and Control Groups



Note. CHR-NM = clinical high-risk group without comorbid mood disorders; CHR-M = clinical-high-risk group with comorbid mood disorders; CN = control group.

CHAPTER 5

CONCLUSION

The three studies within the current compilation dissertation applied Cacioppo's ESM to determine whether reductions in the positivity offset account for the liking-wanting and schizophrenia-spectrum anhedonia paradoxes.

Liking-wanting Anhedonia Paradox

The liking-wanting anhedonia paradox refers to the failure of intact hedonic responding to lead to motivated behavior in SZ (Strauss & Cohen, 2008). It was hypothesized that the liking-wanting paradox may be explained by a diminished positivity offset that impedes approach motivation facilitating engagement in pleasurable activities and acquisition of rewards and resources in neutral contexts. To unpack the liking-wanting anhedonia paradox, Manuscript 1 used the ESM in conjunction with digital phenotyping to assess whether assumptions of hedonic normality in SZ that are central to current conceptual models of negative symptoms are premature and if the diminished positivity offset, in addition to reward processing abnormalities, contributes to real-world deficits in motivation and pleasure. It was expected that hedonic capacity measured by the slope for the positivity function would be comparable or elevated in SZ relative to healthy controls and that the positivity offset would be reduced in daily life and associated with clinically rated and real-world measures of anhedonia and avolition. Manuscript 2 also explored the possibility that the liking-wanting paradox reflects a diminished positivity offset using the same laboratory-based emotional experience paradigm administered by Strauss et al., (2017). Similar to Manuscript 1, it was hypothesized that hedonic capacity measured by

the positivity slope would be intact, the positivity offset would be diminished, and greater reductions in the positivity offset would be associated with more severe clinically rated anhedonia and avolition.

At the group level, evidence for intact hedonic capacity in SZ was replicated using the EMA data in Manuscript 1 and lab-based data in Manuscript 2 as indicated by the nonsignificant group effects for the positivity slope; however, both EMA and lab-based studies indicated a reduction in the positivity offset at the group level, despite intact hedonic capacity. Broadly speaking, these group contrasts are consistent with the notion that the positivity offset reduction may account for the liking-wanting anhedonia paradox. Results of correlational analyses in Manuscript 1 demonstrated this more clearly by providing evidence for associations between the diminished positivity offset and real-world motivated behaviors measured via active and passive digital phenotyping. Thus, among individuals with SZ, the hypothesis that the positivity offset may explain the liking-wanting anhedonia paradox was supported. See Tables 1 and 3.

Schizophrenia-Spectrum Anhedonia Paradox

The schizophrenia-spectrum anhedonia paradox refers to different patterns of hedonic responsivity across the psychosis continuum, such that individuals with SZ demonstrate intact hedonic responding and individuals at CHR for psychosis and with schizotypy demonstrate deficits in hedonic responding (Cohen et al., 2011; Cohen et al., 2012; Martin et al., 2020; Najolia et al., 2011). The current body of work explored whether both mild and severe ends of the psychosis continuum are characterized by a diminished positivity offset that contributes to anhedonia measured via structured clinical interviews and active and passive digital phenotyping. Manuscript 2 set to determine whether the schizophrenia-spectrum anhedonia paradox truly is a paradox or if anhedonia and avolition are attributed to the same hedonic

abnormality (i.e., deficits in the positivity offset) across the psychosis continuum. To this end, individuals with full psychotic disorders (i.e., SZ and SZaff) and individuals at CHR for psychosis completed a laboratory-based emotional experience paradigm to determine whether both groups demonstrated a diminished positivity offset that is associated with clinically rated anhedonia and avolition. Additionally, Manuscript 2 aimed to determine whether phases of illness are differentiated by abnormalities in hedonic capacity measured by the slope for the positivity function. It was expected that both groups would demonstrate a reduction in the lab-based positivity offset, with greater reductions associated with more severe clinically rated anhedonia and avolition. It was also hypothesized that individuals at CHR for psychosis would demonstrate a true hedonic deficit measured via the slope for the positivity function, whereas individuals with SZ would demonstrate intact or elevated hedonic capacity. Manuscript 3 extended Manuscripts 1 and 2 to further explore the schizophrenia-spectrum anhedonia paradox by using digital phenotyping to determine whether the positivity offset is reduced in the context of daily life and contributes to real-world anhedonia among individuals at CHR for psychosis. It was expected that individuals at CHR would exhibit deficits in the positivity offset and hedonic capacity in daily life, both of which would be associated with more severe anhedonia and avolition measured via clinical interviews and digital phenotyping.

As expected, results of Manuscripts 1 and 2 indicated that individuals with SZ displayed intact hedonic capacity measured by the slope for the positivity function. Both studies also indicated that SZ had reductions in the positivity offset. However, contrary to hypotheses, the positivity offset reduction was only present among individuals at CHR for psychosis in the EMA study (Manuscript 3) and not in the laboratory-based study (Manuscript 2), which may reflect reduced power due to sample size since effects were in the right direction and similar effect size

as the SZ sample. Furthermore, the hypothesis that CHR would uniquely display true anhedonia (i.e., reduced hedonic capacity measured by the slope for the positivity function) was not supported. This was evidenced by an intact positivity slope among individuals at CHR for psychosis across all studies using both laboratory-based and EMA methodology. Similarly, the slope for the positivity function was intact among individuals with full psychotic disorders across studies, as expected. Thus, results examining hedonic capacity from the perspective of the ESM did not support the existence of the schizophrenia-spectrum anhedonia paradox at all, as had been concluded from prior laboratory-based studies using traditional univariate analyses of positive emotion to pleasant stimuli. This is because neither individuals at CHR for psychosis or with SZ appeared to have a true anhedonia (i.e., reduced hedonic capacity as indicated by the slope for the positivity function). However, there was some support, albeit inconsistent, for our proposal that the both groups would display a reduction in the positivity offset. However, these reductions did not appear greater among the SZ group compared to individuals at CHR for psychosis, as would be expected if there was indeed a schizophrenia-spectrum anhedonia paradox, as comparisons of effect sizes in the positivity offset difference score across studies suggest a small to medium reduction in both groups compared to healthy controls (eta squared range = .02 to .11). See Table 1.

However, correlational analyses (Table 3) provided further clarification that clinically rated anhedonia was associated with reductions in the positivity offset among individuals with SZ at a comparable magnitude in both laboratory and EMA studies. Additionally, more severe anhedonia was associated with greater deficits in hedonic capacity (i.e., slope for the positivity function) among individuals with SZ in the context of daily life. The positivity offset reduction but not diminished hedonic capacity showed robust associations with behavioral markers of

motivation and recreational behavior among individuals with psychotic disorders, suggesting that the positivity offset may indeed be a more apt explanation for anhedonia/avolition in this population than diminished capacity. Among individuals at CHR for psychosis, correlational results were inconsistent. Manuscript 2 showed an association between anhedonia and diminished hedonic capacity but not the positivity offset, whereas Manuscript 3 found the opposite. Additionally, associations with more objective active and passive digital phenotyping measures of negative symptoms were nonsignificant in Manuscript 3.

Overall, there was some evidence that both individuals at CHR for psychosis and with full psychotic disorders display hedonic abnormalities at the group level that were of comparable magnitude. However, these abnormalities were better described as a reduction in the positivity offset than diminished hedonic capacity (i.e., true anhedonia). Furthermore, reductions in the positivity offset and hedonic capacity were both associated with individual differences in clinically rated anhedonia, but when observed, significant associations with real-world deficits in recreational and motivated behavior were more strongly linked to the positivity offset than hedonic capacity. Thus, while the ESM-based analyses did not support the existence of the schizophrenia-spectrum anhedonia paradox (i.e., lower hedonic capacity at the less severe end of the psychosis continuum compared to intact or elevated capacity at the more severe end), there was evidence that the positivity offset accounted for hedonic abnormalities moreso than diminished hedonic capacity in both groups. Individual differences in anhedonia across the psychosis continuum were also generally more robustly associated with the positivity offset than hedonic capacity, consistent with the prediction that the positivity offset would provide a meaningful explanation for anhedonia throughout the SZ-spectrum.

The Role of Mood in the Positivity Offset and Hedonic Capacity Reductions

All 3 manuscripts applied a transdiagnostic approach by examining the impact of mood pathology on the positivity offset and associations with anhedonia across the psychosis continuum. Specifically, groups with full psychotic disorders were separated into affective versus non-affective psychosis, and individuals at CHR for psychosis were separated into those with and without comorbid mood disorder diagnoses. Correlations with clinically rated depressive symptoms and the positivity offset were also examined to determine dimensional effects.

Results were inconclusive regarding the extent to which mood symptoms account for the positivity offset among individuals with full psychotic disorders and those at CHR. In Manuscript 1, individuals with SZ and SZaff did not differ in positivity offset. In contrast, Manuscript 2 found that only SZaff had a positivity offset reduction compared to CN. In both Manuscripts 1 and 2, correlations between the positivity offset and depression were nonsignificant. Thus, in those diagnosed with full psychotic disorders, Manuscripts 1 and 2 showed consistent results that current depression severity was not associated with individual differences in the positivity offset, although the two studies produced conflicting results when mood was considered categorically. Collectively, these findings make it unclear whether positivity offset reductions are most significant among those with mood symptoms or common to both those with and without mood pathology in people with full psychotic disorders.

Manuscripts 2 and 3 differed regarding whether the positivity offset was found at the group level in CHR (present in Manuscript 3 but not Manuscript 2) and whether the positivity offset reduction was greater with those carrying a comorbid mood diagnosis (Manuscript 3

indicated greater reductions among those with comorbid mood pathology while Manuscript 2 indicated no differences). Furthermore, dimensional correlational analyses also produced inconsistent results in Manuscripts 2 and 3, as depression was significantly associated with reductions in the positivity offset in Manuscript 3 but not Manuscript 2. Thus, similar to conclusions that can be drawn among those with full psychotic disorders, the inconsistent findings across studies (both using categorical and dimensional approaches) makes the extent to which mood symptoms are contributing to the positivity offset reduction in CHR unclear.

Regarding mood, another prediction made by the literature is that hedonic capacity reductions would be more prominent in (or even exclusive to) those with co-occurring mood symptoms. Categorical analyses examining traditionally calculated variables (i.e., positive emotion to pleasant stimuli in laboratory studies or pleasant contexts in daily life measured via EMA) were consistent with this notion of lower positive affect in those with mood diagnoses (see supplemental materials in manuscripts 1-3). However, when viewed in relation to the ESM, findings were less clear. Categorical analyses conducted on mood vs non-mood groups provided no evidence for significantly greater reductions in positivity slope in those with mood compared to those without mood diagnoses in laboratory or EMA studies. Additionally, any trend level effects were inconsistent across manuscripts. Specifically, individuals with SZ showed a greater trend toward a hedonic capacity deficit than SZaff in the laboratory study, but this trend was reversed in the EMA study. Individuals at CHR with mood diagnoses also showed a trend toward having greater slopes for the positivity function than CN and those without mood diagnoses (i.e., greater hedonic capacity and the opposite of what would be expected if those with mood symptoms have a deficit in hedonic capacity). Thus, there was limited evidence that mood disorders are associated with a reduction in hedonic capacity when viewed through the lens of

the ESM. Rather, emotional experience abnormalities occurring in those with and without mood disorders seem more likely to be explained by the positivity offset than true reductions in hedonic capacity.

Importantly, there is significant etiological and symptom heterogeneity across mood and psychotic disorders that is unaccounted for in conceptual and mechanistic models of anhedonia (Treadway & Zald, 2011). In particular, these models fail to discriminate between hedonic and motivational components of anhedonia, or "liking" and "wanting," respectively. Similar to what is observed in psychosis, there is inconsistent evidence regarding whether major depressive disorder (MDD) is characterized by deficits in consummatory pleasure (i.e., hedonic responding) (Treadway & Zald, 2011), which has led researchers to explore the role of reward processing abnormalities that may underlie deficits in anticipatory pleasure and motivational anhedonia. Specifically, individuals with MDD exhibit poorer learning from positive feedback, deficits in reward anticipation, and reduced willingness to expend effort for rewards stemming from impaired effort-cost computation (Treadway et al., 2012; Treadway & Zald, 2011; Treadway & Zald, 2013). Thus, consistent with the current nonsignificant results of mood-based categorical analyses comparing the slope for the positivity function, anhedonia in MDD is not solely characterized by deficits in hedonic capacity. Therefore, the current findings add to current conceptual models by suggesting that anhedonia, regardless of which disorder it occurs in (i.e., affective versus nonaffective psychosis, attenuated psychosis syndrome with and without comorbid mood disorders, MDD, bipolar disorder, etc.), may not always or even often be due to a true hedonic capacity deficit. Rather, in addition to a broad range of reward processing impairments, anhedonia may stem from other affective abnormalities, such as reductions in the positivity offset and emotion regulation (e.g., difficulties up-regulating positive emotion and

down-regulating negative emotion). Together, these abnormalities impede decision-making processes that promote reward-seeking behavior and acquisition of resources that reflect the transdiagnostic construct of motivational anhedonia.

Limitations and Future Directions

Although the transphasic and transdiagnostic nature of the current body of work is a major strength, this approach was limited by certain aspects of study design. First, both the laboratory-based and digital phenotyping studies were not longitudinal in nature. Collectively, the studies examined how the positivity and negativity functions are calibrated and related to anhedonia at different stages of illness; however, they did not assess how these processes change with illness progression (i.e., from the prodromal phase into active and enduring psychosis using a within-subjects design rather than between). Given that negative symptoms are highly predictive of conversion and worse functional outcomes among individuals at CHR for psychosis (Carrión et al., 2016; Cornblatt et al., 2006; Devoe et al., 2021; Kwapil, 1998; Mason et al., 2004; Piskulic et al., 2012; Velthorst et al., 2009; Yung et al., 2005), identifying whether the positivity offset also contributes to conversion as a mechanism of anhedonia is imperative for improving approaches to early intervention and prevention. Although results from Studies 2 and 3 indicated that the positivity offset was not associated with cross-sectional conversion risk, longitudinal studies examining how abnormalities in the calibration of the affective system differ between converters and nonconverters are still needed. From a transdiagnostic perspective, longitudinal studies in individuals at CHR for psychosis are also necessary for determining whether the positivity offset, negativity bias, or any of the other parameters of the positivity and negativity functions predict the development of a psychotic disorder versus the development or maintenance a mood disorder. To this end, it would be beneficial to add multiple clinical

comparisons groups. Another limitation of the study design is only using hedonic responding at the subjective level to measure the positivity and negativity functions. Future studies should also incorporate laboratory-based and ambulatory psychophysiological measures (e.g., skin conductance, heart rate variability, event-related potentials, pupil dilation) to determine if the positivity offset reduction is detected in both the subjective and physiological components of emotional responding and the impact of these components subsequent behavior.

Implications

The current results have important clinical implications for the modification and development of treatments targeting negative symptoms, including anhedonia and avolition, across phases of psychosis. Specifically, the present findings suggest that psychosocial interventions aiming to reduce anhedonia and avolition should focus on elevating positive emotion and dampening negative emotion in neutral contexts to normalize the positivity offset and facilitate goal-directed behavior. In addition to evidence across studies for a diminished positivity offset among individuals with full psychotic disorders, this is further supported by evidence for a heightened intercept for the negativity function in Study 1 and a reduced intercept for the positivity function in Study 2. Potential approaches for reducing negative emotion in neutral contexts include a combination of emotion regulation, cognitive-behavioral (CBT), and dialectical-behavioral therapy (DBT) strategies. Focusing on helping patients effectively identify and implement contextually adaptive emotion regulation strategies may be beneficial for lowering the intercept for the negativity function. In addition to training in using distraction and reappraisal to down-regulate negative emotion in neutral contexts, interventions should also aim to build awareness into factors causing and maintaining negative emotions, such as maladaptive beliefs. For example, a person with defeatist performance or anhedonic beliefs (particularly

stable beliefs that persist in neutral contexts) may benefit from cognitive restructuring to generate more adaptive beliefs over time. Behavioral interventions stemming from CBT and DBT approaches, including pleasant activity scheduling, behavioral activation, building positive experiences, and practicing opposite action, can also alleviate negative emotion in neutral contexts, while also directly challenging maladaptive beliefs that maintain negative emotionality (Choi et al., 2016; Perivoliotis et al., 2010). Lastly, physiological interventions (e.g., paced or diaphragmatic breathing, progressive muscle relaxation) are response modulation emotion regulation strategies (McRae & Gross, 2020) that may also be helpful for targeting heightened resting autonomic nervous system activity that is characteristic of psychotic disorders and contributes to negative affect even in neutral contexts (Guccione et al., 2019; Stogios et al., 2021; Zahn et al., 1981).

In terms of psychosocial interventions for elevating levels of positive emotion in neutral contexts, the therapeutic approach outlined in the Positive Emotion Program for Schizophrenia (PEPS) (Favrod et al., 2019a; Favrod et al., 2015; Favrod et al., 2019b; Nguyen et al., 2016), appears highly promising. PEPS utilizes an experiential learning approach to facilitate greater anticipation and maintenance of positive emotion while also decreasing defeatist performance beliefs (Nguyen et al., 2016). It is a skills-based intervention that aims to teach patients how to savor pleasant experiences, increase positive emotional expression, make the most of positive experiences by sharing with others, and increase anticipatory pleasure via imaginal exposures of pleasant experiences. A recent randomized controlled clinical trial indicated that PEPS was effective at improving anhedonia post-intervention and at 6-month follow-up (Favrod et al., 2019a). Applying the strategies outlined in PEPS in neutral contexts may increase the intercept for the positivity function and subsequently increase the positivity offset. Of note, the reduced

intercept for the positivity function found in Study 2 was specific to individuals with schizoaffective disorder, suggesting PEPS may be especially beneficial for affective psychosis. In contrast, the results of Study 1 suggest that psychosocial interventions targeting the elevated intercept for the negativity function may be warranted for both affective and nonaffective psychosis.

Examining the biological basis of the positivity offset reduction may inform additional treatments for anhedonia, which is especially important given the lack of FDA-approved treatments for negative symptoms in SZ. Several neurotransmitter systems have been implicated in anhedonia, including dopamine, serotonin, and γ-aminobutyric acid (GABA). A recent randomized, placebo-controlled, double-blind study in healthy controls indicated that administration of Amisulpride, a D2 receptor (D2R) antagonist, induced consummatory anhedonia indexed by subjective and psychophysiological (i.e., skin conductance) responding to pleasant visual stimuli (Berg et al., 2023). These findings support the role of D2 mediated neurotransmission in hedonic responding and suggest that D2R blocking antipsychotics may contribute to anhedonia (Berg et al., 2023); however, the current studies did not find evidence for medication effects on the positivity offset. Still, an important future direction is to determine how abnormalities in the dopaminergic system relate to the positivity offset, and as suggested by Berg et al., (2023), whether partial D2R/D3R agonists are more effective at preserving the hedonic response and normalizing the positivity offset.

Regarding the serotoninergic system, prior evidence in healthy controls indicates that HTR1A and HTR2A polymorphisms are differentially related to the positivity offset based on sex, such that the 102C and 1019C alleles are associated with an increased positivity offset in males and females, respectively (Ashare et al., 2013). The HTR1A and HTR2A genes are

involved in affective responding; however, the status of these polymorphisms and associations with the positivity offset in schizophrenia is unclear (Melkersson & Hulting, 2009; Newman-Tancredi et al., 2012; Zhang et al., 2004). Treatments for anhedonia in depression have recently expanded to applying repetitive transcranial magnetic stimulation (rTMS) to cortical regions like the dorsolateral prefrontal cortex (Fukuda et al., 2021), which in turn increases serotonergic transmission (Peng et al., 2018). Future studies should explore whether rTMS increases the positivity offset and improves anhedonia across the psychosis continuum.

Lastly, repeated ketamine infusions have also shown promise for improving anhedonia (Nogo et al., 2022), including as an intervention for individuals with treatment-resistant depression and bipolar disorder (Lally et al., 2014; Wilkowska et al., 2021). Ketamine is an NMDA-receptor antagonist that inhibits GABAergic interneurons, leading to increased glutamate and BDNF release. Historically, there has been concern that ketamine could exacerbate psychotic symptoms in patients with psychotic disorders (Beck et al., 2020; Lahti et al., 2001a, 2001b; Malhotra et al., 1997; Xu et al., 2015); however, there is evidence refuting this theory from studies examining depression with psychotic features and psychotic disorders (Galuszko-Wegielnik et al., 2023; Kim et al., 2020; Le et al., 2021; Veraart et al., 2021). Research into the effectiveness of ketamine for improving symptoms of depression and negative symptoms in SZ is sparse, and to our knowledge, no studies to date have isolated the effects of ketamine on anhedonia in this population. The majority of available research consists of case series and pilot studies with small sample sizes, and findings generally point to only short-term symptom improvement (Bartova et al., 2018; Nunes et al., 2018; Ye et al., 2019; Zhou et al., 2020). Despite these major gaps in knowledge, consistent evidence for the effectiveness of ketamine treatment for individuals with mood disorders suggests that it may be worth exploring

whether ketamine infusions improve anhedonia in psychosis by increasing the positivity offset. Such findings would help elucidate the biological basis of the positivity offset and mechanistic pathways to increasing positive relative to negative emotion in neutral contexts as a means of improving anhedonia in psychosis.

Conclusions

In conclusion, the results of the current compilation dissertation add to the field's conceptual understanding of the liking-wanting and schizophrenia-spectrum anhedonia paradoxes in several important ways. First, laboratory and digital phenotyping evidence for the diminished positivity offset and its association with clinically rated and real-world measures of anhedonia in both attenuated and fully psychotic phases of illness indicate that hedonic deficits do exist across the psychosis continuum in the form of reduced positive relative to negative emotion in neutral contexts that fails to yield engagement in pleasurable and goal-directed activities. The influence of mood pathology on the positivity offset and hedonic capacity in both individuals with full psychotic disorders and at CHR for psychosis was inconsistent across manuscripts and methods (i.e., laboratory-based and EMA studies). Further research incorporating longitudinal and more expansive transdiagnostic approaches is warranted to better understand the nature of affective abnormalities across psychosis and mood spectrums and identify mechanisms that can be targeted for individualized psychosocial and pharmacological interventions.

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Table 5.1

Results of One-way ANOVAs Comparing Positivity and Negativity Parameters Between Clinical and Control Groups Across Manuscripts – Dimensional Analyses

	Manuscript 1		
	SZ (n=44)	CN (n=48)	Test Statistic
Positivity Intercept	45.38 (24.86)	52.91 (21.06)	$F(1, 92) = 2.47$, $p = .12$, $\eta^2 = .03$
Negativity Intercept	30.41 (30.25)	11.19 (12.60)	$F(1, 92) = 16.30$, $p < .001$, $\eta^2 = .15$
Positivity Slope	-.15 (1.06)	.06 (.26)	$F(1, 92) = 1.72$, $p = .19$, $\eta^2 = .02$
Negativity Slope	.29 (1.38)	.03 (.19)	$F(1, 92) = 1.73$, $p = .19$, $\eta^2 = .02$
Positivity Offset	14.97 (46.66)	41.72 (30.07)	$F(1, 92) = 10.86$, $p = .001$, $\eta^2 = .11$
Negativity Bias	.44 (1.73)	-.03 (.40)	$F(1, 92) = 3.37$, $p = .07$, $\eta^2 = .04$
	Manuscript 2 – Study 1		
	SZ (n=98)	CN (n=84)	Test statistic
Positivity Intercept	1.88 (9.19)	1.83 (1.84)	$F(1, 182) = .002$, $p = .972$, $\eta^2 = 0$
Negativity Intercept	.92 (7.95)	-1.35 (8.66)	$F(1, 182) = 3.39$, $p = .07$, $\eta^2 = .02$
Positivity Slope	.25 (3.02)	.30 (.67)	$F(1, 182) = .03$, $p = .88$, $\eta^2 = 0$
Negativity Slope	.55 (2.64)	1.21 (2.87)	$F(1, 182) = 2.57$, $p = .11$, $\eta^2 = .01$
Positivity Offset	.96 (5.70)	3.19 (8.60)	$F(1, 182) = 4.35$, $p = .04$, $\eta^2 = .02$
Negativity Bias	.30 (1.86)	.91 (2.88)	$F(1, 182) = 2.89$, $p = .09$, $\eta^2 = .02$
	Manuscript 2 – Study 2		
	CHR (n=45)	CN (n=51)	Test Statistic
Positivity Intercept	1.15 (1.53)	1.53 (1.71)	$F(1, 96) = 1.26$, $p = .26$, $\eta^2 = .01$

Negativity Intercept	-.42 (2.01)	-.80 (12.60)	$F(1, 96) = .82$, $p = .37$, $\eta^2 = .01$
Positivity Slope	.49 (.43)	.41 (.57)	$F(1, 96) = .63$, $p = .43$, $\eta^2 = .01$
Negativity Slope	.91 (.58)	1.11 (.61)	$F(1, 96) = 2.79$, $p = .10$, $\eta^2 = .03$
Positivity Offset	1.57 (2.14)	2.32 (2.73)	$F(1, 96) = 2.20$, $p = .14$, $\eta^2 = .02$
Negativity Bias	.42 (.66)	.70 (.93)	$F(1, 96) = 2.95$, $p = .09$, $\eta^2 = .03$
		Manuscript 3	
	CHR (n=100)	CN (n=57)	Test Statistic
Positivity Intercept	37.06 (43.92)	47.76 (16.04)	$F(1, 156) = 3.14$, $p = .08$, $\eta^2 = .02$
Negativity Intercept	23.57 (29.60)	8.58 (12.14)	$F(1, 156) = 13.31$, $p < .001$, $\eta^2 = .08$
Positivity Slope	.18 (.86)	.13 (.35)	$F(1, 156) = .17$, $p = .68$, $\eta^2 = .001$
Negativity Slope	-.04 (.47)	.01 (.21)	$F(1, 156) = .75$, $p = .39$, $\eta^2 = .01$
Positivity Offset	13.49 (63.24)	39.18 (23.61)	$F(1, 156) = 8.69$, $p = .004$, $\eta^2 = .05$
Negativity Bias	-.22 (1.16)	-.12 (.48)	$F(1, 156) = .43$, $p = .51$, $\eta^2 = .003$

Note. SZ = overall schizophrenia group (i.e., individuals with schizophrenia and schizoaffective disorder); CHR = overall clinical high-risk for psychosis group (i.e., individuals at CHR with and without comorbid mood disorders); CN = control group.

Table 5.2

Results of One-way ANOVAs Comparing Positivity and Negativity Parameters Between Clinical and Control Groups Across Manuscripts – Categorical Analyses

Manuscript 1				
	SZ (n=18)	SZaff (n=26)	CN (n=48)	Test Statistic
Positivity Intercept	46.12 (20.25)	44.87 (28.00)	52.91 (21.06)	$F(2.\ 92) = 1.24$ $p = .30$, $\eta^2 = .03$
Negativity Intercept	35.11 (32.73)	27.16 (28.61)	11.19 (12.60)	$F(2, 92) = 8.82$ $p < .001$ $\eta^2 = .17$ SZaff, SZ > CN
Positivity Slope	.11 (.49)	-.33 (1.30)	.06 (.26)	$F(2, 92) = 2.80$ $p = .07$, $\eta^2 = .06$
Negativity Slope	.45 (1.73)	.18 (1.10)	.03 (.19)	$F(2, 92) = 1.26$ $p = .29$, $\eta^2 = .03$
Positivity Offset	11.01 (40.65)	17.71 (51.01)	41.72 (30.07)	$F(2, 92) = 5.54$ $p = .01$, $\eta^2 = .11$ SZaff, SZ < CN
Negativity Bias	.33 (1.84)	.52 (1.69)	-.03 (.40)	$F(2, 92) = 1.79$ $p = .17$, $\eta^2 = .04$
Manuscript 2 – Study 1				
	SZ (n=62)	SZaff (n=36)	CN (n=84)	Test Statistic
Positivity Intercept	3.16 (10.38)	-.34 (6.18)	1.83 (1.84)	$F(1, 182) = 3.06$ $p = .049$, $\eta^2 = .03$ SZaff < SZ
Negativity Intercept	1.28 (9.64)	.28 (3.57)	-1.35 (8.66)	$F(1, 182) = 1.85$ $p = .16$, $\eta^2 = .02$
Positivity Slope	-.11 (3.45)	.88 (2.00)	.30 (.67)	$F(1, 182) = 2.24$ $p = .11$, $\eta^2 = .02$
Negativity Slope	.45 (3.20)	.73 (1.18)	1.21 (2.87)	$F(1, 182) = 1.39$ $p = .25$, $\eta^2 = .02$
Positivity Offset	1.88 (4.34)	-.63 (7.29)	3.19 (8.60)	$F(2, 182) = 3.60$ $p = .03$, $\eta^2 = .04$ SZaff < CN
Negativity Bias	.57 (1.42)	-.15 (2.40)	.91 (2.88)	$F(1, 182) = 2.50$ $p = .09$, $\eta^2 = .03$
Manuscript 2 – Study 2				

	CHR-NM (n=28)	CHR-M (n=17)	CN (n=51)	Test Statistic
Positivity Intercept	1.44 (1.31)	.68 (1.76)	1.53 (1.71)	$F(1, 96) = 1.80$ $p = .17$, $\eta^2 = .04$
Negativity Intercept	-.01 (1.91)	-1.10 (2.04)	-.80 (12.60)	$F(1, 96) = 2.01$ $p = .14$, $\eta^2 = .04$
Positivity Slope	.39 (.40)	.65 (.43)	.41 (.57)	$F(1, 96) = 1.68$ $p = .19$, $\eta^2 = .04$
Negativity Slope	.79 (.56)	1.11 (.59)	1.11 (.61)	$F(1, 96) = 2.96$ $p = .06$, $\eta^2 = .06$
Positivity Offset	1.44 (2.35)	1.79 (1.81)	2.32 (2.73)	$F(1, 96) = 1.19$ $p = .31$, $\eta^2 = .03$
Negativity Bias	.39 (.74)	.45 (.54)	.70 (.93)	$F(1, 96) = 1.49$ $p = .23$, $\eta^2 = .03$
		Manuscript 3		
	CHR-NM (n=34)	CHR-M (n=66)	CN (n=57)	Test Statistic
Positivity Intercept	49.91 (60.40)	30.45 (30.91)	47.76 (16.04)	$F(2. 156) = 4.95$ $p = .01$, $\eta^2 = .06$ CHR-M < CN, CHR-NM
Negativity Intercept	35.11 (32.73)	27.16 (28.61)	8.58 (12.14)	$F(2, 156) = 10.79$ $p < .001$, $\eta^2 = .12$ CN, CHR-NM < CHR-M
Positivity Slope	-.06 (1.06)	.30 (.72)	.13 (.35)	$F(2, 156) = 2.92$ $p = .06$, $\eta^2 = .04$
Negativity Slope	.04 (.32)	-.09 (.53)	.01 (.21)	$F(2, 156) = 1.59$ $p = .21$, $\eta^2 = .02$
Positivity Offset	35.71 (69.87)	2.05 (56.77)	39.18 (23.61)	$F(2, 156) = 9.46$ $p < .001$, $\eta^2 = .11$ CHR-M < CN, CHR-NM
Negativity Bias	.10 (1.15)	-.39 (1.14)	-.12 (.48)	$F(2, 156) = 3.12$ $p = .047$, $\eta^2 = .04$ CHR-M < CHR-NM

Note. SZ = schizophrenia; SZaff = schizoaffective disorder; CHR-M = clinical high-risk for psychosis with comorbid mood disorders; CHR-NM = clinical high-risk for psychosis without comorbid mood disorders.

Table 5.3

Results of Bivariate Correlations Across Manuscripts – Dimensional Analyses

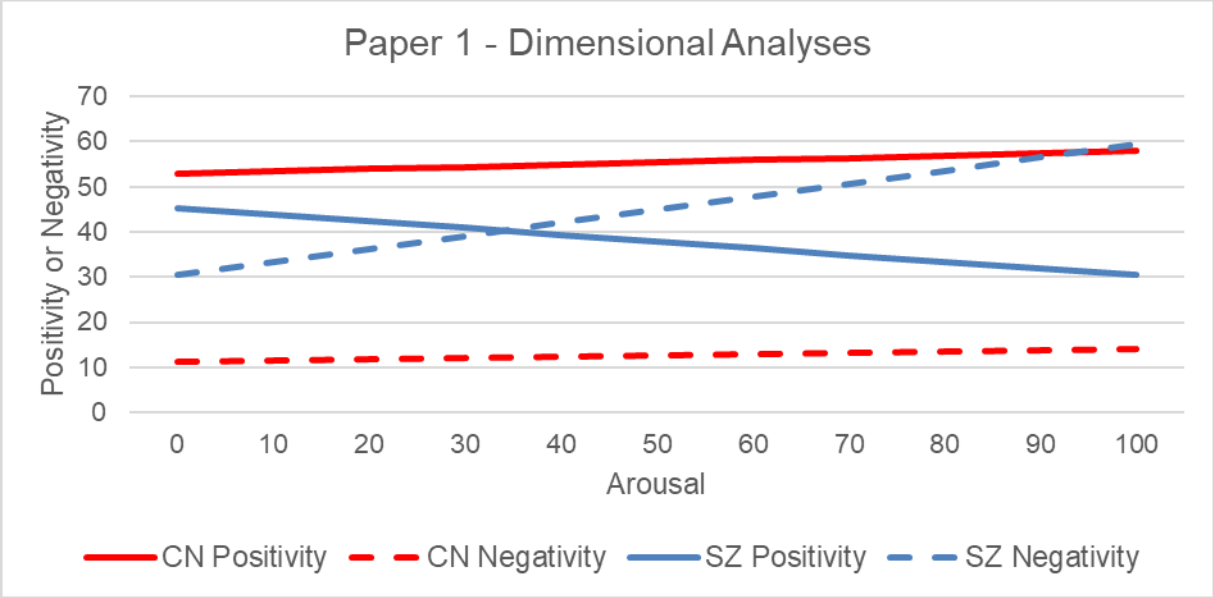
		Positivity Intercept	Positivity Slope	Negativity Intercept	Negativity Slope	Positivity Offset	Negativity Bias
Manuscript 1 (SZ)	BNSS Anhedonia	***r* = -.36** ***p* = .02**	***r* = -.36** ***p* = .02**	ns	ns	***r* = -.43** ***p* < .01**	ns
	BNSS Avolition	***r* = -.32** ***p* = .03**	ns	ns	ns	***r* = -.34** ***p* =.03**	ns
	EMA Anhedonia	ns	ns	***r* = .34** ***p* = .02**	ns	***r* = -.58** ***p* < .001**	ns
	EMA Avolition	ns	ns	***r* = .41** ***p* = .01**	ns	***r* = -.57** ***p* < .001**	ns
	Number of Positive Events	ns	ns	ns	ns	***r* = .34** ***p* = .03**	ns
	GPS	ns	ns	ns	ns	ns	ns
	ACL.mean	ns	ns	ns	ns	***r* = .53** ***p* = .02**	ns
	ACL.SD	ns	ns	***r* = .54** ***p* = .01**	ns	***r* = -.52** ***p* = .02**	ns
	Depression	ns	ns	ns	ns	ns	ns
Manuscript 2 (SZ)	BNSS Anhedonia	ns	ns	ns	ns	***r* = -.23** ***p* = .03**	ns
	BNSS Avolition	***r* = -.35** ***p* = .001**	***r* = .30** ***p* = .004**	ns	ns	***r* = -.31** ***p* = .003**	***r* = .23** ***p* = .03**
	Depression	ns	ns	ns	ns	ns	ns
Manuscript 2 (CHR)	BNSS Anhedonia	ns	***r* = -.30** ***p* = .045**	ns	ns	ns	ns
	BNSS Avolition	ns	ns	ns	ns	ns	ns
	Depression	***r* = -.30** ***p* = .04**	ns	ns	ns	ns	ns

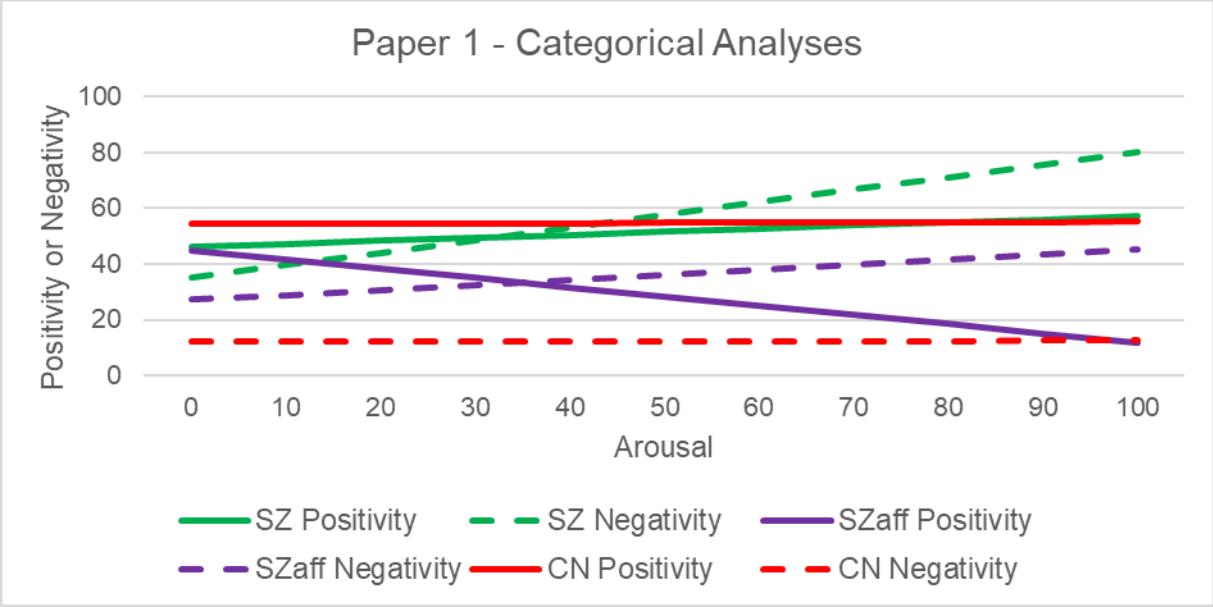
Manuscript 3 (CHR)	NSIPR Anhedonia	***r* = -.22** ***p* = .04**	ns	***r* = .40** ***p* < .001**	***r* = -.28** ***p* = .01**	***r* = -.33** ***p* < .001**	ns
	NSIPR Avolition	ns	ns	ns	ns	ns	ns
	EMA Anhedonia	ns	ns	ns	ns	ns	ns
	EMA Avolition	ns	ns	ns	ns	ns	ns
	Number of Positive Events	ns	ns	ns	ns	ns	ns
	ACL.mean	***r* = -.24** ***p* = .02**	ns	ns	***r* = .44** ***p* = .03**	ns	***r* = .45** ***p* = .02**
	ACL.SD	***r* = -.42** ***p* = .03**	ns	ns	ns	ns	ns
	Depression	ns	ns	***r* = .23** ***p* = .03**	ns	***r* = -.22** ***p* = .03**	ns

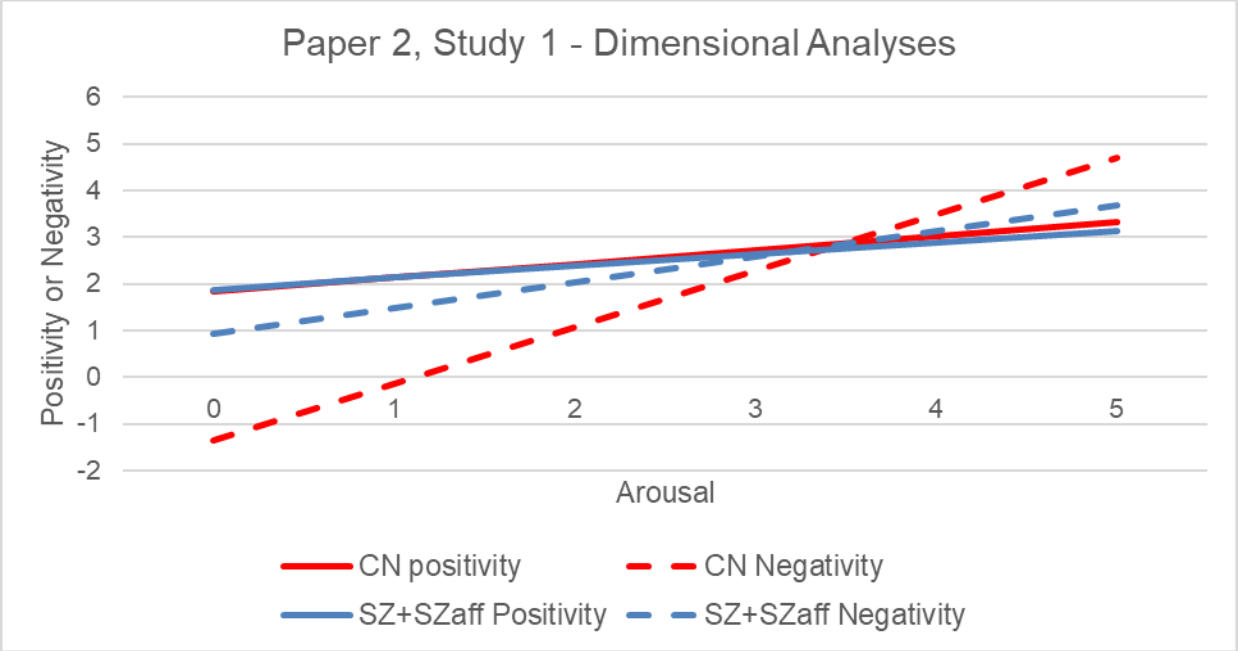
Note. SZ = overall schizophrenia group (i.e., individuals with schizophrenia and schizoaffective disorder); CHR = overall clinical high-risk for psychosis group (i.e., individuals at CHR with and without comorbid mood disorders); BNSS = Brief Negative Symptom Scale; NSIPR = Negative Symptom Inventory – Psychosis Risk; EMA = ecological momentary assessment (mobile surveys); ACL.mean = mean accelerometry; ACL.SD = accelerometry standard deviation; GPS = geolocation; ns = nonsignificant.

Figure 5.1

Positivity and Negativity Functions in Clinical and Control Groups Across Manuscripts



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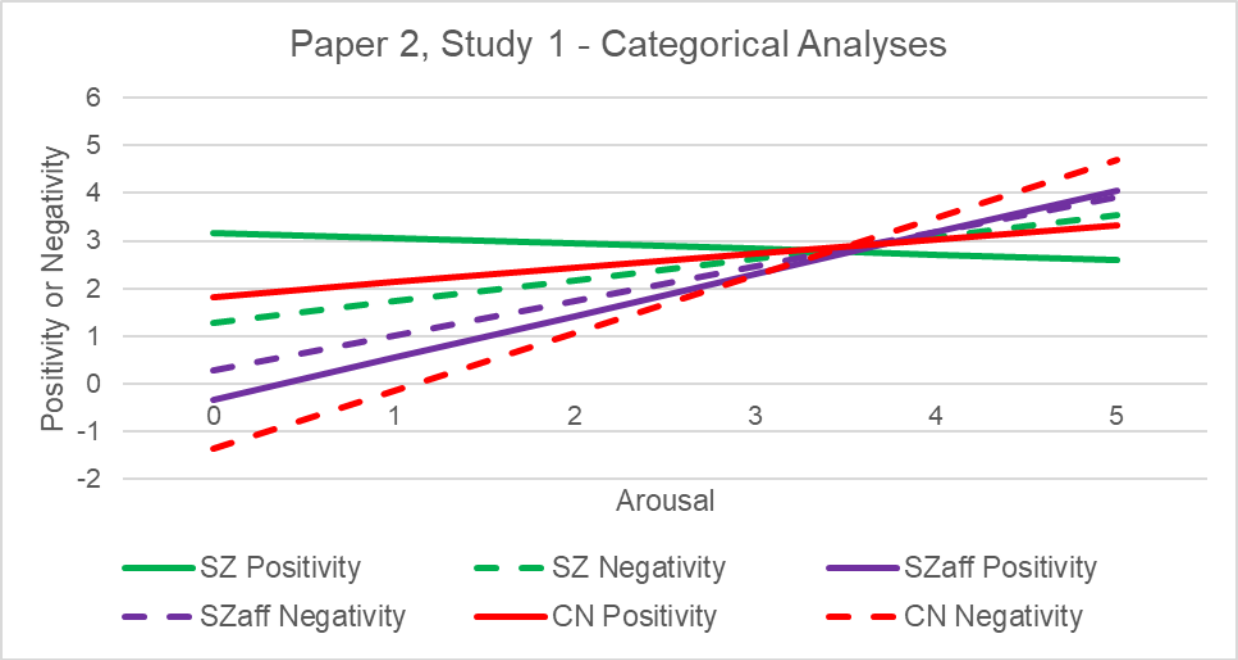
Arousal
CN positivity
CN Negativity
SZ+SZaff Positivity
SZ+SZaff Negativity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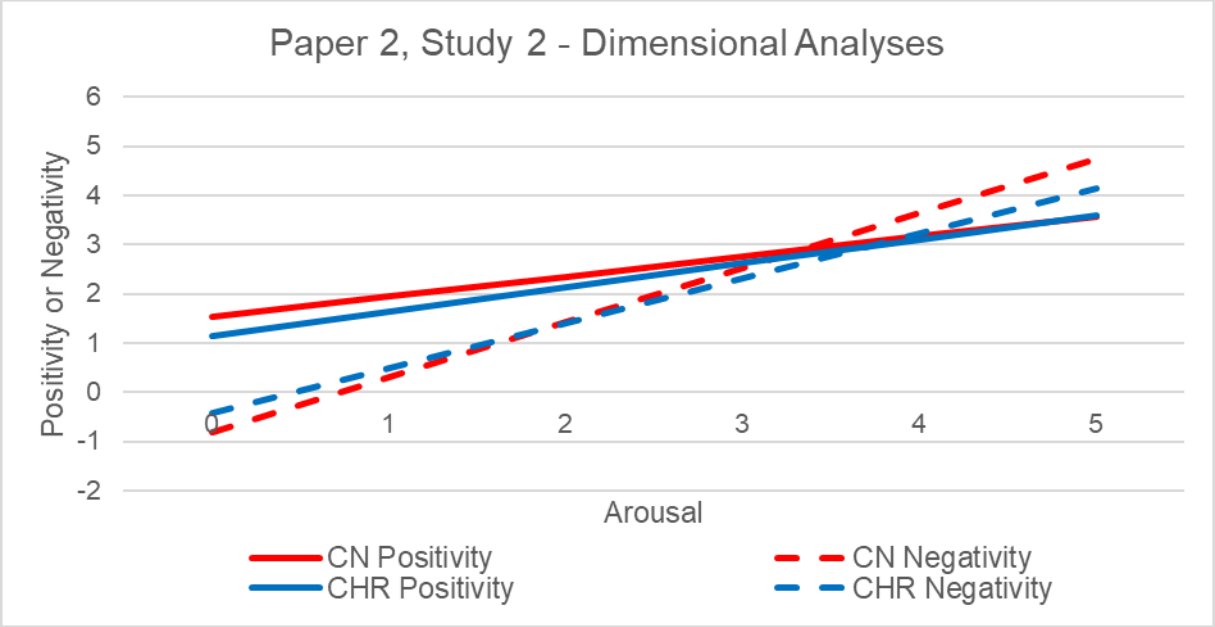
SZ Negativity
SZaff Positivity
SZaff Negativity
CN Positivity
CN Negativity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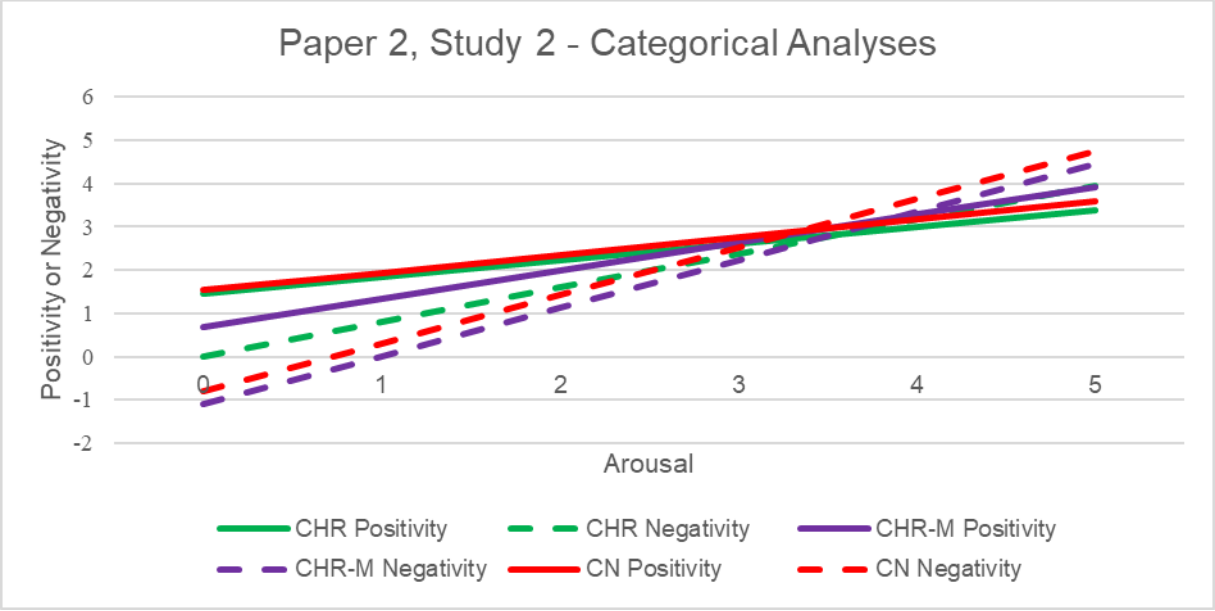
CHR Positivity
CN Negativity
CHR Negativity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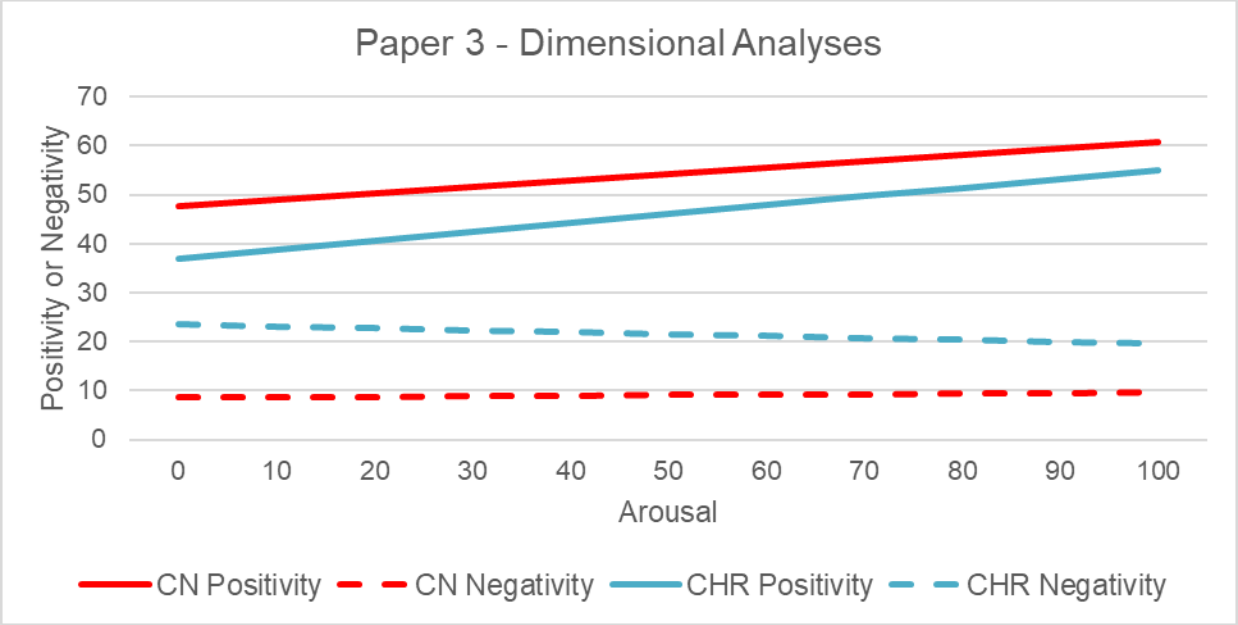
CHR Negativity
CHR-M Positivity
CHR-M Negativity
CN Positivity
CN Negativity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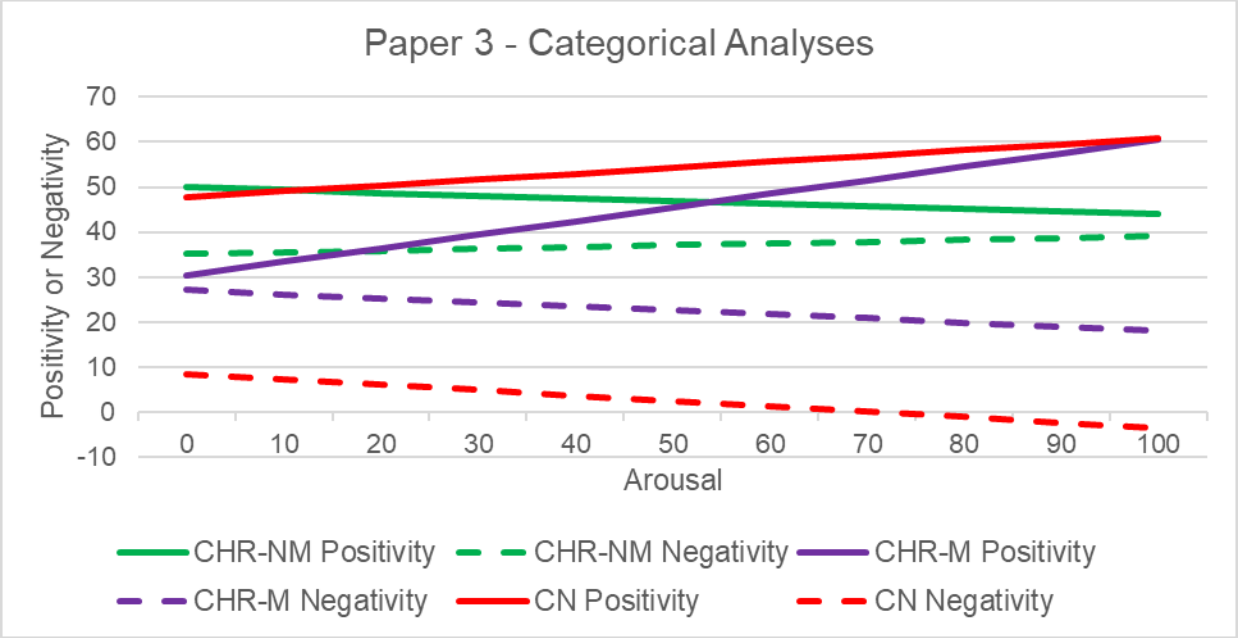
CN Negativity
CHR Positivity
CHR Negativity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80
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100
Arousal
CHR-NM Positivity
CHR-NM Negativity
CHR-M Positivity
CHR-M Negativity
CN Positivity
CN Negativity

APPENDEX A

CHAPTER 2 SUPPLEMENTAL MATERIAL

Supplemental Data Analysis

Standard preliminary analyses of self-reported emotional experience variables were conducted similar to past studies comparing subjective positivity, negativity, and arousal to pleasant, unpleasant, and neutral stimuli. The average level of positive affect, negative affect, and arousal were calculated for each participant using responses to surveys completed during positive, negative and neutral contexts and used as the dependent variables for separate Group (SZ, CN) x Emotion Context (Positive, Negative, Neutral) mixed models ANOVAs. Multi-level models were also conducted in R to examine the effects of Group, Emotion Context, and Day on positive affect, negative affect, and arousal. Separate one-way ANOVAs were also used to compare the frequency of positive, negative, and neutral contexts endorsed by SZ and CN participants during the digital phenotyping period.

Exploratory analyses consisted of conducting univariate ANOVA to examine the effects of two between-subjects factors, Sex (Male, Female) and Group (SZ, CN), and the Sex X Group interaction on the positivity offset and negativity bias difference scores, as well as the raw positivity and negativity parameters. Significant interactions were decomposed using one-way ANOVAs. One-way ANOVA was used to examine group (SZ, SZaff, CN) differences in the positivity offset and negativity bias difference scores and raw scores between CN and participants with schizophrenia and schizoaffective disorder. LSD tests were used for post-hoc comparisons between groups. To examine medication effects in the SZ group, exploratory point-

biserial correlations were conducted to examine the association between medication status (i.e., whether a participant was prescribed antipsychotics, coded as No = 0 and Yes = 1) and positivity offset and negativity bias difference scores. Lastly, bivariate correlations were used to examine the association between positivity offset and negativity bias scores with cognitive performance measured via the MATRICS Consensus Cognitive Battery (MCCB).

Supplemental Results

Preliminary Analyses

Preliminary analyses are displayed in Supplemental Table 1 and Figure 1. Mixed-models ANOVA indicated that for positive affect, there was a significant main effect of Emotion Context ($F(2, 48) = 40.63$, $p < .001$, $\eta_p^2 = 0.63$), while the main effect of Group ($F(1, 24) = 1.83$, $p = .19$, $\eta_p^2 = 0.07$) and the Group X Emotion Context interaction were nonsignificant ($F(2, 48) = .65$, $p = .49$, $\eta_p^2 = 0.03$). Similarly, multi-level models indicated significant effects of Context ($t = 4.99$, $p < .001$), Day ($t = -2.93$, $p = .003$), and the Group x Day interaction ($t = 3.36$, $p < .001$) on positive affect. The effects of Group ($t = .79$, $p = .43$), the Group X Emotion Context interaction ($t = 1.57$, $p = .12$), and the Group x Day x Context interaction ($t = -.52$, $p = .60$) were nonsignificant.

For negative affect, there were significant main effects of Context ($F(2, 48) = 45.33$, $p < .001$, $\eta_p^2 = 0.65$) and Group ($F(1, 24) = 5.69$, $p = .03$, $\eta_p^2 = 0.19$), as well as a nonsignificant Group X Context interaction ($F(2, 48) = 1.04$, $p = .33$, $\eta_p^2 = 0.04$). Multi-level models mirrored these results, yielding significant effects of Group ($t = -5.15$, $p < .001$) and Context ($t = -5.49$, $p = < .001$) on negative affect. The Group x Day ($t = -.66$, $p = .51$), Group x Context ($t = -.58$, $p = .57$), Day x Context ($t = .15$, $p = .88$), and Group x Day x Context interactions were all nonsignificant.

For arousal ratings, there was a significant main effect of Context ($F(2, 56) = 7.87$, $p = .001$, $\eta_p^2 = 0.22$), while the main effect of Group ($F(1, 28) = .02$, $p = .88$, $\eta_p^2 = 0.001$) and the Group X Context interaction were nonsignificant ($F(2, 56) = 1.30$, $p = .28$, $\eta_p^2 = 0.04$). The results of multilevel models indicated nonsignificant main effects and interactions, including Group ($t = -1.11$, $p = .27$), Day ($t = -.04$, $p = .97$), Context ($t = -.86$, $p = .39$), Group x Day ($t = -.45$, $p = .65$), Group x Context ($t = 1.95$, $p = .05$), Day x Context ($t = 1.21$, $p = .23$), and Group x Day x Context ($t = -.94$, $p = .35$).

SZ endorsed significantly fewer neutral contexts throughout the 6-day digital phenotyping period than CN ($M_{CN} = 30.25$, $SD_{CN} = 9.26$; $M_{SZ} = 23.61$, $SD_{sz} = 10.86$; $F(1, 58) = 10.00$, $p = .002$, $\eta_p^2 = 0.10$) and significantly more mixed (i.e., positive and negative) contexts ($M_{CN} = 1.13$, $SD_{CN} = 1.62$; $M_{SZ} = 2.23$, $SD_{sz} = 2.78$; $F(1, 91) = 5.52$, $p = .02$, $\eta_p^2 = 0.06$). Group differences in the frequency of positive ($M_{CN} = 2.52$, $SD_{CN} = 2.24$; $M_{SZ} = 3.42$, $SD_{sz} = 5.75$; $F(1, 44) = .52$, $p = .48$, $\eta_p^2 = 0.01$) and negative contexts ($M_{CN} = 2.30$, $SD_{CN} = 1.75$; $M_{SZ} = 2.34$, $SD_{sz} = 1.57$; $F(1, 59) = .01$, $p = .92$, $\eta_p^2 = 0$) were nonsignificant.

Exploratory Analyses

The results of univariate ANOVA indicated significant main effects of Group ($F(1, 92) = 5.31$, $p = .02$, $\eta_p^2 = .06$) and Sex ($F(1, 92) = .004$, $p = .95$, $\eta_p^2 = 0$) on the positivity offset difference score. The Group X Sex interaction was significant ($F(1, 92) = 4.74$, $p = .03$, $\eta_p^2 = 0.05$), such that, on average, the positivity offset was larger in females (M = 46.65, SD = 29.53) than males (M = 28.42, SD = 28.42) in the CN group and larger in males (M = 27.31, SD = 46.62) than females (M = 7.92, SD = 46.03) in the SZ group; however, post-hoc one-way ANOVA comparisons indicated that sex differences in the positivity offset were nonsignificant in both CN ($F(1, 47) = 3.69$, $p = .06$, $\eta_p^2 = 0.07$) and SZ ($F(1, 43) = 1.79$, $p = .19$, $\eta_p^2 = 0.04$). For

the negativity bias difference score, the main effect of Group was significant ($F(1, 92) = 4.45$, $p = .04$, $\eta_p^2 = .05$) and the main effect of Sex was nonsignificant ($F(1, 92) = .57$, $p = .45$, $\eta_p^2 = .01$), as was the Group x Sex interaction ($F(1, 92) = 1.72$, $p = .19$, $\eta_p^2 = .02$). When using the raw positivity slope to represent hedonic capacity, the main effects of Group ($F(1, 92) = 2.75$, $p = .10$, $\eta_p^2 = .03$), Sex ($F(1, 92) = .69$, $p = .41$, $\eta_p^2 = .01$), and the Group x Sex interaction were nonsignificant ($F(1, 92) = 1.99$, $p = .16$, $\eta_p^2 = .02$). When using the raw negativity slope score to represent the negativity bias, the main effects of Group ($F(1, 92) = 1.91$, $p = .17$, $\eta_p^2 = .02$), Sex ($F(1, 92) = .09$, $p = .76$, $\eta_p^2 = .001$), and the Group x Sex interaction were nonsignificant ($F(1, 92) = .32$, $p = .58$, $\eta_p^2 = .004$). See Supplemental Table 2 summary of Group and Sex effects for all positivity and negativity parameters.

Group (SZ, SZaff, and CN) comparisons indicated significant differences in the positivity offset difference score ($F(2, 92) = 5.54$, $p = .01$, $\eta_p^2 = .11$), such that individuals with both SZ (M = 11.01, SD = 40.65) and SZaff (M = 17.71, SD = 51.01) demonstrated a reduced positivity offset relative to CN (M = 41.72, SD = 30.07). Both SZ (M = 35.11, SD = 32.73) and SZaff (M = 27.16, SD = 28.61) groups also exhibited greater raw negativity intercepts than CN (M = 11.19, SD = 12.60) ($F(2, 92) = 8.82$, $p < .001$, $\eta_p^2 = .17$). Group differences for all other positivity and negativity parameters were nonsignificant (see Supplemental Table 3).

Correlations between antipsychotic medication status and the positivity offset ($r = .10$, $p = .55$) and negativity bias differences scores ($r = .29$, $p = .05$) were nonsignificant in SZ. Correlations between cognitive performance measured via the MCCB and the positivity offset ($r = -.12$, $p = .44$) and negativity bias difference scores ($r = -.18$, $p = .24$) were nonsignificant.

Supplemental Table 1

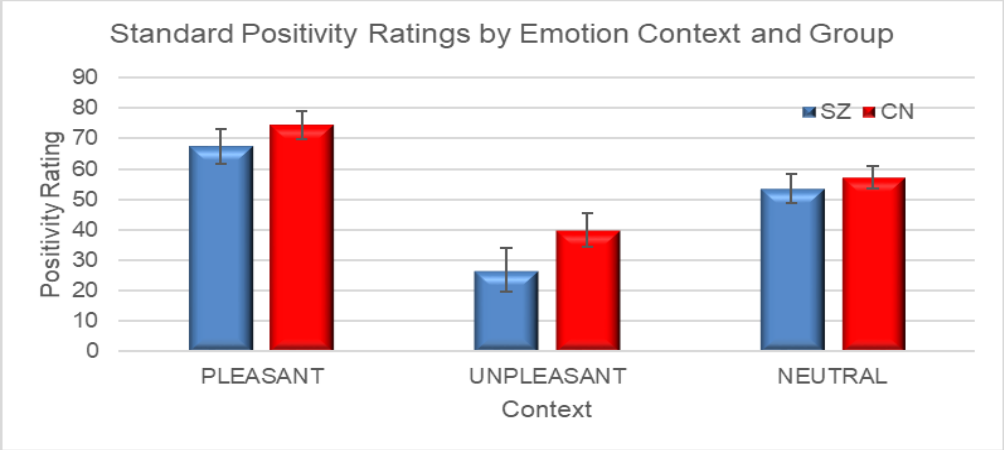
Mixed Models ANOVA Results Examining the Effects of Context and Group on Momentary Affect and Arousal

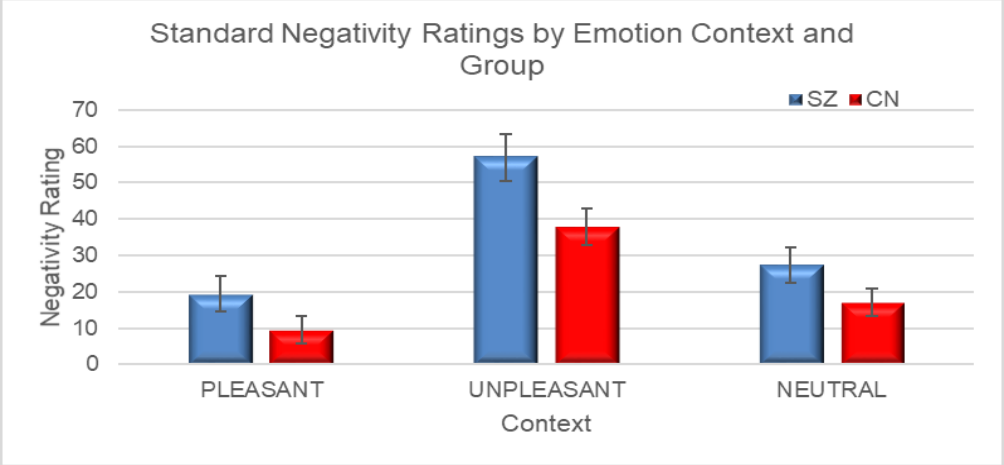
	Within subjects (Context)	Between Subjects (Group)	Interaction (Context x Group)
Positive Affect	$F(2, 48) = 40.63$ $p < .001$, $\eta_p^2 = 0.63$	$F(1, 24) = 1.83$ $p = .19$, $\eta_p^2 = 0.07$	$F(2, 48) = .65$ $p = .49$, $\eta_p^2 = 0.03$
Negative Affect	$F(2, 48) = 45.33$ $p < .001$, $\eta_p^2 = 0.65$	$F(1, 24) = 5.69$ $p = .03$, $\eta_p^2 = 0.19$	$F(2, 48) = 1.04$ $p = .33$, $\eta_p^2 = 0.04$
Arousal	$F(2, 56) = 7.87$ $p = .001$, $\eta_p^2 = 0.22$	$F(1, 28) = .02$ $p = .88$, $\eta_p^2 = 0.001$	$F(2, 56) = 1.30$ $p = .28$, $\eta_p^2 = 0.04$

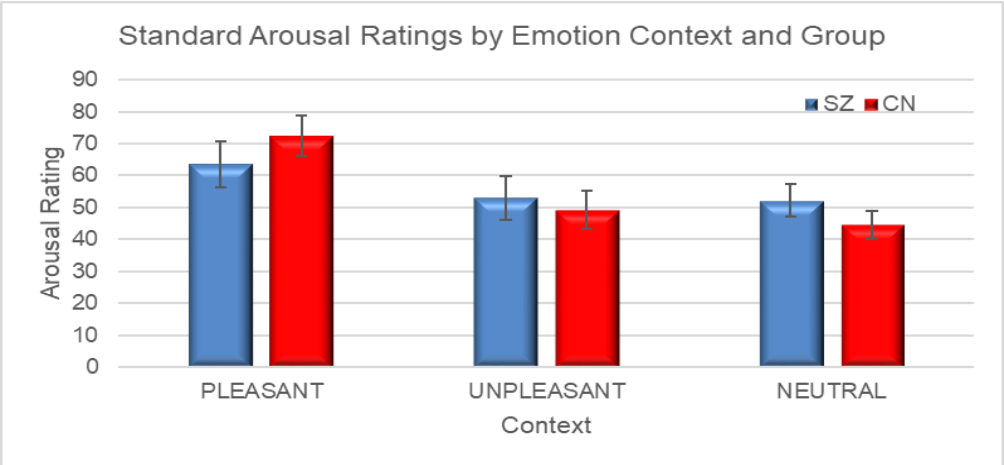
Note. Context = positive, negative, or neutral momentary emotional context. Group = SZ or CN.

Supplemental Figure 1

Positivity, Negativity, and Arousal Ratings by Group and Emotion Context







Note. SZ = Schizophrenia group; CN = Control group.

Supplemental Table 2

Univariate ANOVA Results Examining the Effects of Sex and Group on Positivity and Negativity Parameters

	Between subjects (Sex)	Between Subjects (Group)	Interaction (Sex x Group)	Post-hoc
Positivity Intercept	$F(1, 92) = .02$ $p = .90, \eta_p^2 = 0$	$F(1, 92) = .55$ $p = .46, \eta_p^2 = .01$	$F(1, 92) = 3.77$ $p = .06, \eta_p^2 = .04$	-
Negativity Intercept	$F(1, 92) = .06$ $p = .81, \eta_p^2 = .001$	$F(1, 92) = 9.90$ $p = .002, \eta_p^2 = .10$	$F(1, 92) = 2.99$ $p = .09, \eta_p^2 = .03$	-
Positivity Slope	$F(1, 92) = .69$ $p = .41, \eta_p^2 = .01$	$F(1, 92) = 2.75$ $p = .10, \eta_p^2 = .03$	$F(1, 92) = 1.99$ $p = .16, \eta_p^2 = .02$	-
Negativity Slope	$F(1, 92) = .09$ $p = .76, \eta_p^2 = .001$	$F(1, 92) = 1.91$ $p = .17, \eta_p^2 = .02$	$F(1, 92) = .32$ $p = .58, \eta_p^2 = .004$	-
Positivity Offset	$F(1, 92) = .004$ $p = .95, \eta_p^2 = 0$	$F(1, 92) = 5.31$ $p = .02, \eta_p^2 = .06$	$F(1, 92) = 4.74$ $p = .03, \eta_p^2 = 0.05$	-
Negativity Bias	($F(1, 92) = .57$ $p = .45, \eta_p^2 = .01$	$F(1, 24) = 5.69$ $p = .03, \eta_p^2 = 0.19$	($F(1, 92) = 1.72$ $p = .19, \eta_p^2 = .02$)	-

Note. Sex = male (M) or female (F). Group = SZ or CN.

Supplemental Table 3

One-way ANOVAs Comparing Positivity and Negativity Parameters in Clinical and Control Groups

	SZ (n=18)	SZaff (n=26)	CN (n=48)	Test Statistic	Post-hoc
Positivity Intercept	46.12 (20.25)	44.87 (28.00)	52.91 (21.06)	$F(2.\ 92) = 1.24$ $p = .30$, $\eta_p^2 = .03$	-
Negativity Intercept	35.11 (32.73)	27.16 (28.61)	11.19 (12.60)	$F(2, 92) = 8.82$ $p < .001$, $\eta_p^2 = .17$	SZaff, SZ > CN
Positivity Slope	.11 (.49)	-.33 (1.30)	.06 (.26)	$F(2, 92) = 2.80$ $p = .07$, $\eta_p^2 = .06$	-
Negativity Slope	.45 (1.73)	.18 (1.10)	.03 (.19)	$F(2, 92) = 1.26$ $p = .29$, $\eta_p^2 = .03$	-
Positivity Offset	11.01 (40.65)	17.71 (51.01)	41.72 (30.07)	$F(2, 92) = 5.54$ $p = .01$, $\eta_p^2 = .11$	SZaff, SZ < CN
Negativity Bias	.33 (1.84)	.52 (1.69)	-.03 (.40)	$F(2, 92) = 1.79$ $p = .17$, $\eta_p^2 = .04$	-

Note. SZ = Schizophrenia group; SZaff = Schizoaffective group; CN = Control group. Positivity Offset = Positivity Intercept – Negativity Intercept. Negativity Bias = Negativity Slope – Positivity Slope. Values reflect Mean (SD) unless otherwise indicated.

APPENDEX B

CHAPTER 3 SUPPLEMENTAL MATERIAL

Supplemental Demographic and Clinical Participant Information on Mood-based Subgroups

Study 1

Supplemental analyses on mood-based subgroups included participants with schizophrenia (SZ; n = 62) and schizoaffective disorder (SZaff = 36). Both SZ and SZaff subgroups had lower personal education compared to CN, but group differences in education between SZ and SZaff were nonsignificant. Individuals with SZ had significantly lower cognitive performance than both SZaff and CN, and individuals with SZaff also had lower cognitive performance than CN. SZ and SZaff subgroups were matched on age, parental education, sex, race, and medication. Individuals with SZ had significantly more severe alogia than individuals with SZaff. See Supplemental Table 1.

Study 2

Supplemental analyses on mood-based subgroups included participants at CHR for psychosis with (CHR-M; n = 17) and without comorbid mood disorder diagnoses (CHR-NM; n = 28). Groups (CHR-M, CHR-NM, CN and CHR-M vs CHR-NM) were matched on all demographic variables and clinically rated negative symptoms. See Supplemental Table 1.

Supplemental Data Analysis

Standard preliminary analyses of self-reported emotional experience variables were conducted similar to past studies comparing subjective positivity, negativity, and arousal to

pleasant, unpleasant, and neutral stimuli. The average level of positive affect, negative affect, and arousal were calculated for each participant using ratings collected from the emotional experience task and used as the dependent variables for separate Condition (Pleasant, Unpleasant, Neutral) X Group (SZ+SZaff, CN or CHR-M+CHR-NM, CN; SZ, SZaff, CN or CHR-M, CHR-NM, CN) mixed models ANOVAs.

Exploratory analyses consisted of conducting univariate ANOVAs to examine the effects of Sex (Male, Female), Group (SZ+SZaff, CN or CHR-M+CHR-NM, CN; SZ, SZaff, CN or CHR-M, CHR-NM, CN) and the Sex X Group interaction on the positivity offset and negativity bias difference scores. To examine medication effects in individuals with full-psychotic disorders (SZ+SZaff) and mood-based subgroups (SZ, SZaff), separate point-biserial correlations were conducted to examine the association between medication status (i.e., whether a participant was prescribed antipsychotics, coded as No = 0 and Yes = 1) and positivity offset and negativity bias difference scores. Bivariate (Spearman) correlations were also conducted with the positive offset and negativity bias difference scores and MCCB overall scores to examine associations with cognition among individuals with full-psychotic disorders (SZ+SZaff) and mood-based subgroups (SZ, SZaff). All analyses using the positivity offset and negativity bias difference scores were repeated with the raw positivity and negativity intercepts and slopes as the dependent variable.

All primary analyses conducted in the main manuscript within the overall SZ and CHR groups were repeated after splitting groups into affective vs nonaffective subgroups. The SZ group was split into individuals with schizophrenia (SZ) and schizoaffective disorder (SZaff). The CHR group was split into individuals at CHR for psychosis with (CHR-M) and without a co-morbid mood disorder (CHR-NM).

Supplemental Results

Study 1

Preliminary Analyses in SZ+SZaff and CN. Standard analyses of self-reported levels of positivity, negativity, and arousal in response to pleasant, unpleasant, and neutral stimuli were compared between groups. The SZ+SZaff group endorsed significantly greater negative emotion in response to pleasant and neutral stimuli compared to CN. There was evidence for hedonic deficits in the SZ+SZaff group, such that participants endorsed lower levels of positive emotion in response to pleasant stimuli compared to CN. Additionally, participants in the SZ+SZaff group endorsed greater levels of negative emotion in response to pleasant stimuli than CN. See Supplemental Table 2 and Supplemental Figure 2 for results.

Preliminary Analyses in SZ, SZaff, and CN. When mood-based subgroups and CN were compared on self-reported levels of positivity, negativity, and arousal in response to pleasant, unpleasant, and neutral stimuli, results for the standard negativity ratings indicated significant main effects of Condition and Group, while the Condition X Group interaction was nonsignificant. For standard positivity ratings, the effects of Condition, Group, and the Condition X Group interaction were significant. Follow-up post-hoc analyses indicated that participants with SZaff endorsed significantly less positive emotion in response to neutral and pleasant images compared to CN, as well as less positive emotion in response to pleasant and unpleasant stimuli compared to SZ participants. CN also endorsed significantly less positive emotion than SZ in response to unpleasant stimuli. This suggests that when using standard positivity ratings, hedonic capacity is intact in SZ but not SZaff. Additionally, these results indicate that greater coactivation of positive and negative emotion in response to unpleasant stimuli may be specific to nonaffective psychosis. Lastly, analyses of standard arousal ratings indicated a significant

main effect of Condition while the effect of Group and the Condition X Group interaction were nonsignificant. See Supplemental Table 2 and Supplemental Figure 1.

Exploratory Analyses in SZ+SZaff and CN. For the positivity offset difference score, univariate ANOVA revealed a significant main effect of Group, a nonsignificant effect of Sex, and a nonsignificant Group X Sex interaction. For the negativity bias difference score, the effects of Group, Sex, and the Group X Sex interaction were nonsignificant. All main effects and interactions were nonsignificant when using the raw positivity and negativity parameters. See Supplemental Table 3. Among participants in the SZ+SZaff group, correlations between medication status and the positivity offset (r = -.06, p = .59) and negativity bias (r = -.07, p = .51) were nonsignificant.

Exploratory Analyses in SZ, SZaff, and CN. Among participants with SZ and SZaff, correlations with medication status and the positivity offset (SZ: r = -.09, p = .55; SZaff: r = -.15, p = .39) and negativity bias (SZ: r = -.05, p = .75; SZaff: r = -.23, p = .19) difference scores were nonsignificant. This was also the case when using the raw positivity and negativity parameters. In both the SZ and SZaff group, correlations between overall MCCB scores and the positivity offset (SZ: r = .10, p = .51; SZaff: r = .32, p = .06) and negativity bias (SZ: r = -.05, p = .73; SZaff: r = .29, p = .09) difference scores were nonsignificant, as were correlations with the raw positivity and negativity parameters.

Repeated Primary Analyses with SZ and SZaff Subgroups. CN (t = 3.40, p = .001) and SZ (t = 3.42 p = .001) demonstrated the prototypical positivity offset, with significantly higher intercepts for positivity than negativity. In contrast, the positivity offset was not detected among individuals with SZaff, who demonstrated nonsignificant differences between positivity and negativity intercepts (t = -.52, p = .61). The positivity offset difference score was significantly

reduced in SZaff compared to CN, while group differences in the positivity offset between SZ versus SZaff and SZ versus CN were nonsignificant. The raw positivity intercept was significantly reduced in SZaff compared to SZ, whereas group differences in the raw positivity slope were nonsignificant. CN (t = -2.88, p = .01) and SZ (t = -3.16, p = .002) also demonstrated the prototypical negativity bias, evidenced by a significantly greater slope for negativity than positivity. SZaff participants displayed nonsignificant differences between slopes for the positivity and negativity functions, suggesting a lack of negativity bias (t = .38, p = .71). Groups did not significantly differ on negativity bias difference scores or any of the raw negativity parameters. See Supplemental Table 4 for results of group comparisons and Supplemental Figure 3 for regression equations depicting the positivity and negativity functions.

In SZ, correlations between negative symptoms and the positivity offset (avolition: r = -.21, p = .11; anhedonia: r = -.15, p = .25) and negativity bias difference scores (avolition: r = -.13, p = .31; anhedonia: r = -.06, p = .65) were nonsignificant. Also in the SZ group, lower raw positivity intercepts (r = -.39, p = .003) and higher raw positivity slopes (r = .32, p = .01) were associated with more severe avolition, while correlations with the raw negativity parameters were nonsignificant. Among individuals with SZaff, lower positivity offset difference scores were associated with greater avolition (r = -.49, p = .004) and anhedonia (r = -.37, p = .04), while lower negativity bias difference scores were associated with more severe avolition (r = -.44, p = .01). In SZaff, correlations between the raw positivity and negativity parameters with clinically rated anhedonia and avolition were nonsignificant. Lastly, greater reductions in the positivity offset difference score were significantly correlated with greater depression symptoms in the SZaff group (r = -.38, p = .03). None of the positivity or negativity parameters were associated with depression in the SZ group.

Study 2

Preliminary Analyses in CHR-M+CHR-NM and CN. Standard analyses of self-reported levels of positivity, negativity, and arousal in response to pleasant, unpleasant, and neutral stimuli were compared between groups. CHR-M+CHR-NM endorsed significantly less negative emotion in response to unpleasant stimuli compared to CN. Similar to the current sample of adults with psychotic disorders, CHR-M+CHR-NM participants also endorsed significantly lower levels of positive emotion in response to pleasant stimuli compared to CN (see Supplemental Table 4 and Supplemental Figure 5).

Preliminary Analyses in CHR-M, CHR-NM, and CN. For standard negativity, positivity, and arousal ratings, there were significant main effects of Condition, nonsignificant effects of Group, and nonsignificant Group X Condition interactions. Together, these findings indicate intact hedonic capacity in both CHR groups relative to CN. See Supplemental Table 2 and Supplemental Figure 4 for results.

Exploratory Analyses in CHR-M+CHR-NM and CN. Univariate ANOVA indicated nonsignificant effects of Group, Sex, and a nonsignificant Group X Sex interaction on the positivity offset and negativity bias difference scores. When using the raw positivity intercept, there was a significant Group X Sex interaction. Follow-up post-hoc one-way ANOVAs indicated that within the CHR-M+CHR-NM group, females had lower raw positivity intercepts than males, while sexes did not significantly differ in CN. Within female participants, those in the CHR-M+CHR-NM group demonstrated lower raw positivity intercepts than CN, whereas groups did not differ within male participants. There was also a significant main effect of Sex on the Negativity Intercept, while all other effects on the raw positivity and negativity parameters were nonsignificant (See Supplemental Table 3).

Exploratory Analyses in CHR-M, CHR-NM, and CN. For the positivity offset and negativity bias difference scores, univariate ANOVA indicated nonsignificant main effects of Group and Sex and a nonsignificant Group X Sex interaction. When using the raw negativity intercept and slope, the main effects of Sex were significant and the main effects of Group and the Group X Sex interactions were nonsignificant. None of the effects were significant when using the raw positivity parameters (see Supplemental Table 3).

Repeated Primary Analyses with CHR-M, CHR-NM, and CN. All groups demonstrated the prototypical positivity offset, with significantly higher intercepts for positivity than negativity (CN: $t = 6.08$, $p < .001$; CHR-NM: $t = 3.26$, $p = .003$; CHR-M: $t = 4.07$, $p = .001$). Group differences in the positivity offset difference score were nonsignificant, as were differences in the raw positivity slope and intercept. All groups also demonstrated the negativity bias, with significantly higher slopes for negativity than positivity (CN: $t = -5.40$, $p < .001$; CHR-NM: $t = -2.83$, $p = .01$; CHR-M: $t = -3.48$, $p = .003$). Group differences in negativity bias difference scores and the raw negativity parameters were nonsignificant. See Supplemental Table 3 for results of group comparison sand Supplemental Figure 6 for regression equations depicting the positivity and negativity functions.

In the CHR-NM group, correlations between negative symptoms and the positivity offset (avolition: $r = .08$, $p = .67$; anhedonia: $r = .23$, $p = .24$) and negativity bias difference scores (avolition: $r = .16$, $p = .42$; anhedonia: $r = .30$, $p = .65$) were nonsignificant. Additionally, lower raw positivity slopes were associated with more severe anhedonia ($r = -.45$, $p = .02$), while all other correlations with raw parameters were nonsignificant. Among CHR-M participants, more severe anhedonia was associated with smaller positivity offset difference scores ($r = -.68$, $p = .003$), smaller negativity bias difference scores ($r = -.71$, $p = .002$), larger raw negativity

intercepts (r = .69, p = .002), and smaller raw negativity slopes (r = -.68, p = .002). In both CHR-NM and CHR-M groups, correlations between cross-sectional conversion risk and depressive symptoms with the positivity offset, negativity bias, and raw positivity and negativity parameters were nonsignificant.

Supplemental Table 1

Participant demographics

	Study 1			
	SZ (n=62)	SZaff (n=36)	CN (n=84)	Test statistic
Age	39.37 (12.17)	40.00 (12.02)	39.77 (11.47)	F = .04
Parental Education	13.26 (2.58)	14.14 (2.87)	13.62 (2.44)	F = .30
Personal Education	12.64 (2.10)	13.29 (2.42)	15.74 (2.83)	F = 22.86*** SZ, SZaff < CN
Female (%)	41.90	61.10	60.70	χ^2 = 5.89
Race (%)	-	-	-	χ^2 = 9.04
Black	14.00	19.40	16.70	-
Asian	1.60	0.00	6.00	-
LatinX	2.00	2.80	9.50	-
White	64.50	69.40	60.70	-
Multiracial	6.50	8.30	4.80	-
Other	1.60	2.38	2.40	-
Medication (n)				
Antipsychotic	29	21	-	-
Mood Stabilizer	10	11	-	-
Antidepressant	20	15	-	-
Anxiolytic	13	8	-	-
Stimulant	2	3	-	-
None	10	8	-	-
MCCB	34.28 (12.53)	42.29 (13.02)	50.29 (10.82)	F = 28.12*** SZ < SZaff < CN
BNSS Total	18.78 (15.33)	16.38 (14.51)	-	F = .53
Avolition	2.10 (1.82)	2.08 (1.73)	-	F = .004
Anhedonia	1.55 (1.43)	1.85 (1.80)	-	F = .74
Asociality	1.57 (1.40)	1.44 (1.56)	-	F = .16
Alogia	.94 (1.54)	.30 (.68)	-	F = 5.02*
Blunted Affect	1.45 (1.78)	1.20 (1.46)	-	F = .47
	Study 2			

	CHR-NM (n=28)	CHR-M (n=17)	CN (n=51)	Test statistic
Age	20.14 (2.29)	20.76 (2.82)	20.22 (1.94)	F = .48
Parental Education	14.82 (2.34)	15.53 (2.75)	15.61 (2.29)	F = 1.03
Personal Education	13.36 (1.81)	13.94 (1.52)	14.00 (1.54)	F = 1.50
Female (%)	75.00	76.50	80.39	χ^2 = .34
Race (%)	-	-	-	χ^2 = 5.36
Black	7.10	5.90	3.92	-
Asian	17.90	5.90	17.65	-
LatinX	10.70	11.80	5.89	-
White	60.70	76.50	72.55	-
Multiracial	3.60	0.00	0.00	-
Medication (n)				
Antipsychotic	1	1	-	-
Mood Stabilizer	1	1	-	-
Antidepressant	3	6	-	-
Anxiolytic	2	0	-	-
Stimulant	0	1	-	-
None	22	10	-	-
BNSS Total	12.29 (11.47)	13.94 (12.64)	-	F = .20
Avolition	1.16 (1.36)	1.44 (1.33)	-	F = .46
Anhedonia	1.50 (1.28)	1.78 (1.62)	-	F = .43
Asociality	.68 (1.20)	1.21 (1.03)	-	F = 2.25
Alogia	.61 (1.14)	.29 (.99)	-	F = .88
Blunted Affect	.87 (1.43)	.86 (1.39)	-	F = 0.00

Note. SZ = individuals with SZ; SZaff = individuals with schizoaffective disorder; CHR-NM = individuals clinical high-risk without comorbid mood disorders; CHR-M = individuals with clinical high-risk with comorbid mood disorders; 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. *$p < .05$, ***$p < .001$.

Supplemental Table 2

Repeated Measures ANOVA Results for Standard Analyses of Valence and Arousal

Condition	Within subjects (Condition)	Between Subjects (Group)	Interaction (Condition X Group)	Post hoc
Study 1: Comparing SZ, SZaff, and CN				
Negativity	$F(2, 358) = 654.75$ $p < .001$, $\eta_p^2 = .79$	$F(2, 179) = 3.64$ $p = .03$, $\eta_p^2 = .04$	$F(4, 358) = 1.67$ $p = .18$, $\eta_p^2 = .02$	-
Positivity	$F(2, 358) = 442.66$ $p < .001$, $\eta_p^2 = .71$	$F(2, 179) = 7.53$ $p = .001$, $\eta_p^2 = .08$	$F(4, 358) = 3.73$ $p = .01$, $\eta_p^2 = .04$	Neutral: SZaff < CN Pleasant: SZaff < CN, SZ Unpleasant: SZaff, CN < SZ
Arousal	$F(2, 358) = 107.43$ $p < .001$, $\eta_p^2 = .38$	$F(2, 179) = 2.00$ $p = .14$, $\eta_p^2 = .02$	$F(4, 358) = .38$ $p = .81$, $\eta_p^2 = .004$	-
Study 1: Comparing SZ+SZaff and CN				
Negativity	$F(2, 360) = 758.33$ $p < .001$, $\eta_p^2 = .81$	$F(1, 180) = 7.01$ $p = .01$, $\eta_p^2 = .04$	$F(2, 360) = 2.49$ $p = .10$, $\eta_p^2 = .01$	-
Positivity	$F(2, 360) = 544.70$ $p < .001$, $\eta_p^2 = .75$	$F(1, 180) = 1.03$ $p = .31$, $\eta_p^2 = .01$	$F(2, 360) = 5.83$ $p = .003$, $\eta_p^2 = .03$	Pleasant: SZ+SZaff < CN Unpleasant: SZ+SZaff > CN
Arousal	$F(2, 360) = 115.37$ $p < .001$, $\eta_p^2 = .39$	$F(1, 180) = .96$ $p = .33$, $\eta_p^2 = .01$	$F(2, 360) = .10$ $p = .90$, $\eta_p^2 = .001$	-
Study 2: Comparing CHR, CHR-M, and CN				
Negativity	$F(2, 186) = 713.85$ $p < .001$, $\eta_p^2 = .89$	$F(2, 93) = 2.82$ $p = .07$, $\eta_p^2 = .06$	$F(4, 186) = 2.35$ $p = .07$, $\eta_p^2 = .05$	-
Positivity	$F(2, 186) = 542.24$ $p < .001$, $\eta_p^2 = .85$	$F(2, 93) = .62$ $p = .54$, $\eta_p^2 = .01$	$F(4, 186) = 2.05$ $p = .09$, $\eta_p^2 = .04$	-
Arousal	$F(2, 186) = 141.46$ $p < .001$, $\eta_p^2 = .60$	$F(2, 93) = 2.23$ $p = .11$, $\eta_p^2 = .05$	$F(4, 186) = .42$ $p = .79$, $\eta_p^2 = .01$	-
Study 2: Comparing CHR+CHR-M and CN				

Negativity	$F(2, 188) = 921.06$ $p < .001$, $\eta_p^2 = .91$	$F(1, 94) = 1.24$ $p = .27$, $\eta_p^2 = .01$	$F(2, 188) = 4.67$ $p = .02$, $\eta_p^2 = .05$	Unpleasant: CHR-NM+CHR-M < CN
Positivity	$F(2, 188) = 697.01$ $p < .001$, $\eta_p^2 = .88$	$F(1, 94) = .53$ $p = .47$, $\eta_p^2 = .01$	$F(2, 188) = 4.11$ $p = .02$, $\eta_p^2 = .04$	Pleasant: CHR-NM+CHR-M < CN
Arousal	$F(2, 188) = 177.59$ $p < .001$, $\eta_p^2 = .65$	$F(1, 94) = 1.81$ $p = .18$, $\eta_p^2 = .02$	$F(2, 188) = .56$ $p = .57$, $\eta_p^2 = .01$	-

Note. SZ = individuals with SZ; SZaff = individuals with schizoaffective disorder; CHR-NM = individuals clinical high-risk without comorbid mood disorders; CHR-M = individuals with clinical high-risk with comorbid mood disorders; CN = control group

Supplemental Table 3

Univariate ANOVA Results Examining the Effects of Sex and Group on Positivity and Negativity Parameters

	Between subjects (Sex)	Between subjects (Group)	Interaction (Sex x Group)	Post-hoc
		Study 1: Comparing SZ, SZaff, and CN		
Positivity Intercept	$F(1, 182) = .92$ $p = .34, \eta_p^2 = .01$	$F(2, 182) = 2.53$ $p = .08, \eta_p^2 = .03$	$F(2, 182) = .48$ $p = .62, \eta_p^2 = .01$	-
Negativity Intercept	$F(1, 182) = .89$ $p = .35, \eta_p^2 = .01$	$F(2, 182) = 1.55$ $p = .22, \eta_p^2 = .02$	$F(2, 182) = .55$ $p = .58, \eta_p^2 = .01$	-
Positivity Slope	$F(1, 182) = 1.28$ $p = .26, \eta_p^2 = .01$	$F(2, 182) = 1.72$ $p = .18, \eta_p^2 = .02$	$F(2, 182) = .53$ $p = .59, \eta_p^2 = .01$	-
Negativity Slope	$F(1, 182) = 1.08$ $p = .30, \eta_p^2 = .01$	$F(2, 182) = 1.20$ $p = .30, \eta_p^2 = .01$	$F(2, 182) = .65$ $p = .52, \eta_p^2 = .01$	-
Positivity Offset	$F(1, 182) = .03$ $p = .86, \eta_p^2 = 0$	$F(2, 182) = 3.55$ $p = .03, \eta_p^2 = .04$	$F(2, 182) = .07$ $p = .93, \eta_p^2 = .001$	-
Negativity Bias	$F(1, 182) = .02$ $p = .89, \eta_p^2 = 0$	$F(2, 182) = 2.56$ $p = .08, \eta_p^2 = .03$	$F(2, 182) = .12$ $p = .89, \eta_p^2 = .001$	-
		Study 1: Comparing SZ+SZaff and CN		
Positivity Intercept	$F(1, 182) = 1.52$ $p = .22, \eta_p^2 = .01$	$F(1, 182) = 0$ $p = .99, \eta_p^2 = 0$	$F(1, 182) = .97$ $p = .33, \eta_p^2 = .01$	-
Negativity Intercept	$F(1, 182) = .78$ $p = .38, \eta_p^2 = .004$	$F(1, 182) = 3.26$ $p = .07, \eta_p^2 = .02$	$F(1, 182) = .92$ $p = .34, \eta_p^2 = .01$	-
Positivity Slope	$F(1, 182) = 1.70$ $p = .19, \eta_p^2 = .01$	$F(1, 182) = .01$ $p = .92, \eta_p^2 = 0$	$F(1, 182) = 1.15$ $p = .29, \eta_p^2 = .01$	-
Negativity Slope	$F(1, 182) = .75$ $p = .39, \eta_p^2 = .004$	$F(1, 182) = 2.54$ $p = .11, \eta_p^2 = .01$	$F(1, 182) = 1.28$ $p = .26, \eta_p^2 = .01$	-
Positivity Offset	$F(1, 182) = .02$ $p = .88, \eta_p^2 = 0$	$F(1, 182) = 4.33$ $p = .04, \eta_p^2 = .02$	$F(1, 182) = .03$ $p = .87, \eta_p^2 = 0$	-

Negativity Bias	$F(1, 182) = .06$ $p = .81, \eta_p^2 = 0$	$F(1, 182) = 2.97$ $p = .09, \eta_p^2 = .02$	$F(1, 182) = .08$ $p = .78, \eta_p^2 = 0$	-
Study 2: Comparing CHR-M, CHR-NM, and CN				
Positivity Intercept	$F(1, 96) = 2.54$ $p = .11, \eta_p^2 = .03$	$F(2, 96) = .30$ $p = .74, \eta_p^2 = .01$	$F(2, 96) = 2.55$ $p = .08, \eta_p^2 = .05$	-
Negativity Intercept	$F(1, 96) = 7.76$ $p = .01, \eta_p^2 = .08$	$F(2, 96) = 1.11$ $p = .34, \eta_p^2 = .02$	$F(2, 96) = .43$ $p = .65, \eta_p^2 = .01$	-
Positivity Slope	$F(1, 96) = .22$ $p = .64, \eta_p^2 = .002$	$F(2, 96) = .63$ $p = .54, \eta_p^2 = .01$	$F(2, 96) = 1.94$ $p = .15, \eta_p^2 = .04$	-
Negativity Slope	$F(1, 96) = 4.93$ $p = .03, \eta_p^2 = .05$	$F(2, 96) = 1.74$ $p = .18, \eta_p^2 = .04$	$F(2, 96) = .64$ $p = .53, \eta_p^2 = .01$	
Positivity Offset	$F(1, 96) = 1.38$ $p = .24, \eta_p^2 = .02$	$F(2, 96) = .30$ $p = .74, \eta_p^2 = .01$	$F(2, 96) = .39$ $p = .68, \eta_p^2 = .01$	-
Negativity Bias	$F(1, 96) = 1.67$ $p = .20, \eta_p^2 = .02$	$F(2, 96) = .40$ $p = .67, \eta_p^2 = .01$	$F(2, 96) = .38$ $p = .69, \eta_p^2 = .01$	-
Study 2: Comparing CHR-M+CHR-NM and CN				
Positivity Intercept	$F(1, 96) = .64$ $p = .43, \eta_p^2 = .01$	$F(1, 96) = .02$ $p = .89, \eta_p^2 = 0$	$F(1, 96) = 4.05$ $p = .047, \eta_p^2 = .04$	CHR-M+CHR-NM: F < M CN: F = M F: CHR-M+CHR-NM < CN M: CHR-M+CHR-NM = CN
Negativity Intercept	$F(1, 96) = 6.93$ $p = .01, \eta_p^2 = .07$	$F(1, 96) = .88$ $p = .35, \eta_p^2 = .01$	$F(1, 96) = .28$ $p = .60, \eta_p^2 = .003$	-
Positivity Slope	$F(1, 96) = .07$ $p = .79, \eta_p^2 = .001$	$F(1, 96) = .15$ $p = .70, \eta_p^2 = .002$	$F(1, 96) = 3.43$ $p = .07, \eta_p^2 = .04$	-
Negativity Slope	$F(1, 96) = 3.91$ $p = .05, \eta_p^2 = .04$	$F(1, 96) = 2.45$ $p = .12, \eta_p^2 = .03$	$F(1, 96) = .22$ $p = .64, \eta_p^2 = .002$	-

Positivity Offset	$F(1, 96) = 2.55$ $p = .11, \eta_p^2 = .03$	$F(1, 96) = .44$ $p = .51, \eta_p^2 = .01$	$F(1, 96) = .79$ $p = .38, \eta_p^2 = .01$	-
Negativity Bias	$F(1, 96) = .2.57$ $p = .11, \eta_p^2 = .03$	$F(1, 96) = .80$ $p = .37, \eta_p^2 = .01$	$F(1, 96) = .66$ $p = .42, \eta_p^2 = .01$	-

Note. Positivity Offset = Positivity Intercept – Negativity Intercept; Negativity Bias = Negativity Slope – Positivity Slope. SZ = individuals with SZ; SZaff = individuals with schizoaffective disorder; CHR-NM = individuals clinical high-risk without comorbid mood disorders; CHR-M = individuals with clinical high-risk with comorbid mood disorders; CN = control group

Supplemental Table 4

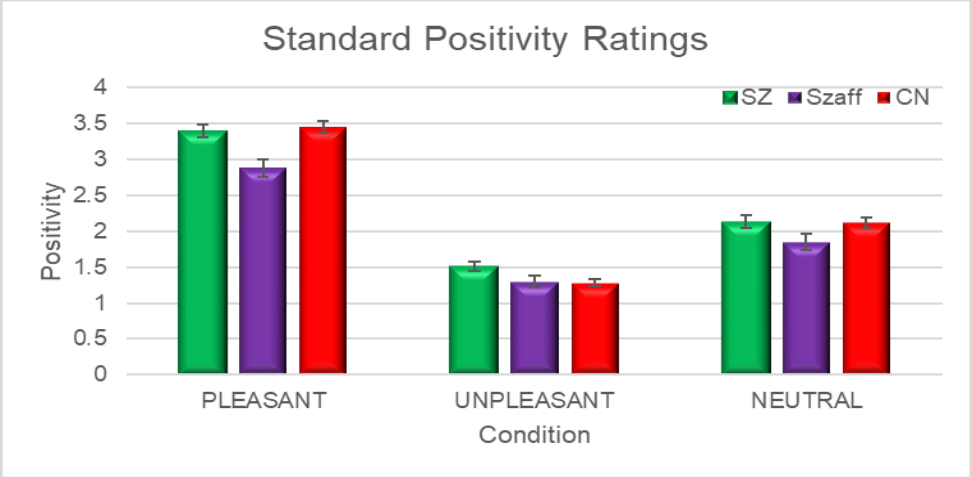
One-way ANOVAs Comparing Positivity and Negativity Parameters Between Groups

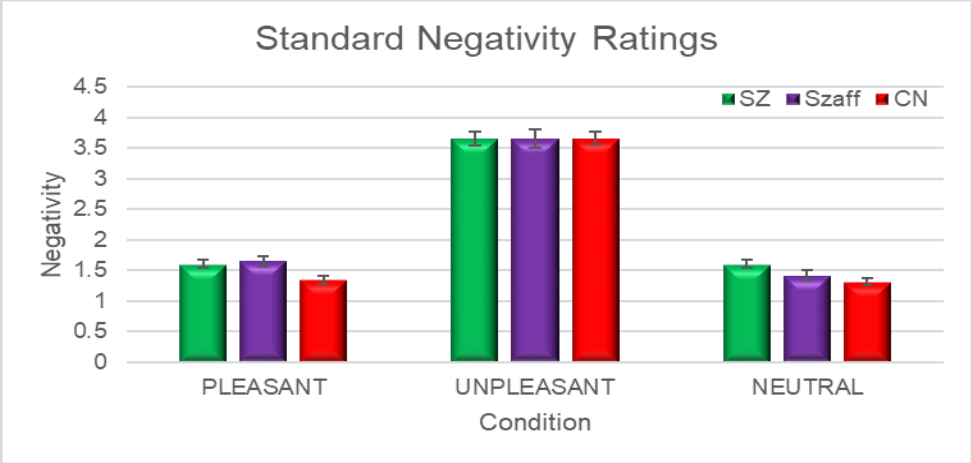
Study 1					
	SZ (n=62)	SZaff (n=36)	CN (n=84)	Test Statistic	Post-hoc
Positivity Intercept	3.16 (10.38)	-.34 (6.18)	1.83 (1.84)	$F(1, 182) = 3.06$ $p = .049$, $\eta_p^2 = .03$	SZaff < SZ
Negativity Intercept	1.28 (9.64)	.28 (3.57)	-1.35 (8.66)	$F(1, 182) = 1.85$ $p = .16$, $\eta_p^2 = .02$	-
Positivity Slope	-.11 (3.45)	.88 (2.00)	.30 (.67)	$F(1, 182) = 2.24$ $p = .11$, $\eta_p^2 = .02$	-
Negativity Slope	.45 (3.20)	.73 (1.18)	1.21 (2.87)	$F(1, 182) = 1.39$ $p = .25$, $\eta_p^2 = .02$	-
Positivity Offset	1.88 (4.34)	-.63 (7.29)	3.19 (8.60)	$F(2, 182) = 3.60$ $p = .03$, $\eta_p^2 = .04$	SZaff < CN
Negativity Bias	.57 (1.42)	-.15 (2.40)	.91 (2.88)	$F(1, 182) = 2.50$ $p = .09$, $\eta_p^2 = .03$	-
Study 2					
	CHR-NM (n=28)	CHR-M (n=17)	CN (n=51)	Test Statistic	Post-hoc
Positivity Intercept	1.44 (1.31)	.68 (1.76)	1.53 (1.71)	$F(1, 96) = 1.80$ $p = .17$, $\eta_p^2 = .04$	-
Negativity Intercept	-.01 (1.91)	-1.10 (2.04)	-.80 (12.60)	$F(1, 96) = 2.01$ $p = .14$, $\eta_p^2 = .04$	-
Positivity Slope	.39 (.40)	.65 (.43)	.41 (.57)	$F(1, 96) = 1.68$ $p = .19$, $\eta_p^2 = .04$	-
Negativity Slope	.79 (.56)	1.11 (.59)	1.11 (.61)	$F(1, 96) = 2.96$ $p = .06$, $\eta_p^2 = .06$	-
Positivity Offset	1.44 (2.35)	1.79 (1.81)	2.32 (2.73)	$F(1, 96) = 1.19$ $p = .31$, $\eta_p^2 = .03$	-
Negativity Bias	.39 (.74)	.45 (.54)	.70 (.93)	$F(1, 96) = 1.49$ $p = .23$, $\eta_p^2 = .03$	-

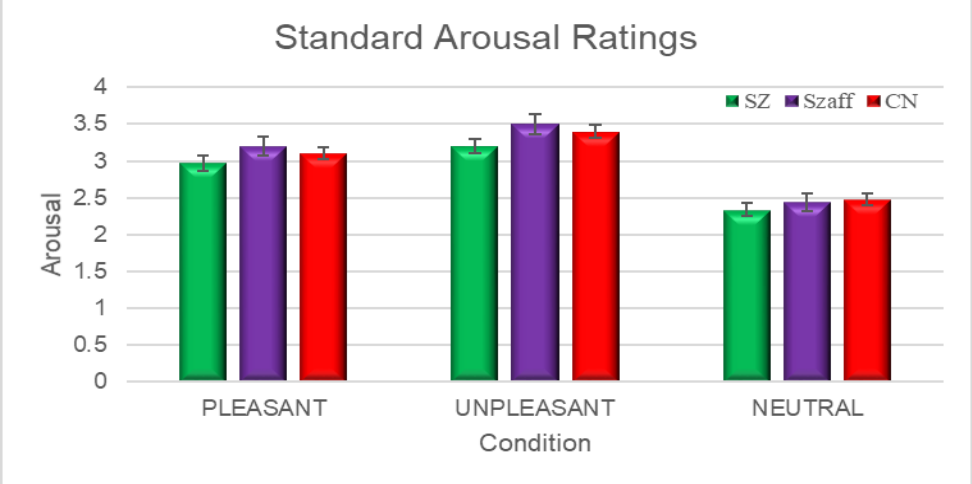
Note. SZ = Schizophrenia group; SZaff = Schizoaffective group; CN = Control group; CHR-NM = clinical high-risk without a comorbid mood disorder diagnosis; CHR-M = clinical high-risk with a comorbid mood disorder diagnosis. Positivity Offset = Positivity Intercept – Negativity

Intercept. Negativity Bias = Negativity Slope – Positivity Slope. Values reflect Mean (SD) unless otherwise indicated.

Supplemental Figure 1

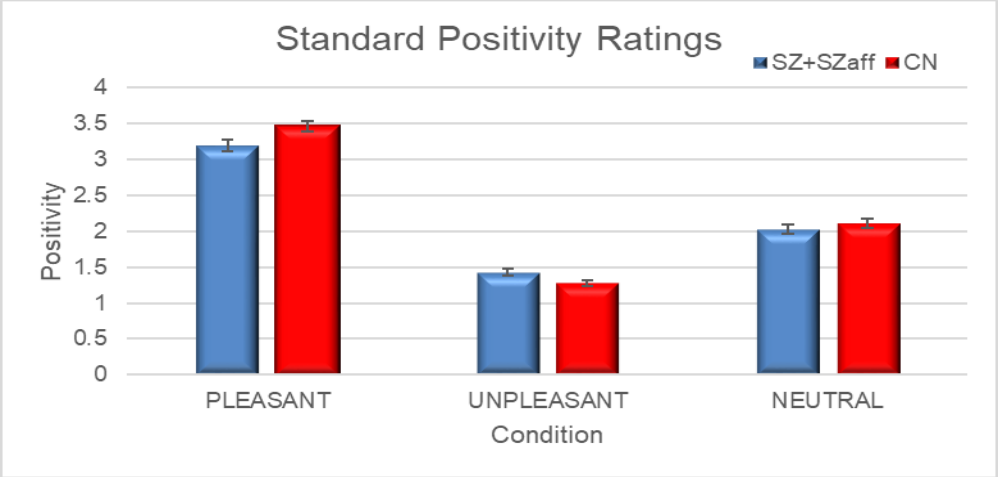


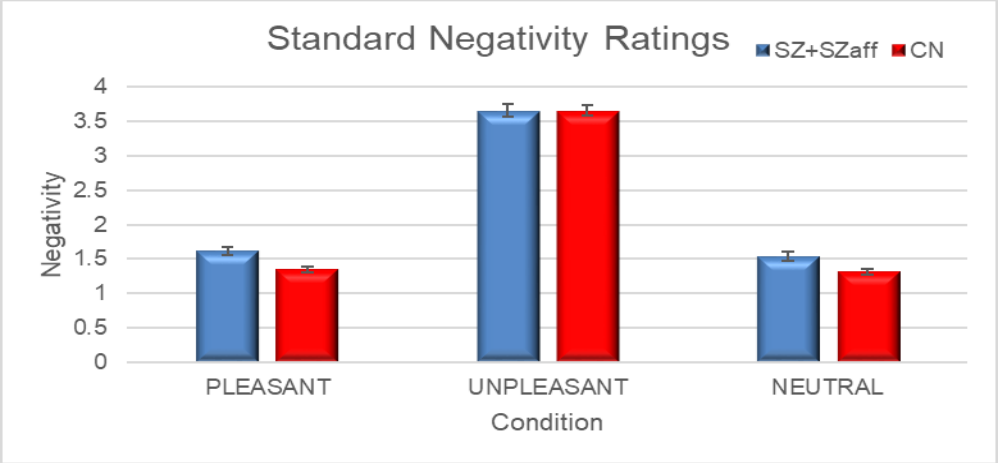


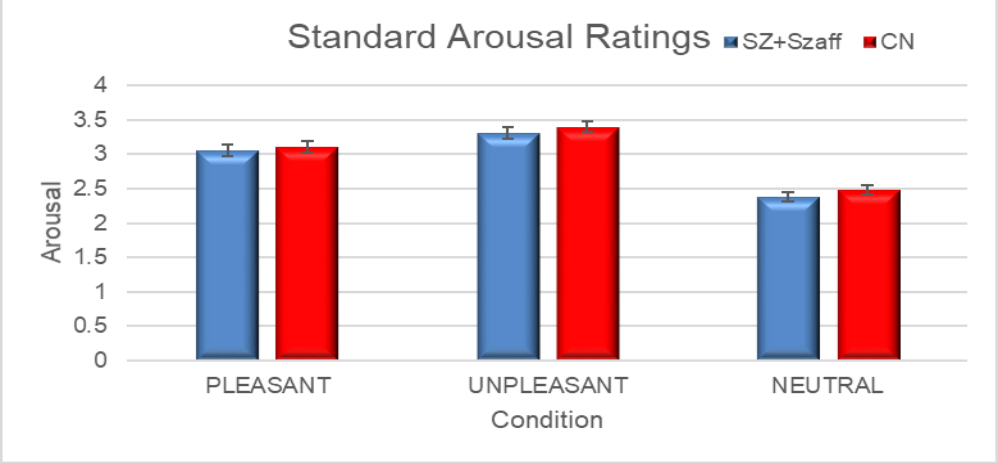


Note. SZ = Schizophrenia group; SZaff = Schizoaffective group; CN = Control group.

Supplemental Figure 2



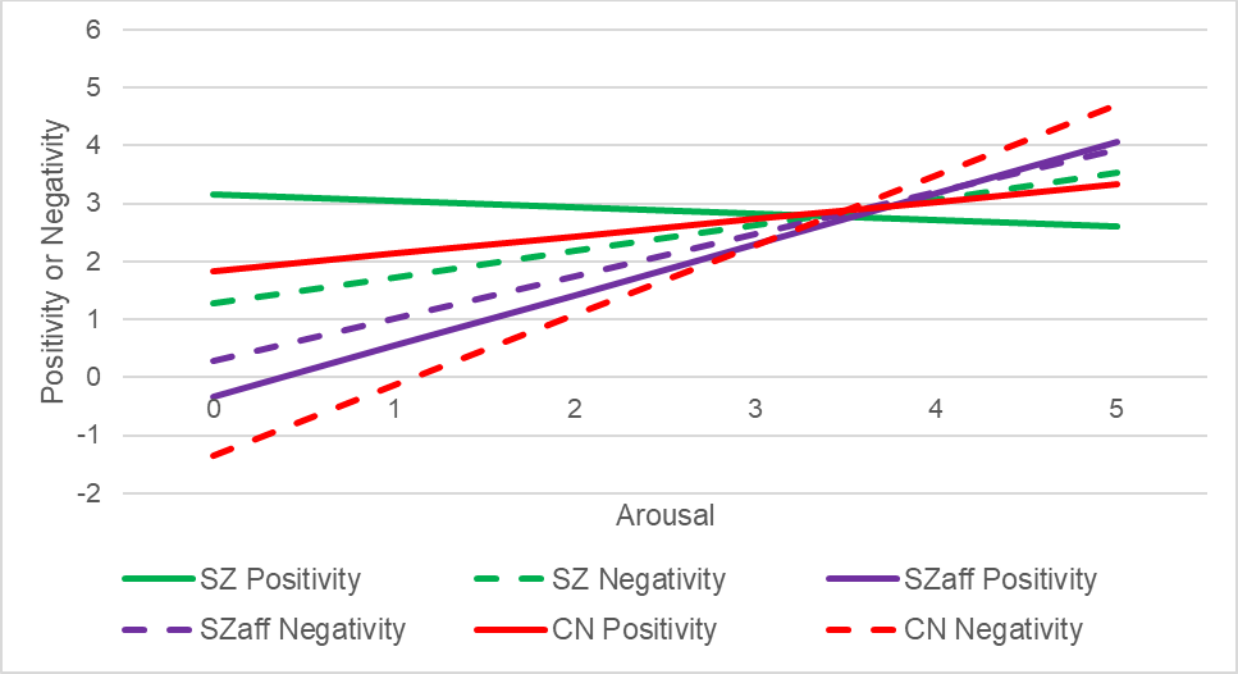




Note. SZ+SZaff = overall psychosis group; CN = Control group.

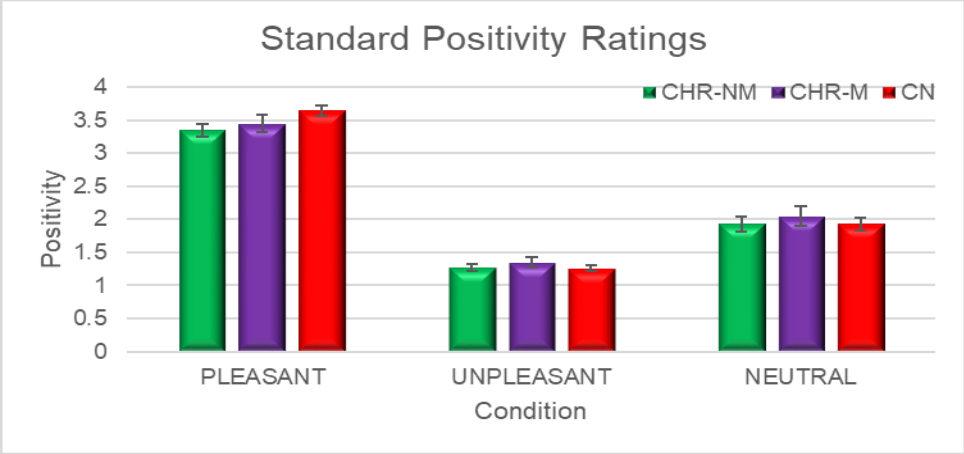
Supplemental Figure 3

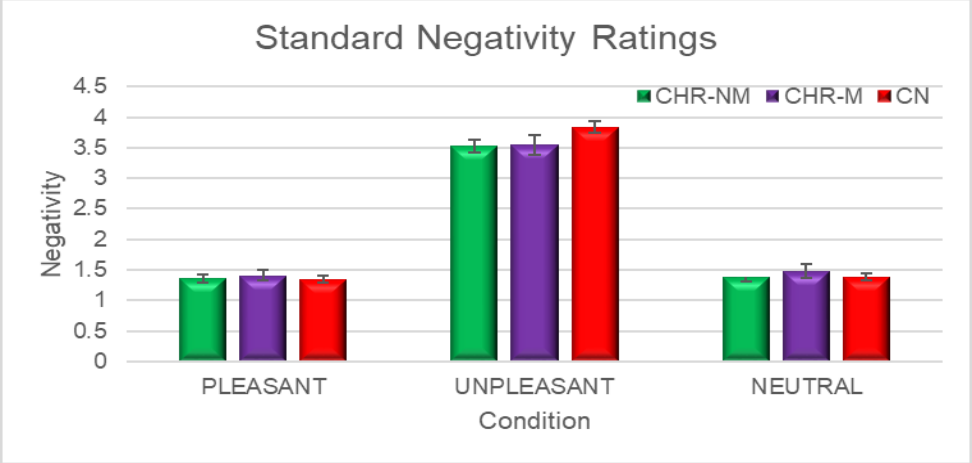
Positivity and Negativity Functions in Schizophrenia, Schizoaffective, and Control Groups

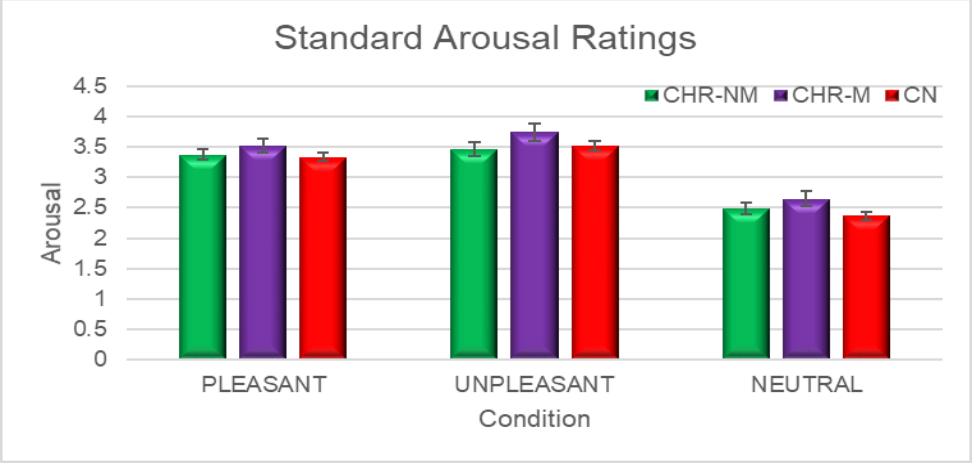


Note. SZ = Schizophrenia group; SZaff = Schizoaffective group; CN = Control group.

Supplemental Figure 4

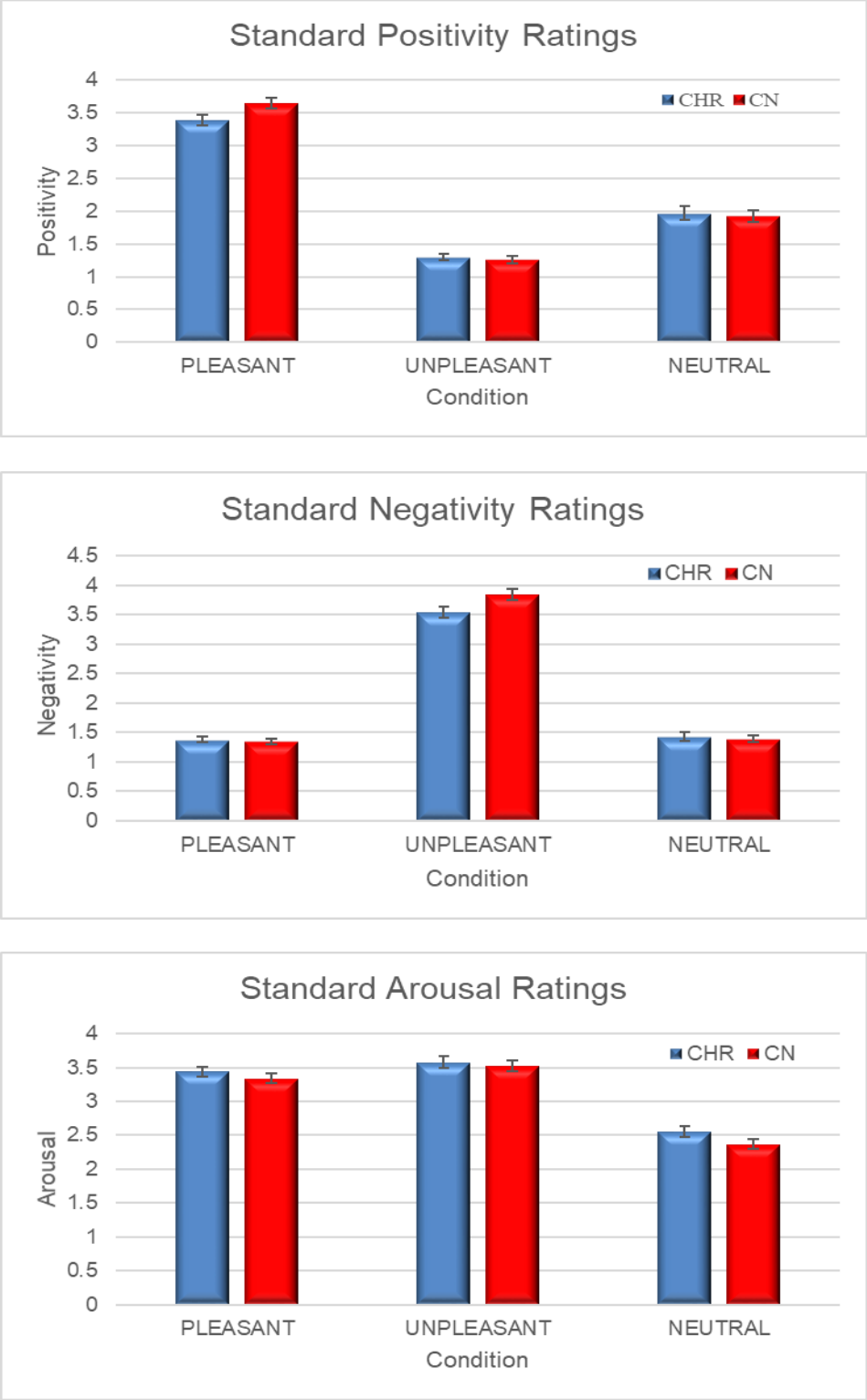






Note. CHR = clinical high-risk without a comorbid mood disorder diagnosis; CHR-M = clinical high-risk with a comorbid mood disorder diagnosis; CN = controls.

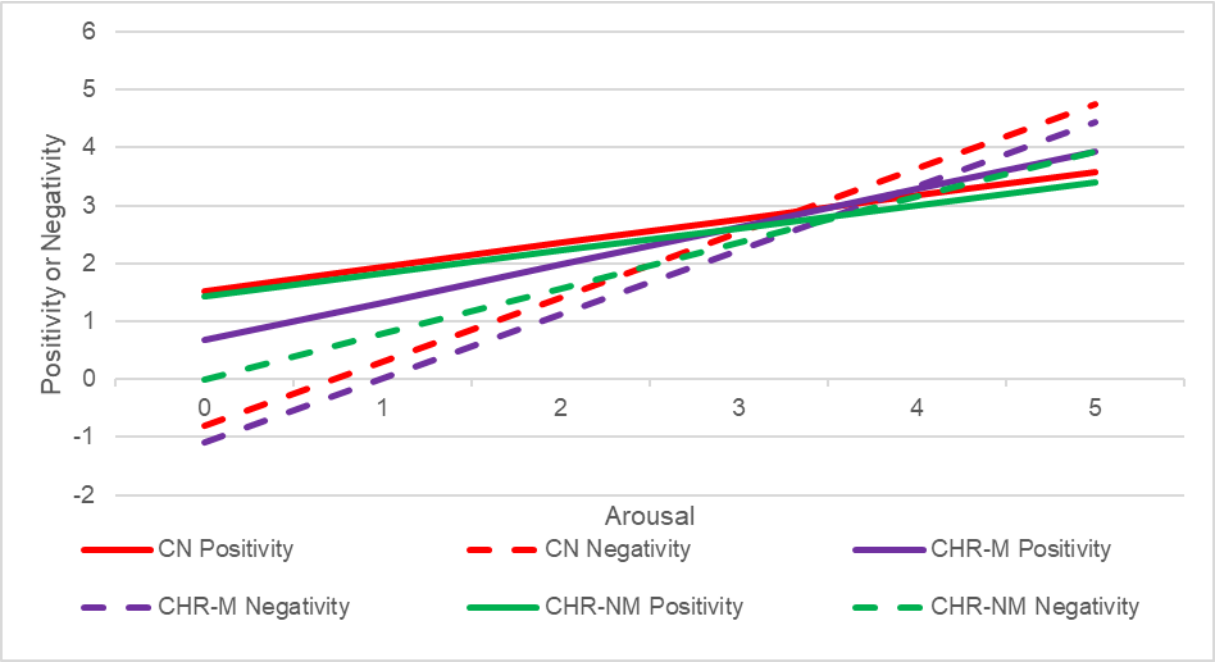
Supplemental Figure 5



Note. CHR = clinical high-risk; CN = controls.

Supplemental Figure 6

Positivity and Negativity Functions in Clinical High-risk Groups with and without Co-morbid Mood disorders and Healthy Controls



Note. CHR-NM = clinical high-risk without a comorbid mood disorder diagnosis; CHR-M = clinical high-risk with a comorbid mood disorder diagnosis; CN = controls.

APPENDEX C

CHAPTER 4 SUPPLEMENTAL MATERIAL

Supplemental Methods

Standard preliminary analyses of self-reported emotional experience variables were conducted similar to past studies comparing subjective positivity, negativity, and arousal to pleasant, unpleasant, and neutral stimuli. The average level of positive affect, negative affect, and arousal were calculated for each participant using responses to surveys completed during positive, negative and neutral contexts and used as the dependent variables for separate Group (SZ, CN) x Emotion Context (Positive, Negative, Neutral) mixed models ANOVAs. Multi-level models were also conducted in R to examine the effects of Group, Emotion Context, and Day on positive affect, negative affect, and arousal. Separate one-way ANOVAs were also used to compare the frequency of positive, negative, and neutral contexts endorsed by SZ and CN participants during the digital phenotyping period. Exploratory analyses consisted of conducting univariate ANOVA to examine the effects of two between-subjects factors, Sex (Male, Female) and Group (CHR, CN), and the Sex X Group interaction on the positivity offset and negativity bias difference scores, as well as the raw positivity and negativity parameters. Significant interactions were decomposed using one-way ANOVAs.

All analyses conducted in the main manuscript and described in the previous paragraph were repeated after splitting the CHR group into individuals at CHR for psychosis with (CHR-M, n = 66) and without (CHR, n = 34) co-morbid mood disorders. One-way ANOVA was used to examine group (CHR, CHR-M, CN) differences in the positivity offset and negativity bias

difference scores and raw parameters between CN, CHR, and CHR-M groups. LSD tests were used for post-hoc comparisons between groups. Groups significantly differed on age, such that individuals in the CHR-M group were older than CHR and CN participants. Groups also differed on personal education, with CHR participants completing fewer years of education compared to the CHR-M and CN groups. Groups were matched on all other demographic variables and demonstrated comparable adherence rates to momentary surveys. Group differences in clinically-rated symptoms were nonsignificant between CHR and CHR-M groups (see Supplemental Table X). There was insufficient passive digital phenotyping data to conduct correlation analyses in the CHR and CHR-M groups.

Supplemental Results

Supplemental Analyses in CHR (with and without co-morbid mood disorders) and CN

Preliminary Analyses of Standard Momentary Valence and Arousal Ratings

The main effect of Emotion Context on momentary positivity affect was significant, and the effects of Group and the Context X Group interaction were nonsignificant. For momentary negative affect, the main effects of Context and Group were both significant and the Context X Group interaction was nonsignificant. The main effect of Context on arousal was significant, and the main effects of Group and the Context X Group interaction were nonsignificant. See Supplemental Table 1 and Figure 1. Inconsistent with prior evidence supporting a hedonic deficit among individuals at CHR, the current findings indicate comparable levels of momentary positive affect across positive, negative, and neutral contexts in CHR compared to CN.

Effects of Group and Sex on the Positivity and Negativity Parameters

Univariate ANOVA indicated that the effects of Sex, Group, and the Sex X Group interaction on the positivity intercept, the positivity slope, and the positivity offset difference

score were nonsignificant. Similarly, all main effects and interactions on the negativity slope and negativity bias difference score were nonsignificant. For the negativity intercept, there was a significant main effect of Group and a nonsignificant effect of Sex and Sex X Group interaction. See Supplemental Table 2.

Supplemental Analyses with CHR-NM and CHR-M Subgroups Compared to Controls

Preliminary Analyses of Standard Momentary Valence and Arousal Ratings

Analyses of momentary positive affect ratings indicated a significant main effect of Emotion Context, a nonsignificant effect of Group, and a nonsignificant Context X Group interaction. For momentary levels of negative affect, the main effects of Context and Group were both significant and the Context X Group interaction was nonsignificant. Lastly, the main effect of Context on momentary arousal ratings was significant, and the effect of Group and the Context X Group interaction were nonsignificant. See Supplemental Table 3 and Figure 2. Collectively, these results indicate intact hedonic momentary responding across positive, negative, and neutral contexts in CHR and CHR-M groups.

Effects of Group and Sex on the Positivity and Negativity Parameters

Univariate ANOVA indicated that for both the positivity and negativity intercepts, the main effect of Group was significant, while the main effects of Sex and the Sex X Group interaction were nonsignificant. All effects on the positivity and negativity slopes were nonsignificant. For both the positivity offset and negativity bias difference scores, the main effect of Group was significant, and the effects of Sex and the Sex X Group interaction were nonsignificant. See Supplemental Table 4.

Supplemental Table 1

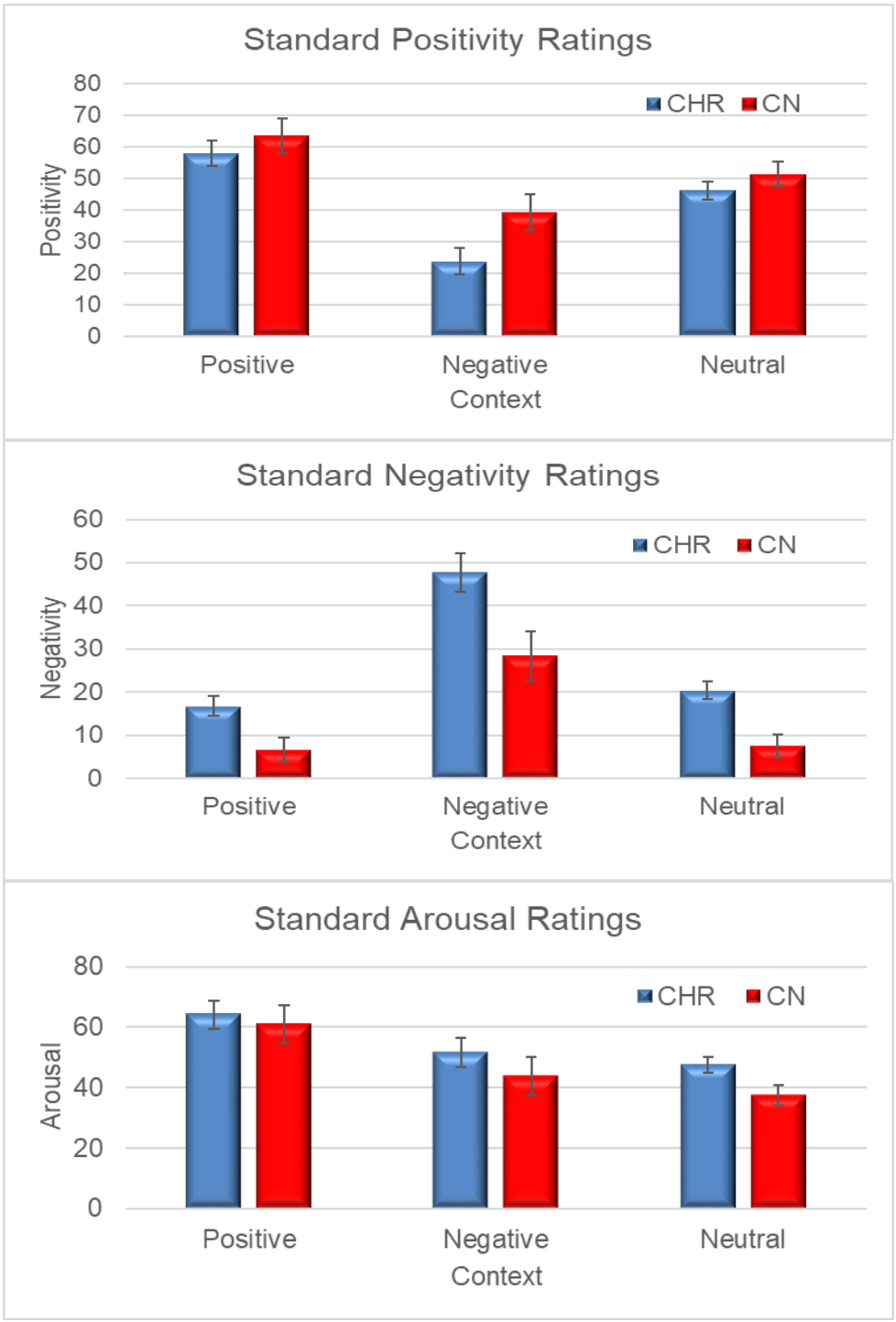
Mixed Models ANOVA Results Examining the Effects of Context and Group on Momentary Affect and Arousal

	Within subjects (Context)	Between Subjects (Group)	Interaction (Context x Group)
Positive Affect	$F(2, 64) = 38.37$ $p < .001$, $\eta_p^2 = .55$	$F(1, 32) = 3.13$ $p = .09$, $\eta_p^2 = .09$	$F(2, 64) = 1.6$ $p = .21$, $\eta_p^2 = .05$
Negative Affect	$F(2, 60) = 58.59$ $p < .001$, $\eta_p^2 = .66$	$F(1, 30) = 12.28$ $p = .001$, $\eta_p^2 = .29$	$F(2, 60) = 1.58$ $p = .22$, $\eta_p^2 = .05$
Arousal	$F(2, 82) = 13.04$ $p < .001$, $\eta_p^2 = .24$	$F(1, 41) = .21$ $p = .65$, $\eta_p^2 = .01$	$F(2, 82) = .35$ $p = .66$, $\eta_p^2 = .01$

Note. Context = positive, negative, or neutral momentary emotional context. Group = CHR or CN.

Supplemental Figure 1

Positivity, Negativity, and Arousal Ratings by Group and Emotion Context



Note. CHR = clinical high-risk; CN = controls.

Supplemental Table 2

Univariate ANOVA Results Examining the Effects of Sex and Group on Positivity and Negativity Parameters

	Between subjects (Sex)	Between Subjects (Group)	Interaction (Sex x Group)	Post-hoc
Positivity Intercept	$F(1, 157) = .80$ $p = .37, \eta_p^2 = .01$	$F(1, 157) = .84$ $p = .36, \eta_p^2 = .01$	$F(1, 157) = .93$ $p = .34, \eta_p^2 = .01$	-
Negativity Intercept	$F(1, 157) = .40$ $p = .53, \eta_p^2 = .003$	$F(1, 157) = 8.14$ $p = .01, \eta_p^2 = .05$	$F(1, 157) = .07$ $p = .79, \eta_p^2 = 0$	-
Positivity Slope	$F(1, 157) = .75$ $p = .39, \eta_p^2 = .01$	$F(1, 157) = .001$ $p = .98, \eta_p^2 = .60$	$F(1, 157) = .28$ $p = .60, \eta_p^2 = .002$	-
Negativity Slope	$F(1, 157) = .87$ $p = .35, \eta_p^2 = .01$	$F(1, 157) = .49$ $p = .48, \eta_p^2 = .003$	$F(1, 157) = 0$ $p = .99, \eta_p^2 = 0$	-
Positivity Offset	$F(1, 157) = .84$ $p = .36, \eta_p^2 = .01$	$F(1, 157) = 3.94$ $p = .049, \eta_p^2 = .03$	$F(1, 157) = .63$ $p = .43, \eta_p^2 = .004$	-
Negativity Bias	$F(1, 157) = 1.05$ $p = .31, \eta_p^2 = .01$	$F(1, 157) = .10$ $p = .76, \eta_p^2 = .001$	$F(1, 157) = .15$ $p = .70, \eta_p^2 = .001$	-

Note. Sex = male (M) or female (F). Group = CHR or CN.

Supplemental Table 3

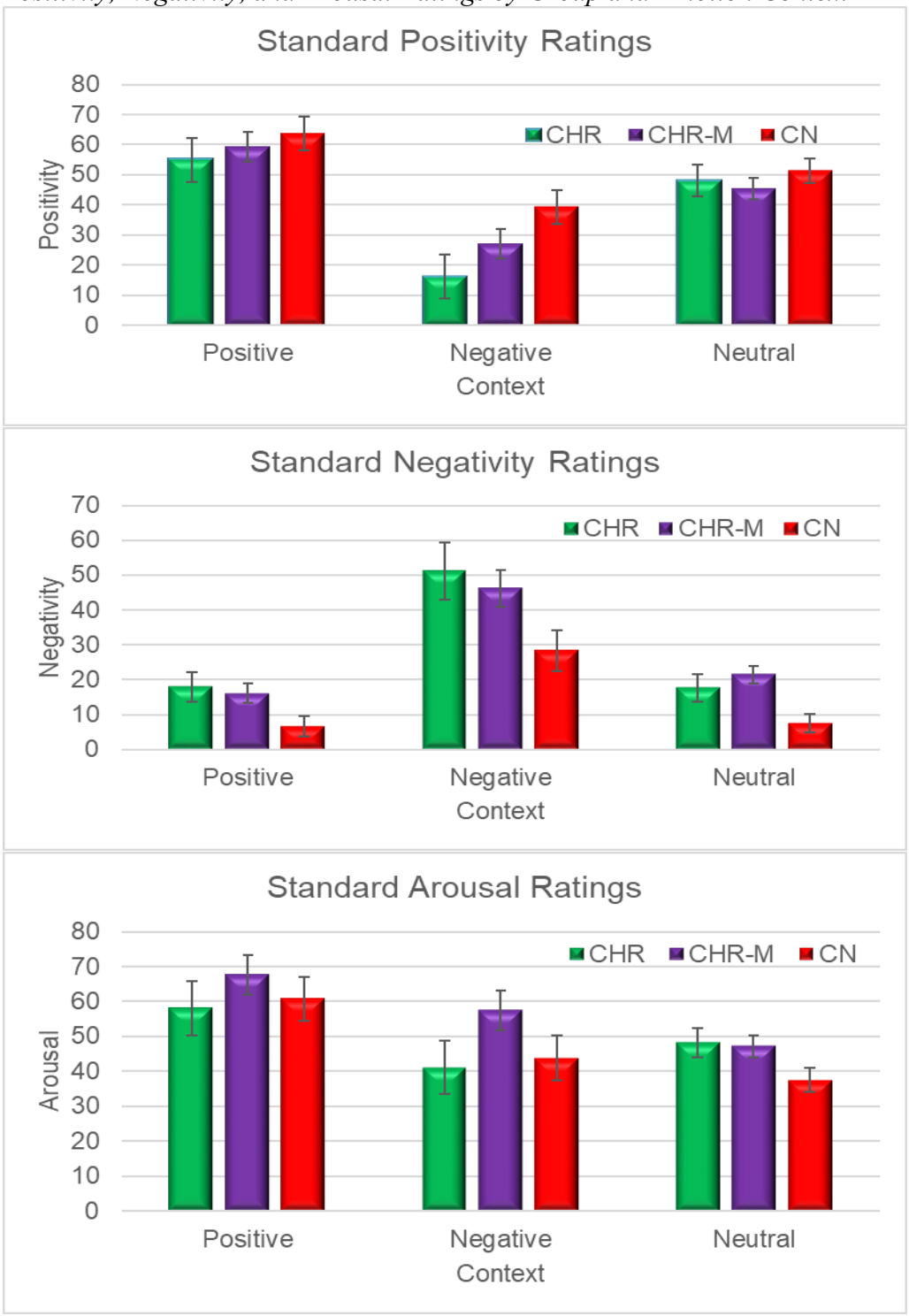
Mixed Models ANOVA Results Examining the Effects of Context and Group on Momentary Affect and Arousal

	Within subjects (Context)	Between Subjects (Group)	Interaction (Context x Group)
Positive Affect	$F(2, 62) = 45.74$ $p < .001$, $\eta_p^2 = .60$	$F(2, 31) = 1.75$ $p = .19$, $\eta_p^2 = .10$	$F(4, 62) = 1.42$ $p = .24$, $\eta_p^2 = .08$
Negative Affect	$F(2, 58) = 63.66$ $p < .001$, $\eta_p^2 = .69$	$F(2, 29) = 5.96$ $p = .01$, $\eta_p^2 = .29$	$F(4, 58) = 1.17$ $p = .33$, $\eta_p^2 = .07$
Arousal	$F(2, 80) = 11.56$ $p < .001$, $\eta_p^2 = .22$	$F(2, 40) = 1.88$ $p = .17$, $\eta_p^2 = .09$	$F(4, 80) = .95$ $p = .43$, $\eta_p^2 = .05$

Note. Context = positive, negative, or neutral momentary emotional context. Group = CHR-NM (CHR without comorbid mood disorders), CHR-M (CHR with comorbid mood disorders), CN.

Supplemental Figure 2

Positivity, Negativity, and Arousal Ratings by Group and Emotion Context



Note. CHR = clinical high-risk without a comorbid mood disorder diagnosis; CHR-M = clinical high-risk with a comorbid mood disorder diagnosis; CN = controls.

Supplemental Table 4

Univariate ANOVA Results Examining the Effects of Sex and Group on Positivity and Negativity Parameters

	Between subjects (Sex)	Between Subjects (Group)	Interaction (Sex x Group)	Post-hoc
Positivity Intercept	$F(1, 157) = 1.13$ $p = .29$, $\eta_p^2 = .01$	$F(2, 157) = 3.60$ $p = .03$, $\eta_p^2 = .05$	$F(2, 157) = .77$ $p = .47$, $\eta_p^2 = .01$	-
Negativity Intercept	$F(1, 157) = .19$ $p = .67$, $\eta_p^2 = .001$	$F(2, 157) = 6.63$ $p = .002$, $\eta_p^2 = .08$	$F(2, 157) = .01$ $p = .99$, $\eta_p^2 = 0$	-
Positivity Slope	$F(1, 157) = .81$ $p = .37$, $\eta_p^2 = .01$	$F(2, 157) = 2.88$ $p = .06$, $\eta_p^2 = .04$	$F(2, 157) = .52$ $p = .60$, $\eta_p^2 = .01$	-
Negativity Slope	$F(1, 157) = .64$ $p = .42$, $\eta_p^2 = .004$	$F(2, 157) = 1.24$ $p = .29$, $\eta_p^2 = .02$	$F(2, 157) = .08$ $p = .93$, $\eta_p^2 = .001$	-
Positivity Offset	$F(1, 157) = .90$ $p = .35$, $\eta_p^2 = .01$	$F(2, 157) = 6.07$ $p = .003$, $\eta_p^2 = .07$	$F(2, 157) = .44$ $p = .65$, $\eta_p^2 = .01$	-
Negativity Bias	$F(1, 157) = .99$ $p = .32$, $\eta_p^2 = .01$	$F(2, 157) = 2.84$ $p = .06$, $\eta_p^2 = 0.04$	$F(2, 157) = .39$ $p = .68$, $\eta_p^2 = .01$	-

Note. Sex = male (M) or female (F). Group = CHR-NM (CHR without comorbid mood disorders), CHR-M (CHR with comorbid mood disorders), CN.